Management of Behavioral and Psychological Symptoms in People with Dementia Living in Care Homes: A UK Perspective

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King’s College London
And Director of Research, Alzheimer’s Society (UK)
750,000 people now
750,000 families
1 million by 2025

Numbers of people with late onset dementia by age group

- 750,000 people now
- 750,000 families
- 1 million by 2025
Where are people with dementia?

- 424k in the community (64%)
- 244k in care homes (36%)

Proportion in care rises with age

Bar chart showing the distribution of people with dementia by age group and care setting.
Care Homes in the UK

• Independent of the NHS: Vast majority are privately owned and run
• >70% of places funded by social services (means tested)
• 28,000 care homes: nursing homes and residential homes
• 25% places allocated for people with dementia
• Care Quality Commission acts as the regulator
Care Homes and Dementia

- 750,000 people with dementia in the UK. 250,000 of these individuals live in care homes (Dementia UK report)
- >70% of people in care homes in the UK have dementia, despite only 25% of places being specifically registered for dementia patients
- No mandatory dementia training for care staff
- Nursing homes have legal requirement for minimum of trained nurses, no requirements in residential homes
- Almost all hands on care provided by care assistants on minimum wage, with no or minimal formal training (small proportion have NVQs)
- Massive turnover of care home staff, substantial proportion of care home staff speak poor English and often do not have a good grasp of relevant cultural issues
Care Quality Commission

• Governance body, answerable to government, responsible for ensuring adequate quality of care home services

• Role
  – Inspect care homes, but criteria very centred around “hands-on” care needs not social needs
  – Investigate complaints, reports of abuse and neglect, safeguarding issues
  – Assess quality of care
  – Produce a publicly available report for each care home
  – No responsibility for prescribing/pharmacotherapy issues
Antipsychotics in Care Homes

- Estimated that 180,000 people with dementia on antipsychotics in the UK, the majority residing in care homes
- Research studies suggest >40% of care home residents with dementia prescribed antipsychotics
- Median duration of antipsychotic prescriptions to people with dementia in care homes are 1-2 years
- Reducing Antipsychotic prescribing has become a major clinical and political issue in the UK, but is a medical rather than a care home responsibility
- Some people benefit from these medications (eg where there is severe and complex risk) where trials have not been completed but there may be particular value in using these medications.

- I estimate that we are treating 180,000 people with dementia with antipsychotic medication across the country per year. Of these, up to 36,000 will derive some benefit.

- Negative effects that are directly attributable to the use of antipsychotic medication at this level equates to
  - 1,620 cerebrovascular adverse events, around half of which may be severe
  - an additional 1,800 deaths per year on top of those that would be expected in this frail population

- I estimate that we can reduce the rate of use of antipsychotic medication to a third of its current level over a 36 month period.
2010-11: Action on antipsychotics (UK)

• Minister Paul Burstow pledges to reduce antipsychotic use by 2/3
• Department of Health Stakeholder group set up
• National audit and ongoing audits of antipsychotic prescribing
• Ministerial Advisory Group for dementia research prioritizes research to improve the treatment of neuropsychiatric symptoms
• Best practice guide (draft launched 9th June) – Developed by the Alzheimer’s Society with DH, with support of expert group and the Dementia Action Alliance
Department of Health Actions

- Target: to reduce antipsychotic prescribing by two thirds
- Beginning to Implement audit of medical prescribers, with goal of making information publicly available
- Mandatory enforcement of 12 week reviews (advisory up to now)
- Best Practice Guide
- Modest support for training initiatives (eg FITS)
- So far in 1 year – estimated reduction of 21% achieved, but government very dissatisfied with slow progress
Personal Reflections

• Care Quality Commission Need to monitor and report upon prolonged antipsychotic prescribing

• Substantial safe reductions in antipsychotic use and improved practice can only be achieved with a more consistent commitment to evidence based staff training to provide alternatives

• Without increased training, substantial risk that antipsychotics will be replaced by “non-evidence based” alternatives which may be equally or even more harmful

• Pharmacological and non-pharmacological management of Behavioural and Psychological Symptoms in people with dementia needs to be supported as a research priority
Agitation and other BPSD are common

Non AD dementias

• Vascular dementia (VaD) – Some VaD patients in 2 risperidone studies, but no separate analysis and no specific trials of VaD. Cochrane review of memantine in VaD indicates modest but significant benefit on NPI.

• DLB/PDD – only 1 RCT (with quetiapine), showing no significant benefit. Serious potential concerns re neuroleptic sensitivity. Several trials suggesting some benefit in DLB/PDD with rivastigmine. One poster of RCT indicating benefit of Pimavanserin in PD psychosis

• Marked need for treatment studies examining treatment of neuropsychiatric symptoms in non-AD dementias
Risperidone Efficacy: BEHAVE-AD  
Ballard & Howard 2006 Nature Neuroscience Reviews

<table>
<thead>
<tr>
<th>Target symptom</th>
<th>Mean Difference from placebo</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychosis</td>
<td>-0.79</td>
<td>p=0.03</td>
</tr>
<tr>
<td>Aggression</td>
<td>-0.84</td>
<td>p=0.0002</td>
</tr>
<tr>
<td>Aggression</td>
<td>-1.50</td>
<td>p&lt;0.0001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risperidone 1mg</th>
<th>-1.31 to -0.27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risperidone 1mg</td>
<td>-1.28 to -0.40</td>
</tr>
<tr>
<td>Risperidone 2mg</td>
<td>-2.05 to -0.95</td>
</tr>
</tbody>
</table>
### STAR TRIAL: Zhong et al 2007

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Quetiapine 200mg (N=114)</th>
<th>Quetiapine 100mg (N=120)</th>
<th>Placebo (N=92)</th>
<th>PANSS-EC</th>
<th>NPI (total)</th>
<th>NPI (agitation)</th>
<th>NPI (psychosis)</th>
<th>CGIC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-5.7 (0.9)</td>
<td>-4.9 (0.8)</td>
<td>-3.9 (0.9)</td>
<td>NS</td>
<td>-9.7 (2.2)</td>
<td>-8.9 (2.1)</td>
<td>-8.2 (2.4)</td>
<td>NS</td>
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<tr>
<td></td>
<td>-1.1 (0.5)</td>
<td>-0.9 (0.5)</td>
<td>-1.2 (0.5)</td>
<td>NS</td>
<td>-2.5 (0.9)</td>
<td>-1.8 (0.8)</td>
<td>-2.5 (0.9)</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>3.0 (0.2)</td>
<td>3.2 (0.2)</td>
<td>3.6 (0.2)</td>
<td>NS</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
### Adverse events with Risperidone

**Ballard & Howard 2006, Nature Neuroscience Reviews**

<table>
<thead>
<tr>
<th>Adverse events</th>
<th>Dose / day</th>
<th>Risperidone</th>
<th>Placebo</th>
<th>Odds Ratio</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Extra pyramidal symptoms</strong></td>
<td>1mg</td>
<td>32 / 500</td>
<td>20 / 571</td>
<td>1.78</td>
<td>1.00 to 3.17</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>2mg</td>
<td>35 / 165</td>
<td>12 / 163</td>
<td>3.39</td>
<td>1.69 to 6.80</td>
<td>p=0.0006</td>
</tr>
<tr>
<td><strong>Gait</strong></td>
<td>1mg</td>
<td>21 / 402</td>
<td>1 / 408</td>
<td>7.47</td>
<td>2.21 to 25.28</td>
<td>p=0.001</td>
</tr>
<tr>
<td><strong>Somnolence</strong></td>
<td>1mg</td>
<td>138 / 665</td>
<td>72 / 685</td>
<td>2.36</td>
<td>1.71 to 3.24</td>
<td>p&lt;0.00001</td>
</tr>
<tr>
<td></td>
<td>2mg</td>
<td>46 / 165</td>
<td>13 / 163</td>
<td>2.36</td>
<td>2.30 to 8.64</td>
<td>p&lt;0.00001</td>
</tr>
<tr>
<td><strong>Respiratory tract infection</strong></td>
<td>1mg</td>
<td>15 / 149</td>
<td>6 / 163</td>
<td>2.93</td>
<td>1.11 to 7.76</td>
<td>p=0.03</td>
</tr>
<tr>
<td><strong>fever</strong></td>
<td>2mg</td>
<td>24 / 165</td>
<td>12 / 163</td>
<td>2.14</td>
<td>1.03 to 4.44</td>
<td>p=0.04</td>
</tr>
<tr>
<td><strong>Peripheral oedema</strong></td>
<td>0.5mg</td>
<td>24 / 149</td>
<td>9 / 163</td>
<td>3.29</td>
<td>1.47 to 7.32</td>
<td>p=0.004</td>
</tr>
<tr>
<td></td>
<td>1mg</td>
<td>32 / 315</td>
<td>15 / 333</td>
<td>2.43</td>
<td>1.29 to 4.59</td>
<td>p=0.006</td>
</tr>
<tr>
<td></td>
<td>2mg</td>
<td>30 / 165</td>
<td>9 / 163</td>
<td>3.80</td>
<td>1.74 to 8.29</td>
<td>p=0.0008</td>
</tr>
</tbody>
</table>
Major Adverse Outcomes with antipsychotics over 6-12 weeks (Schneider et al 2005, Ballard et al 2009)

- Parkinsonism
- Sedation
- Gait disturbance
- Increased respiratory infections
- Oedema
- Accelerated cognitive decline
- Stroke (>3 fold)
- Other thrombo-embolic events
- Mortality (1.5-1.7 fold)
## No Benefit and Accelerated Cognitive Decline with Quetiapine

<table>
<thead>
<tr>
<th></th>
<th>rivastigmine</th>
<th>quetiapine</th>
<th>placebo</th>
<th>ChI v plac</th>
<th>Nlp v plac</th>
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</thead>
<tbody>
<tr>
<td><strong>Week 6</strong></td>
<td>N=24 (15 completed SIB)</td>
<td>N=26 (14 completed SIB)</td>
<td>N=29 (17 completed SIB)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diff CMAI</td>
<td>-8.3±18.4</td>
<td>-4.7±17.3</td>
<td>-6.2±17.2</td>
<td>T=0.4 P=0.67</td>
<td>T=0.3 P=0.74</td>
</tr>
<tr>
<td>Diff SIB</td>
<td>+4.2±15.4</td>
<td>-10.5±14.8</td>
<td>+2.8±15.5</td>
<td>T=0.3 P=0.80</td>
<td>T=2.4 P=0.02*</td>
</tr>
<tr>
<td><strong>Week 26</strong></td>
<td>N=24 (16 completed SIB)</td>
<td>N=26 (15 completed SIB)</td>
<td>N=29 (17 completed SIB)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diff SIB</td>
<td>-1.1±21.1</td>
<td>-11.6±15.6</td>
<td>+2.3±18.1</td>
<td>T=0.5 P=0.61</td>
<td>T=2.3 P=0.03*</td>
</tr>
<tr>
<td>Diff CMAI</td>
<td>-10.5±20.4</td>
<td>-4.4±15.7</td>
<td>-7.9±16.6</td>
<td>T=0.5 P=0.62</td>
<td>T=0.1 P=0.87</td>
</tr>
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</table>
### Change from Baseline to 6 months DART AD

**Ballard et al PLOS Medicine 2008**

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline</th>
<th>6 months</th>
<th>Change</th>
<th>p-value</th>
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<tbody>
<tr>
<td><strong>Total NPI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=56)</td>
<td>1.3 (15.5)</td>
<td>(n=53) 4.5 (17.6)</td>
<td>-2.4 (-8.2 to 3.5)&lt;sup&gt;3&lt;/sup&gt;</td>
<td>0.4</td>
</tr>
<tr>
<td><strong>MUPDRS</strong></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>(n=41)</td>
<td>0.8 (4.1)</td>
<td>(n=43) -0.4 (3.2)</td>
<td>1.3 (-0.4 to 3.0)&lt;sup&gt;4&lt;/sup&gt;</td>
<td>0.1</td>
</tr>
<tr>
<td><strong>Bristol ADL</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(n=54)</td>
<td>1.8 (8.9)</td>
<td>(n=52) 0.2 (7.2)</td>
<td>1.7 (-1.2 to 4.6)&lt;sup&gt;3&lt;/sup&gt;</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>Change in FAST&lt;sup&gt;5&lt;/sup&gt;</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-2</td>
<td></td>
<td>0</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td>-1</td>
<td></td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td></td>
<td>34</td>
<td>32</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td></td>
<td>12</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>4</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td><strong>CGIC&lt;sup&gt;5&lt;/sup&gt;</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very much improved</td>
<td>(n=48) 1 (2%)</td>
<td>(n=48) 0</td>
<td>1</td>
<td>0.9</td>
</tr>
<tr>
<td>Much improved</td>
<td>3 (6%)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Minimally improved</td>
<td>7 (15%)</td>
<td>14 (29%)</td>
<td>14 (29%)</td>
<td></td>
</tr>
<tr>
<td>No change</td>
<td>18 (37%)</td>
<td>14 (29%)</td>
<td>14 (29%)</td>
<td></td>
</tr>
<tr>
<td>Minimally worse</td>
<td>9 (19%)</td>
<td>10 (21%)</td>
<td>10 (21%)</td>
<td></td>
</tr>
<tr>
<td>Much worse</td>
<td>7 (15%)</td>
<td>10 (21%)</td>
<td>10 (21%)</td>
<td></td>
</tr>
<tr>
<td>Very much worse</td>
<td>3 (6%)</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>
DART AD: Differential Survival
Ballard et al Lancet Neurology 2009

Differences in the survival rates in the DART-AD trial

<table>
<thead>
<tr>
<th>Number of months</th>
<th>Survival rate on placebo</th>
<th>Survival rate on antipsychotic</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>71%</td>
<td>46%</td>
</tr>
<tr>
<td>36</td>
<td>59%</td>
<td>30%</td>
</tr>
<tr>
<td>42</td>
<td>53%</td>
<td>26%</td>
</tr>
</tbody>
</table>

Why do people die?

• Causes of death (Ballard et al 2010)
  – Pneumonia
  – Stroke
  – Pulmonary embolism
  – Sudden cardiac arrhythmias

• Likely Mediating Factors
  – Dehydration
  – Chest infection
  – Over sedation
  – Q-T prolongation
### FITS: Stopping Neuroleptics: Impact on Quality of Life

<table>
<thead>
<tr>
<th></th>
<th>n=42</th>
<th>Baseline (sd)</th>
<th>Follow-up</th>
<th>Evaluation (Baseline v Follow-up)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>FITS (sd)</td>
<td>Control (sd)</td>
</tr>
<tr>
<td><strong>Social Withdrawal</strong></td>
<td>42</td>
<td>6.64 (8.96)</td>
<td>-5.24 (13.56)</td>
<td>-1.29 (5.42)</td>
</tr>
<tr>
<td><strong>Daytime sleep</strong></td>
<td>-20.69 (23.24)</td>
<td>-6.20 (24.58)</td>
<td>-1.29 (24.38)</td>
<td>T 1.1 p=0.27</td>
</tr>
<tr>
<td><strong>Type 1 Behaviours</strong></td>
<td>+34.74 (19.53)</td>
<td>+13.44 (23.73)</td>
<td>+1.47 (24.29)</td>
<td>T 2.3 p=0.03</td>
</tr>
<tr>
<td><strong>Wellbeing</strong></td>
<td>0.65 (0.69)</td>
<td>+0.34 (0.59)</td>
<td>+0.15 (0.98)</td>
<td>T 2.2 p=0.03</td>
</tr>
<tr>
<td><strong>CMAI</strong></td>
<td>42.88 (14.57)</td>
<td>+0.75 (22.35)</td>
<td>+5.29 (12.74)</td>
<td>T 0.83 p=0.41</td>
</tr>
</tbody>
</table>
Standardized tailored psychological Interventions

- Care Homes:
  - Cohen-Mansfield 2007 (n=167) Placebo controlled trial of personalized non-pharmacological interventions for 4 hours over days resulted in significant reduction in agitation (p=0.002)
  - Cohen-Mansfield 1997 (n=58) Placebo controlled trial of “social interaction”, music or simulated presence resulted in significant 25% reduction in abnormal vocalizations over 6 weeks

- Teri and Colleagues (Seattle protocols), Gitlin and others have shown similar benefits with structured intervention programmes for people living in their own homes
Efficacy improves with severity of agitation
BPST “tool Box” intervention from CALM-AD STUDY

(Ballard et al Am J Ger Psychiatry 2009)

<table>
<thead>
<tr>
<th>N= 200</th>
<th>CMAI baseline</th>
<th>CMAI week 4</th>
<th>Evaluation (paired sample t test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>62.2±14.3</td>
<td>55.6±17.2</td>
<td>T=5.6 P&lt;0.0001</td>
</tr>
<tr>
<td>Baseline CMAI &lt;53</td>
<td>47.1±3.8</td>
<td>48.6±15.9</td>
<td>T=-0.7 P=0.46</td>
</tr>
<tr>
<td>Baseline CMAI 53-70</td>
<td>61.2±4.8</td>
<td>54.7±16.2</td>
<td>T=4.1 P&lt;0.0001</td>
</tr>
<tr>
<td>Baseline CMAI &gt;70</td>
<td>82.4±12.7</td>
<td>67.1±18.9</td>
<td>T=5.3 P&lt;0.0001</td>
</tr>
<tr>
<td>Study</td>
<td>Design Type</td>
<td>Sample Size</td>
<td>Outcome Measures</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------------------------</td>
<td>-------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Buettner L &amp; Fitzsimmons 2002</td>
<td>RCT</td>
<td>12</td>
<td>Significant results on depression</td>
</tr>
<tr>
<td>Choi AN et.al. 2008</td>
<td>Pilot-controlled trial</td>
<td>5</td>
<td>Sig. effect on agitation</td>
</tr>
<tr>
<td>Cooke ML et.al. 2009</td>
<td>Randomised cross-over design</td>
<td>8</td>
<td>NS</td>
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<tr>
<td>Ledger AJ &amp; Baker FA 2007</td>
<td>Longitudinal repeated measure design</td>
<td>42</td>
<td>NS</td>
</tr>
<tr>
<td>Lin Y et.al. 2010</td>
<td>Pretest-posttest control group design</td>
<td>6</td>
<td>Sig. decrease in agitation, total and 4 subfactors</td>
</tr>
<tr>
<td>Raglio A et.al. 2008</td>
<td>RCT</td>
<td>16</td>
<td>Sig. Decrease NPI in intervention group Sig. Diff. Between groups</td>
</tr>
<tr>
<td>Sung HC et.al.</td>
<td>Quasi-experiment</td>
<td>6</td>
<td>Sig. lower agitation</td>
</tr>
<tr>
<td>Sung HC et al 2010</td>
<td>Quasi-experiment pretest-posttes</td>
<td>6</td>
<td>Sig lower anxiety in intervention group p=0.001</td>
</tr>
</tbody>
</table>
## Validation and Reminiscence

<table>
<thead>
<tr>
<th>Study</th>
<th>Study design</th>
<th>Length</th>
<th>Sample</th>
<th>Impact</th>
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</thead>
<tbody>
<tr>
<td><strong>Validation therapy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deponte A &amp; Missan R 2006</td>
<td>Pre-test-post-test Randomly assigned</td>
<td>12</td>
<td>30</td>
<td>Within-group effects. SR, VT</td>
</tr>
<tr>
<td><strong>Reminiscence therapy</strong></td>
<td></td>
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<td></td>
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<tr>
<td>Chiang, KJ., et.al. 2010</td>
<td>Experimental design</td>
<td>8</td>
<td>130</td>
<td>Significant positive short-term effect on depression, psychological well-being and lonliness p&lt;0.0001</td>
</tr>
<tr>
<td>Haslam, C. et.al. 2010</td>
<td>RCT</td>
<td>6</td>
<td>115</td>
<td>Cognitive performance improved significantly in GR condition. p=0.04. Well-being in control group condition improved p=0.07</td>
</tr>
<tr>
<td>Jones ED 2003</td>
<td>RCT</td>
<td>3</td>
<td>30</td>
<td>Reduction GDS in intervention group. Significant diff between groups, p=0.002</td>
</tr>
<tr>
<td>Karimi, K., et.al. 2010</td>
<td>Three-group pre-post-test design randomised allocation</td>
<td>6</td>
<td>39</td>
<td>Sig diff between integrative RT and control condition</td>
</tr>
<tr>
<td>Lai, CKY., 2004</td>
<td>Single-blinded parallel-groups RCT</td>
<td>6</td>
<td>101</td>
<td>NS T1 and T0 p=0.014 on WIB</td>
</tr>
<tr>
<td>Wang, J-J., et.al. 2003</td>
<td>Quasi experimental random assignment</td>
<td>16</td>
<td>94</td>
<td>Sig diff pretest-posttest on depression, p=0.041</td>
</tr>
<tr>
<td>Wang, J-J., et.al. 2004</td>
<td>Longitudinal experimental</td>
<td>16</td>
<td>48</td>
<td>Depression, p=0.05 Mood, p=0.05</td>
</tr>
<tr>
<td>Wang, J-J., et.al. 2007</td>
<td>RCT</td>
<td>8</td>
<td>102</td>
<td>MMSE, p=0.015 CSDD, p=0.026</td>
</tr>
<tr>
<td>Wang, J-J., et.al. 2008</td>
<td>Longitudinal experimental</td>
<td>8</td>
<td>77</td>
<td>NS (sig.) p=0.011 on social disturbance subscale of CAPE-BRS</td>
</tr>
</tbody>
</table>
Intervention by a Clinical Psychologist –

- Bird et al 2009: 44 consecutive referrals for challenging behaviour (2/3 in residential care). Assessment and interventions were undertaken in collaboration with family carers and care staff. Outcomes Measures taken pre-intervention and up to 5-month follow-up. Psychotropic medication was used with a minority of participants but, overall, antipsychotic use was reduced. Psychosocial methods predominated, with 77% of cases judged as mainly or entirely psychosocial by expert panel. There were significant improvements in behaviour and carer distress. Using conservative criteria there was a 65.9% clinical success rate.

- Bird et al 2007: 33 residential care clients with BPSD referred to a community psychogeriatric service (intervention group) received treatment with focus on causes of behavior (ABC). Cases were managed primarily by psychosocial means with psychopharmacology as an adjunct. A control group was made up of 22 referrals to an adjacent service, which used primarily psychopharmacology with psychosocial methods as an occasional adjunct. Measures of behavior showed significant improvement in both groups at two- and five-months' follow-up. Antipsychotic use in the intervention group decreased over time while in the control group it increased. Five control group participants spent extended periods as inpatients in a psychogeriatric unit.
Person Centred Care – Kitwood 1995

Person’s Experience

= B + P + H + NI + SP

Background and Lifestyle

Personality

Cognitive Support Needs

Health

Illness

Life at the Moment
Example – shared formulations using PCC and CBT ideas. (See Fossey and James 2008)
Two interventions: Person Centred Care Training and Dementia Care Mapping (DCM)

4 month cluster trial, 15 care homes, 289 residents with dementia

Significant mean difference of 10.9 on CMAI (95% CI 0.7-21.1; p=0.04) was achieved with DCM and a difference of 13.6 on the CMAI (95% CI 3.3-23.9; p=0.01) with Person Centred Care Training

Standardized Effect size of 0.55

Neither intervention reduced antipsychotic use
Proportion on neuroleptics (%)

Figure 1: Fossey et al 2006 BMJ 12 NH n=347
WHELD Pilot Study

Main aim:
• To find out the most effective combination of psychosocial treatments for residents to improve quality of life, reduce prescribing and reduce falls

Pilot Interventions:
• Person Centred Care
• Social Intervention and
• Pleasant activities
• Antipsychotic Review
• Exercise
Efficacy of treating pain to reduce behavioural disturbances in residents of nursing homes with dementia: cluster randomised clinical trial

Bettina S Husebo postdoctoral fellow¹, Clive Ballard professor², Reidun Sandvik registered nurse¹, Odd Bjarte Nilsen statistician³, Dag Aarsland professor⁴

¹Department of Public Health and Primary Health Care, University of Bergen, 5020 Bergen, Norway; ²Wolfson Centre for Age-Related Diseases, Wolfson Wing and Hodgkin Building, Guy’s Campus, Kings College, London SE1 1UL, UK; ³Department of Psychiatry, Stavanger University Hospital, 4011 Stavanger, Norway; ⁴Karolinska Institute, Department of Neurobiology, Care Sciences and Society, Karolinska Institute-Alzheimer Disease Research Center, Novum, Stockholm, Stavanger University Hospital, Department of Psychiatry, Stavanger, Norway, and University of Oslo, Oslo, Norway
Table 3 | Comparison of Cohen-Mansfield agitation inventory (CMAI) total score between control and intervention (stepwise protocol for treatment of pain) groups using repeated measures analysis of covariance (ANCOVA)*

<table>
<thead>
<tr>
<th>Week</th>
<th>Mean (SD) CMAI total score</th>
<th>Effect of intervention on CMAI total†</th>
<th>P value</th>
<th>Intracluster correlation coefficient‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Control group: 56.2 (16.1), n=177</td>
<td>Intervention group: 56.5 (15.2), n=175</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>53.9 (17.0), n=161</td>
<td>52.0 (19.5), n=158</td>
<td>-3.6 (-0.5 to -6.7)</td>
<td>0.022</td>
</tr>
<tr>
<td>4</td>
<td>52.5 (16.3), n=160</td>
<td>49.4 (19.0), n=148</td>
<td>-4.1 (-0.9 to -7.4)</td>
<td>0.012</td>
</tr>
<tr>
<td>8</td>
<td>52.8 (16.8), n=157</td>
<td>46.9 (18.7), n=147</td>
<td>-7.0 (-3.7 to -10.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>12</td>
<td>52.5 (16.0), n=152</td>
<td>50.3 (20.3), n=142</td>
<td>-3.2 (0.1 to -6.4)</td>
<td>0.058</td>
</tr>
</tbody>
</table>

*Baseline score as covariate and least squares weighted by number of patients within cluster; P value from multivariate test of intervention was 0.002, and cross effect between week and intervention was <0.001.
†Variable estimate by week of effect of intervention on CMAI score from estimated model.
‡Proportion of total variance between clusters, and measured within framework of ANCOVA.
### Table 5: Comparison of mobilisation-observation-behaviour-intensity-dementia-2 (MOBID-2) pain scale total score between control and intervention (stepwise protocol for treatment of pain) groups using repeated measures analysis of covariance (ANCOVA)*

<table>
<thead>
<tr>
<th>Week</th>
<th>Control group</th>
<th>Intervention group</th>
<th>Estimate (95% CI)</th>
<th>P value</th>
<th>Intraclass correlation coefficient‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>3.7 (2.5), n=163</td>
<td>3.8 (2.7), n=164</td>
<td></td>
<td></td>
<td>0.094</td>
</tr>
<tr>
<td>2</td>
<td>3.5 (2.4), n=159</td>
<td>2.9 (2.5), n=152</td>
<td>-0.7 (-0.4 to -1.1)</td>
<td>&lt;0.001</td>
<td>0.070</td>
</tr>
<tr>
<td>4</td>
<td>3.3 (2.4), n=155</td>
<td>2.7 (2.2), n=146</td>
<td>-0.8 (-0.4 to -1.2)</td>
<td>&lt;0.001</td>
<td>0.059</td>
</tr>
<tr>
<td>8</td>
<td>3.5 (2.6), n=154</td>
<td>2.3 (2.1), n=145</td>
<td>-1.3 (-0.8 to -1.7)</td>
<td>&lt;0.001</td>
<td>0.082</td>
</tr>
<tr>
<td>12</td>
<td>3.5 (2.5), n=151</td>
<td>2.9 (2.6), n=140</td>
<td>-0.8 (-0.3 to -1.2)</td>
<td>0.001</td>
<td>0.139</td>
</tr>
</tbody>
</table>

*Baseline score as covariate and least squares weighted by number of patients within cluster; P value from multivariate test of intervention was <0.001, and cross effect between week and intervention was 0.009.

†Variable estimate by week of effect of intervention on MOBID-2 from estimated model.

‡Proportion of total variance between clusters, and measured within framework of ANCOVA.
DOMINO. Estimates of mean NPI and GHQ-12 by visit and treatment arm Howard et al NEJM 2012
Best Practice Guide: Treatment and care for behavioural and psychological symptoms

• Developed in partnership with Department of Health
• Led by
  – Clive Ballard
  – Alistair Burns
  – Anne Corbett
• Advisory group: Sube Banerjee; Nina Barnett; Donald Brechin; Peter Connelly; Jane Fossey; Clive Holmes; Julian Hughes; Gill Livingston; Deborah Sturdy; Simon Wright
• Focus on preventing and managing BPSD
• Now available as consultation document
Best Practice Guide: Treatment and care for behavioural and psychological symptoms

- **Green** – No symptoms. Simple preventative measures
- **Amber** – Mild or moderate symptoms. Low intensity, general interventions
- **Red** – Severe symptoms. Specific interventions and guidance for antipsychotic use
Best Practice Guide: Prevention

• Emphasis on person-centred care
  – Care plan
  – Involvement of carers
  – Consider physical environment

• Importance of medical review
• Understanding of dementia
• Recognition of triggers
• Involvement of family and / or carers

Two thirds of people in care homes have dementia

Management of Alzheimer’s Dementia
New Guidance and the Changing NHS

2011
Best Practice Guide: Watchful Waiting

- Ongoing assessment and non-drug treatments

- Person-centred care
  - Positive social interaction
  - Life story book
  - Short, frequent conversations

- Clinical care plan
  - BPSD usually improve after four weeks with no treatment

Watchful waiting is a proactive process over four weeks to assess if contributing factors and non-drug treatments should be considered. Assessing symptoms and reviewing care over the four weeks will determine if treatments are needed. A high proportion of people with behavioural and psychological symptoms experience the four weeks with no specific treatment. Watchful waiting is effective therapeutic practice unless there is severe reactivity. This guide will give you some ideas for assessment, and further improve the likelihood of a favourable outcome.

Clinical checklist

Watchful waiting

1. Clinical checklist
   - Ongoing assessment and non-drug treatments

2. Person-centred care
   - Positive social interaction
   - Life story book
   - Short, frequent conversations

3. Clinical care plan
   - Suggested for four weeks when symptoms emerge
     - BPSD usually improve after four weeks with no treatment

Management of Alzheimer’s Dementia
New Guidance and the Changing NHS

2011
Best Practice Guide: Specific Interventions

- For severe BPSD
- Tailored psychosocial interventions
  - Improving social interactions
  - Promoting positive activities and exercise
  - Brief Psycho-social therapies
  - Specialist referral (e.g. ABC)
- Pharmacological options
  - Depression – sertraline, Citalopram
  - Sleep disturbance
    - Analgesic
- Antipsychotic
  - Risperidone for 6 weeks

Specific interventions

Psychosocial interventions
Psychosocial interventions are more tailored, systematic approaches to person-centred care (as those outlined earlier in watchful waiting).

The following steps should be taken to develop a Specific intervention care plan (8):
- Complete medical and mental health review including 6: Clinical checklist
- Consider all aspects of person-centred care (see 4: Watchful waiting guidance)
- Consult with family or carers on the best approach
- Design specific interventions (the brief and simple approaches below have been shown to be effective and can be administered by care staff with support from any clinician)
- Consider whether care staff require specific dementia training (person-centred care training for staff can reduce antipsychotic use and improve agitation).

Improving social interactions
Brief psychosocial therapies help to engage people in ways that they find interesting and enjoyable. These should generally include 30–35 minutes of daily one-to-one conversation or activity based on the person’s interests, hobbies, history and ability, and feedback from their care and/or family.

Pain is one of the most common causes of BPSD

Management of Alzheimer’s Dementia
New guidance and the changing NHS
Best Practice Guide: Monitoring and Review

**Antipsychotics prescription**

Safety monitoring guidance and Monitoring plan

Antipsychotic drugs are known to be harmful. It is vital that any person prescribed these drugs commonly experiences some degree of progression of symptoms. This plan shows a monitoring plan for a person with dementia when a prescription of antipsychotics is made.

Adverse effects of antipsychotic drugs

The most important adverse effects associated with antipsychotics are postural, falls, delirium, chest infections, olfactory deficits, deep vein thrombosis, pulmonary embolism, cardiac arrhythmia and stroke. (highest risk in first four weeks of treatment)

Antipsychotics are also associated with increased mortality in the long term (related to premature cardiovascular and non-cardiovascular events) which can be caused by over-sedation and dehydration.

**Antipsychotics prescription**

Review chart

This chart should be completed for any patient prior to discontinuation or continued prescription of an antipsychotic. All prescriptions should be reviewed at six weeks (recommended) or 12 weeks.

<table>
<thead>
<tr>
<th>Name:</th>
<th>Current diagnosis:</th>
</tr>
</thead>
</table>

**Current prescription:**

**What are the symptoms?**

- How severe are they? 
  - Mild 
  - Moderate 
  - Severe 

**What are the risks:**

- to the person
- to others

- How distressed is the person?
- How would the person benefit if these risks were addressed?

**Clinical treatment decision:**

- Discontinue 
- Continue prescription 
- Details:

- What would be a sign of ongoing improvement or stabilisation for this person?
- What is the plan for further review?

- If antipsychotics are discontinued, what additional support is needed for the first four weeks of discontinuation?

| Signed: | Date: |

- **Side effects more severe in long term use**
- **Side effects improved through simple monitoring**
  - Sedation
  - Fluid intake
  - Chest infection
- **All antipsychotic prescriptions reviewed at 12 weeks**
  - Discontinuation is default
  - Discontinue by tapering for high doses
- **Return to non-drug interventions**
For access to the guide and to download, go to:
http://www.alzheimers.org.uk/bpsdguide

To access the reference list that supports the recommendations, go to:
Conclusions – the Evidence Base

• Antipsychotics have a focussed but limited role in the short term management of severe aggression and psychosis. The best evidence base for pharmacological treatment is for short term treatment with risperidone as a treatment for aggression, but we are currently overprescribing, the longer term efficacy is limited and the serious adverse risks are considerable.

• The evidence base supports the value of simple non drug interventions and intensive staff training in care homes.

• Recent evidence re-inforces the potential value of analgesia.