GUIDELINES ON THE MANAGEMENT OF DEMENTIA (2003)

Assessment

1. **Who**

   Assessment by members of the multi-disciplinary team, but the doctors must be involved at some stage.

2. **Where**

   Wherever is appropriate and convenient to the patient and the team (e.g. OPD, Old Age Home, at home, A&E etc).

3. **How**

   3.1 Comprehensive history

   “**Dementia is a syndrome of persistent (more than 6 months) decline in memory and other cognitive functions sufficient to affect daily life in an alert patient**”

   **Common symptoms of Dementia:**

   1. Memory loss affecting job skills or other activities
   2. difficulty performing familiar tasks
   3. problems with language
   4. disorientation
   5. impaired judgement
   6. problems with abstract thinking
   7. continuous misplacement of personal possessions
   8. changes in mood or behaviour
   9. changes in personality
   10. loss of initiative

   ◆ History from the patient but beware that patient might minimizes or denies.
◆ Collateral history from the family or carers mandatory for elaboration and clarification of the history, as well as the beginning of the discussion and planning for future care.

◆ Includes the following areas:

1. present history- the onset and the course of illness
2. past psychiatric and medical history
3. drug and alcohol history
4. previous personality
5. family history
6. personal history
7. present level of functioning in personal hygiene and care; activities of daily living; social and interpersonal relationships, competency evaluation if necessary (e.g. ability to make decisions such as management of personal finances; ability to give consent to a specific medical treatment, etc)
8. present family and social supports
9. caregiver evaluation-- including stresses in carers

3.2 Examination

◆ Standard mental examination mandatory, with special emphasis on cognitive assessment. Standardized cognitive tests (e.g. MMSE) desirable esp. for following the progression of the impairment. Also screen for depression.

◆ Adequate physical examination especially a brief neurological examination essential
3.3 Investigations

◆ Routine screening tests include

1. CBP
2. ESR
3. RFT
4. LFT
5. TFT

◆ The following are done if there are sufficient clinical indications:

5. Urinalysis
6. CXR
7. ECG
8. Calcium & phosphate
9. Blood glucose
10. VDRL
11. B12 & folate
12. Immunology
13. CT Brain Scan- to detect brain lesions e.g subdural haematoma, hydrocephalus, tumour, cysts, etc
14. EEG- in mild dementia the EEG may remain entirely normal
15. Neuropsychological assessment (e.g to assist with the diagnosis of very mild or atypical presentations, to assess level of functioning in certain issues such as handling finances, to obtain a detailed baseline of cognitive abilities from which to measure progress over time)
Management

1. Aims

- To be on the lookout for, and to treat, complicating and aggravating physical and/or psychosocial factors as early as possible.
- To prevent clinical deterioration as much as is practicable.
- To maintain optimal physical functioning and well-being.

2. Desirable Outcome

To keep the patient in the community for as long a practicable; To reduce carer burden

3. How

- Management should be by the whole multidisciplinary team.
- Effective and timely communications between the team and the family/carers, other professionals, and between team members themselves.
- As much practical and psychological help to the family/carers as practicable, eg. sympathetic listening adequate explanations, financial help, home help etc.
- Short-term admission to a psychogeriatric unit when necessary for diagnosis, further investigations, or managing acute or short-term problems.
- Psychosocial treatments wherever appropriate, e.g. Reality Orientation, Reminiscence Groups, behavioural treatment etc.
- Most medications are used for symptomatic treatment, e.g. reducing the level of arousal and agitation, helping sleep etc. Others, e.g. nootropics may be used, hopefully to delay or even partly to reverse the deterioration, but to date there is insufficient evidence to show that it is useful in this regard in moderate or severe dementia. No medications should be continued indefinitely without regular review and monitoring. Please refer to the Appendix I for information of
Newer medications such as cholinesterases inhibitors, e.g. donepezil and rivastigmine are symptomatic treatment for mild to moderate Alzheimer’s disease; the overall benefits on cognitive function and the global assessment are modest. They also showed positive effect on emotional behavioural symptoms of Alzheimer’s disease. At present, there is insufficient information to recommend that they be given to patients suffering from severe disease or to patients in nursing home. Although no head-to-head clinical trials have compared the 2 cholinesterase inhibitors, similar modest improvements in ADAS-Cog scores and global clinical change have been reported in the treatment of patients with mild-moderate AD. However these drugs are expensive and have side effects which may be significant in some patients. As a result these medications should only be prescribed for selected patients individually after careful assessment of the indications and contraindications, with provision for regular, frequent review and monitoring. (Please refer to the following Algorithm)
Algorithm for the use of cholinesterase inhibitors (C.I.)

Is this patient suffering from Mild to Moderate Dementia in Alzheimer’s Disease according to ICD-10 criteria and MMSE ≥ 10 OR Dementia with Lewy bodies with behavioural and psychological symptoms of dementia?

Yes

Is there any medical contraindication with C.I.?

No

Is this patient compliant or is there any reliable caregiver to ensure compliance?

Yes

Likely to be beneficial

Start with a C.I. (advice to patient and carer prior to prescription)

Review at 2-4 weeks for any side effect and/or titrate dose

Review at 12 weeks to see if there is positive clinical response (e.g. improvement in cognitive functions, ADL, emotional behavioural problems, or global assessment)

Consider discontinuation if
a. lack of clinical response, or
b. patient has entered into severe stage of dementia in Alzheimer’s Disease (e.g. according to ICD-10 criteria, MMSE < 10, institutionalized due to dependent ADL, etc), or
c. poor compliance, or
d. poor tolerance or safety

Yes

Consider continuation and monitor every 3-6 months to review clinical progress and staging of dementia

No

May try the following strategies:
1) Stop the current drug then restart it later with lower dose, or
2) Switch to another C.I. and start with lower dose

Effective

Ineffective

Poor tolerability

Unlikely to be beneficial

No

Yes

Review at 2-4 weeks for any side effect and/or titrate dose

Good tolerability

Yes

Closer follow-up is needed to monitor deterioration upon discontinuation
Appendix I

References for Pharmacological Treatment of Dementia

<table>
<thead>
<tr>
<th>Treatment of Cognitive symptoms</th>
<th>Sources of Information</th>
<th>Evidence-base briefing: dementia. The Royal College of Psychiatrists, 1999 (other than APA, 1997)</th>
</tr>
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</table>

One systematic review has found that donepezil versus placebo significantly improves cognitive function at up to 52 weeks in people with mild or moderate Alzheimer's disease. One subsequent RCT in people with moderate to severe Alzheimer's disease has found that donepezil versus placebo significantly improves cognitive function and functional and behavioural symptoms at 24 weeks. We found no RCTs assessing the effects of donepezil on quality of life. We found no RCTs of donepezil in vascular dementia or Lewy body dementia.

One systematic review has found that rivastigmine improves cognitive function in people with Alzheimer's disease, but adverse effects such as nausea, vomiting, and anorexia are common. We found no RCTs about the effects of rivastigmine in people with vascular dementia. One RCT in people with Lewy body dementia found improvement of cognitive function and behaviour with rivastigmine versus placebo.

- in light of limited current knowledge, general practitioners should not initiate treatment with donepezil (CHSR, 1997)
- evidence to support the use of donepezil in the treatment of mild to moderate senile dementia of Alzheimer type, is borderline. There is evidence of temporary benefit but at a considerable cost. Treatment should be targeted carefully. Any protocol for its use should include clear criteria and a review two to three months (Stein, 1997)
- donepezil improved cognitive and global function in mild to moderate Alzheimer's disease (EBMH, 1998)
- the Standing Medical Advisory Committee recommends that treatment with donepezil should be initiated and supervised only by a specialist experienced in management of dementia. Benefit should be assessed at 12 weeks. Treatment should continue only for those patients with evidence of benefit (Standing Medical Advisory Committee (DOM), 1998)
- donepezil is an expensive new drug... (Bondolier, 1997)
<table>
<thead>
<tr>
<th><strong>b. Ergoloid mesylates (Hydergine)</strong></th>
<th>The questionable efficacy suggested by extensive study argues against routine use of this medication in the treatment of dementia. However under some circumstances it may be appropriate to offer a trial of this agent for Vascular Dementia. The use of the medication may be safely continued for patients whose families report a benefit. The manufacturer’s recommended dose is 3 mg/day, but studies using 4 or more (up to 9) mg/day were more likely to show significant improvements.</th>
<th>Hydergine was ineffective at 3 mg per day and showed slight memory improvement at 6 mg per day, but did not meet a priori benefit standard.</th>
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<tr>
<td><strong>c. Ginkgo biloba</strong></td>
<td>One systematic review has found that Ginkgo biloba versus placebo significantly improves cognitive function and is well tolerated in Alzheimer’s disease. We found no clear evidence to support its use in vascular or Lewy body dementia.</td>
<td>It was safe in one Class I trial of patients with mixed dementia, but benefits fall short of those expected for clinically effective antidementia treatments. Seven patients have to be treated with 120 mg of Gingko extract daily for one year for one of them to have an improved Alzheimer’s disease Assessment Score (ADAS-Cog) of four points which they would not have had with placebo. For a two-point improvement, about four patients have to be treated for one year. For a patient’s family member to notice an improvement in their daily living and social behavior about seven patients have to be treated for one year. It appears to have a modest stabilizing effect on the general functional decline of otherwise healthy patients with dementia. It appeared to be as safe as placebo, although the small number of patients and the short time period limits the ability to detect uncommon events. The changes reported are of a similar magnitude to those seen with tacrine and donepezil, two currently available medications that, locally, cast three times more than Gingko extracts. Whether it is safer or more effective than this medication are not clear. Recommendations to patients should be made with caution, since Gingko does not face the regulatory scrutiny of prescription medications. Nonetheless it appears that it may have some beneficial effects in demented individuals. Gingko biloba safely and modestly improved dementia (EBM, 1998)</td>
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## Slowing of Disease Progression in Dementia

### a. Vitamin E
May be used in moderately impaired patients with Alzheimer’s disease in order to delay the progression of disease. The efficacy reported here are for a fairly high dose of 2000IU/day. Beware of worsening of coagulation defects in patients with vitamin K deficiency. One RCT in people with Alzheimer’s disease found no significant difference in cognitive function after 2 years’ treatment with vitamin E versus placebo, but found that vitamin E significantly reduced mortality, institutionalisation, loss of ability to perform activities of daily living, and the proportion of people who developed severe dementia. We found no RCTs about vitamin E in vascular or Lewy body dementia. Doses at 2000IU per day significantly delayed the time to composite outcome of primary measures indicative of clinical worsening, and fewer were institutionalized. (NB. there was no additive effect from vitamin E plus selegiline)

### b. Selegiline
Delaying the progression of Alzheimer’s Disease with moderate impairment. If side effects and medication interaction do not pose a problem, it may be continued in patients whose families report a benefit. Possible benefit. Selegiline (5mg BD) is supported by one study, but has a less favourable risk-benefit ratio. There is not yet enough evidence to recommend its routine use in practice (Birks & Flicker, 1998). The role of seligiline in the treatment of Alzheimer’s Disease has still to be established by large well-controlled, long term clinical trials. (DARE;NHS CRD, 1998)

### c. Others (e.g. anti-inflammatory agents, estrogen etc)
Preliminary evidence only. Need to weigh the risks and benefits when one using or considering using these agents. One RCT in people with Alzheimer’s disease found no significant difference in cognitive function after 25 weeks’ treatment with diclofenac plus misoprostol versus placebo. Another RCT in people with Alzheimer’s disease found that indometacin versus placebo significantly improved cognitive function after 6 months’ treatment. We found no RCTs of non-steroidal anti-inflammatory drugs in people with vascular or Lewy body dementia. One systematic review in women with established Alzheimer’s disease has found that oestrogen versus no oestrogen improves cognition. We found no evidence about its use in people with vascular or Lewy body dementia. Not supported by prospective data. Estrogen should not be prescribed to treat Alzheimer’s disease.
<table>
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<tr>
<th>Treatment for psychosis and agitation in dementia</th>
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<tr>
<td><strong>a. antipsychotics</strong></td>
<td>There are no efficacy data to guide the choice among antipsychotic agents. Instead the choice is based on the side effect profile.</td>
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<td></td>
<td>One systematic review in people with various types of dementia found no significant difference in agitation with haloperidol versus placebo, but found limited evidence that haloperidol may significantly reduce aggression. One RCT in people with moderate to severe dementia including Alzheimer's disease and vascular dementia found that risperidone versus placebo significantly improved behavioural and psychological symptoms over 12 weeks, but another RCT in people with severe dementia and agitation found no significant difference in symptoms over 13 weeks. One RCT in people with Alzheimer's disease and behavioural and psychological symptoms found that olanzapine versus placebo reduced agitation, hallucinations, and delusions. RCTs have found no significant difference in efficacy between different antipsychotics.</td>
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<td></td>
<td>Antipsychotics should be used to treat agitation or psychosis in patients with dementia where environmental manipulation fails. Atypical agents may be better tolerated compared with traditional agents.</td>
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<td></td>
<td>-treatment should normally be short-term and be reviewed regularly. (SIGN, 1998)</td>
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<td></td>
<td>-neuroleptics should normally be avoided in Lewy Body Type Dementia (SIGN, 1998)</td>
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<td></td>
<td>-start low, go slow (SIGN, 1998)</td>
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<td></td>
<td>-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 (SIGN, 1998)</td>
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<td></td>
<td>-in crisis situation the short-term use of neuroleptics may be appropriate (CHSR, 1998)</td>
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<td></td>
<td>-there is no clear evidence for the superiority of one neuroleptic drug over any other (SIGN, 1998)</td>
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<td></td>
<td>-Drug treatment should be aimed mainly at controlling behaviour and treating causes of acute deterioration. Major tranquilisers may be useful to reduce disinhibition, aggression or wandering, but may cause considerable sedation, extrapyramidal movement disorder and increased confusion. In general, all drugs should be reviewed regularly. Polypharmacy is particularly likely to lead to adverse effects in patients with dementia, and compliance is often a problem. (Haines &amp; Katona, 1992 (RCRP))</td>
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<tr>
<td><strong>b. benzodiazepine</strong></td>
<td>Useful in those with anxiety, on an as-needed basis. Risk of disinhibition, oversedation, falls and delirium.</td>
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<td></td>
<td>-Consider short-term anxiolytic or hypnotic treatment for severe and persistent symptoms. (SIGN, 1998)</td>
</tr>
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<td></td>
<td>-Triazolam caused sedation and impairment of psychomotor performance in elderly people (AC Journal Club, 1991)</td>
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<tr>
<td><strong>c. anticonvulsants</strong></td>
<td>Given the sparse data on these agents, they cannot be recommended with confidence for the treatment of agitation in demented patients. Nonetheless, a therapeutic trial of one of these agents may be appropriate for some nonpsychotic patients, especially those who are mildly agitated or are sensitive or unresponsive to antipsychotics.</td>
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<tr>
<td></td>
<td>One RCT found that carbamazepine versus placebo significantly reduced agitation and aggression in people with agitation and unspecified dementia. Another RCT found that sodium valproate reduced agitation in unspecified dementia. We found no RCTs about other antiepileptic drugs.</td>
</tr>
<tr>
<td></td>
<td>-Triazolam caused sedation and impairment of psychomotor performance in elderly people (AC Journal Club, 1991)</td>
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<tr>
<td><strong>d. SSRI</strong></td>
<td>Preliminary data suggest that SSRIs may be useful</td>
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<td></td>
<td>One RCT in people with dementia plus agitated behaviour found no significant difference in agitation with trazodone versus haloperidol. Another RCT in people with Alzheimer's disease and agitated behaviours found no significant difference in outcomes between trazodone, haloperidol, behaviour management techniques, and placebo. The RCTs may have been too small to exclude a clinically important difference.</td>
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### Treatment for depression in dementia

<table>
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<tr>
<th>The choice antidepressant is generally based on the side effect profile and the general medical and psychiatric status of each patient.</th>
<th>Selected tricycles, MAOI, and SSRI should be considered with side effects profiles guiding the choice of agent.</th>
<th>Consider a trial of antidepressant medication at a therapeutic dose evaluated against explicit criteria such as activities of daily living, level of functioning, behavioural disturbance and biological features of recent onset. Moclobemide was an effective antidepressant in elderly patients with cognitive decline and depression.</th>
</tr>
</thead>
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<tr>
<td><strong>b.</strong> Electroconvulsive therapy (ECT)</td>
<td>Twice weekly rather than thrice-weekly and unilateral rather than bilateral ECT may decrease the risk of cognitive side effects after ECT</td>
<td></td>
</tr>
</tbody>
</table>
References:

1. Practice Guideline for the treatment of patients with Alzheimer’s Disease and other Dementias of Late Life, American Psychiatric Association, 1997 (http://www.psych.org/clin_resp/pg_dementia.cfm)

2. National Guideline Clearinghouse, American Medical Directors Association (AMDA Dementia. Columbia (MD):American Medical Directors Association (AMDA);1998 32p (http://www.guidelines.gov)


