Screaming in Dementia

Nages Nagaratnam, MD, FRCP, FRACP, FRCPA, FACC, Consultant Physician in Geriatric Medicine, Formerly of Department of Geriatric Medicine, Blacktown-Mt-Druitt Health, Blacktown, NSW, AUS.

Kujan Nagaratnam, MB, FRACP, Consultant Physician in Geriatric Medicine, Department of Geriatric Medicine, Blacktown-Mt Health, Blacktown, NSW, AUS.

Introduction
Several behaviours have been described in dementia and they may be so varied, and in some instances so poorly defined, that their definition continues to be debated. It has been recommended that behavioural disturbances be replaced by the term behavioural and psychological symptoms of dementia (BPSD).1 BPSD form a substantial part of the morbidity associated with dementia; they are more troublesome to the patient and caregivers than the cognitive symptoms, and should be the focus of greater clinical attention.

In a 1907 landmark single case study, Alois Alzheimer2 described a 51-year-old woman suspicious of her husband; she also exhibited rapidly developing and increasing memory impairment, disorientation, the urge to drag objects here and there, and loud screaming. She greeted her doctor as a visitor and on occasion screamed that he wanted to cut her open. She often screamed for hours in a horrible voice and burst into loud screams each time she was approached to be examined. She was paranoid and had delusions of sexual abuse and hallucinations. Although screaming had been recorded as an integral part of dementia from the earliest times it had received little attention in its own right until more recently.

This article outlines an approach to screaming and its possible neuroanatomical substrates and neurotransmitter systems. The ultimate basis for discussion will be the strategies available for management of this challenging behaviour.

Key words: screaming, disruptive vocalization, dementia, limbic system, frontal-subcortical circuitry

Screaming is widely viewed as a common behavioural disturbance in dementia. It influences the performance in daily life of the patient, adds to the burden and embarrassment experienced by the caregiver and the frustrations encountered by the treating physician, and is a decisive factor for institutionalization. This article outlines an approach to screaming and its possible neuroanatomical substrates and neurotransmitter systems. The ultimate basis for discussion will be the strategies available for management of this challenging behaviour.

Key words: screaming, disruptive vocalization, dementia, limbic system, frontal-subcortical circuitry
In many instances there is no cause for the screaming, but often screaming is associated with cognitive impairment, depressed affect, social networks of poor quality, severe functional impairment, a response to social isolation, emotional reactions of nursing staff, communication difficulties, or use of psychotropic medication. Restriction in the ability to communicate in dementia patients may be a determinant of the disturbed behaviour. It is well known that these patients express their frustration and anger by agitation or by “acting out” behaviours. Nagaratnam et al. found no correlation between impaired communication and disturbed behaviour. Screaming could be secondary to physical problems (pain, infection) or activity related (to get dressed or to have a bath).

Acute delirium (acute confusional state) is a common complication of medical illnesses; it may also emerge in the course of dementia and may be the presenting symptom of dementia. Furthermore, these patients often manifest the behavioural and psychological symptoms seen in dementia. Not only is acute delirium an important cause of increased morbidity and mortality in dementia, it is often missed because of the difficulty in diagnosis.

**Subsyndromes**

Several investigators have proposed that behavioural disturbances such as screaming, verbal outbursts, agitation, verbal and physical aggression, and wandering are a continuum of severity of hyperactive behaviour. This is the view adopted in this article. Vocalization is associated with other disturbed behaviours. In one study of 12 dementia patients exhibiting noise making, 10 exhibited aggression. There was no difference in the prevalence of aggression across the diagnostic groups (that is, dementia patients with Alzheimer’s disease, multi-infarct dementia, and mixed). In a study of 91 dementia-related behavioural changes in 12 dementia patients, 44% of the patients had only one behavioural disturbance while 53% exhibited two or more. Apart from aggression, many dementia patients have psychotic symptoms; there is considerable evidence on the cause of behavioural disturbance linking aggression to psychotic manifestations. In dementia patients, the behavioural symptoms often associated with screaming are aggression, psychosis, and depressive symptoms. Alzheimer’s dementia consists of a full panoply of screaming, paranoid delusions, hallucinations, memory impairment, disorientation, and bizarre behaviour. Among patients with screaming, four symptom clusters or sub-syndromes emerge, namely (i) screaming with verbal aggression, (ii) screaming and motor restlessness, (iii) screaming with psychotic symptoms, and (iv) screaming with depressive symptoms. It would be more useful to consider clusters of symptoms rather than individual behaviours to correlate with the neurochemical measures, and the treatment may best be directed towards them rather than towards the individual behaviour.

**Neuroanatomic Correlates**

The frontal lobe is closely associated with human behaviour. The frontal-subcortical circuits provide a comprehensive framework for understanding the anatomy, biochemistry, and pharmacology of behaviour. Alexander et al. proposed five parallel circuits connecting discrete lesions in the frontal lobes with specific subregions of the striatum, globus pallidus, and thalamus (Figure 1). Hyperactivity, disinhibition, impulsiveness, and distractability are characteristic behaviours associated with orbital frontal lesions. Lesions of the dorsal medial nucleus of the thalamus could give rise to manifestations similar to prefrontal lesions. The frontal lobe and the frontal-subcortical circuitry play an important role in the occurrence of aggression and agitation. It is well accepted that the temporal lobe and the limbic system are closely related to a number of psychological syndromes. Delusional syndromes are known to involve the temporal lobe, especially the medial temporal-limbic structures. Alexander et al. have expanded the frontal subcortical circuitry to encompass the limbic system.

The neurogenic pathways that control mood and behaviour are associated with the neurotransmitter systems. Neurotransmitters such as serotonin, dopamine, and acetylcholine have a modulatory role. Serotonin has been associated with disturbed behaviours. While there is considerable variation in the presentation of noise making patients, the clinical features seem to correspond to the functional anatomy of the brain regions affected rather than to the pathology. Noise making may be a single syndrome with different locations and with different clinical presentations.

It may be asked how similar neuroanatomic changes could give rise to syndromes whose features are not uniform. According to Cummings the occurrence of different psychological syndromes could be influenced by several factors, including genetic constitution, age of onset, personality characteristics, early life experiences, and exact location and extent of the lesions. Environmental

<table>
<thead>
<tr>
<th>Screamers</th>
<th>Talkers</th>
<th>Mutterers</th>
<th>Singers</th>
</tr>
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<tbody>
<tr>
<td>Screaming</td>
<td>Perseveration</td>
<td>Muttering</td>
<td>Singing</td>
</tr>
<tr>
<td>Yelling</td>
<td>Palilalia</td>
<td>Mumbling</td>
<td>Chanting</td>
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<tr>
<td>Swearing</td>
<td>Echolalia</td>
<td>Grunting</td>
<td></td>
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<tr>
<td>Shouting obscenities</td>
<td>Incessant talking</td>
<td>Weird noises</td>
<td></td>
</tr>
<tr>
<td>Crying</td>
<td>Continuous chatter</td>
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</tbody>
</table>

**Table 1: Vocalization Subtypes**
provocations might also precipitate behaviour by evoking aggressive responses in patients whose threshold for agitation is affected.9

**Management**

Screaming is widely viewed as a common behavioural disturbance in dementia that poses difficult dilemmas in management. It is difficult to envisage which neurotransmitter system is involved in noise making and it is likely there may be more than one. This may be the reason why there is no effective therapy. Despite these caveats, there are areas of interest to those interested in approaches to managing screaming. As stated above, treatment might best be directed towards syndromes rather than toward individual behaviours.31 Agitation and aggression appear to be prominent features accompanying screaming. Aggressive behaviours are related to disordered frontal-subcortical circuitry and have been linked to the dysfunction of the serotonergic neurotransmission. Conditions that may be characterized by brain serotonergic dysfunction should benefit by the use of drugs with apparent serotonergic selectivity.4,16

There is no single approach that will solve the problem. Instead, a multimodal intervention is needed; nonpharmacological approaches must precede or accompany pharmacological therapies, and are often more effective.

The behavioural and psychological symptoms in dementia patients could be intrinsic to the disorder or can occur across multiple diagnoses: for example, with primary psychiatric illness, chronic alcoholism, or cerebral disorder (cerebral infarction, cerebral tumour, normal pressure hydrocephalus). Figures 2a and 2b illustrate an algorithm describing the steps in recognition, diagnosis, and treatment of behavioural and psychological symptoms.

**Nonpharmacological Intervention**

Nonpharmacological strategies for managing these patients should include a multifaceted approach considering physical, behavioural, environmental, and social factors (Table 2). It is important to identify, minimize, and avoid stressors (for instance, physical stressors such as pain or any other medical illness); to reduce overwhelming stimulation (e.g., too many people around or other provoking situations); and avoid too little activity (e.g., when the patient needs more animation). Behavioural approaches include such strategies as avoiding arguing and confrontation; touching thoughtfully and using a gentle, calm, and reassuring voice; avoidance of physical restraints; and engaging in pleasure-related activities.

There are other adjunctive approaches such as using bright light, aromatherapy, white noise, pet therapy, and music, but none have been evaluated using group study methodology for screaming patients. However, Ballard et al.,26 in a double-blind, placebo-controlled trial, demonstrated that aromatherapy with...
lemon balm was a safe and effective treatment for dementia-related agitation. A recent controlled trial has shown that individualized music therapy reduced agitation but group classical music did not.27

Environmental manipulation may also be helpful. Education of the caregiver and change in the caregiver’s behaviour, will in turn improve the patient’s behaviour as necessary. When behavioural and environmental approaches fail, medications may be helpful as an adjunct.

**Pharmacological Intervention**

In treating screaming, some drug classes should be favoured for initiation in certain subsyndromes because they have evidence of benefit in particular coexisting conditions (e.g., aggression, psychotic symptoms, and depression) (Table 3). Medications are not sufficient to terminate these behaviours, but they can often reduce the severity, transitioning them from disturbed to acceptable. Older adults are particularly sensitive to side effects from neuroleptics, and those drugs with sedative effects frequently cause hypotension. It is important to review the side effect profiles of the common medications used in screaming and the general principles of prescribing them (Tables 3 and 4).

**Antidepressants**

Screaming can be associated with depression5 and there is a favoured association with certain types of dementia such as vascular dementia. Depressive symptoms are common in dementia but their cause remains unclear. Selective serotonin reuptake inhibitors (SSRIs) are better tolerated in older adults than more traditional tricyclic antidepressants or monoamine oxidase inhibitors. Citalopram has been shown to be effective for depression and behavioural disturbance in dementia.17,18

Trazodone, a trizolopyridine derivative, inhibits the neural response of serotonin19 and has been used in continuous and repetitive screaming patients with effect.5,16 Pasion and Kirby16 described an 84-year-old woman with continuous screaming who had been on a number of medications with little or no effect. They were ceased and the patient commenced on trazodone, which selectively inhibits neuronal reuptake of serotonin. The dose was increased over the next two weeks to 300mg per day with no adverse effects. Two weeks after starting trazodone the screaming stopped and verbal outbursts were limited to intermittent calls for assistance. Greenwald et al.4 described an 82-year-old woman with repetitive screaming and table and head banging with moderately advanced dementia. The medications haloperidol, oxazepam, tranylcypromine, and thiothixene provoked unwanted side effects. A trial with increasing doses of trazodone up to 300mg at three weeks was without side effects. The head banging decreased, her mood improved, and screaming was no longer constant. She was then commenced on 1g/day L-tryptophan as an adjunctive therapy, which was titrated to 2.5g daily, and within two weeks the screaming and echolalia almost stopped. They concluded that screaming and banging in demented patients may be a related symptom responsive to serotonergic modifiers. Single-case studies such as these in relation to treatment success or failure are useful and contribute to an assumption that can be validated by group study.

The doses are given in Table 3. In general, it is advisable to start on a small dose and to increase it to the target dose that is likely to be effective for a typical patient.

Fluoxetine should be avoided in those with agitation and anxiety.

**Antipsychotics**

The typical antipsychotic haloperidol is commonly prescribed but has a high
Screaming in Dementia

**Figure 2a:** Behavioural and Psychological Symptoms (BPS)—Their Recognition, Diagnosis, and Management, Part One

- **Your patient has**
  - Behavioural and psychological symptoms: assess nature, frequency, and severity

- **SUDDEN ONSET**
  - YES
  - see Figure 2b
  - NO

- **Convincing evidence of BPS:** agitation, aggression, motor restlessness, delusions, hallucinations, etc.
  - YES
  - Decline in memory, intellect, personality, etc.
  - NO
  - History of mental illness
  - NO
  - History of alcoholic disease
  - NO
  - Focal neurological signs

- **Apathetic**
  - YES
  - DEMENTIA assess severity, etc.
  - NO

- **Depression**

**Are there concomitant illnesses, medications, environmental factors, etc.?**

- YES
  - Investigate cause and treat accordingly
  - NO

**Determine type of dementia**

- **History of strokes, labile mood, stepwise deterioration, hypertension, etc.**
  - NO
  - Multi-infarct Dementia
  - YES

- **Fluctuating periods of confusion, hallucinations, extrapyramidal signs, unsteadiness, falls**
  - NO

- **Disinhibition or overactivity, unconcerned reduced speech output, clowning, singing, etc.**
  - NO

- **Insidious onset progressive, memory loss, personality change, exclusion of other dementias**
  - NO

- **Symptoms of Alzheimer’s and vascular dementias**
  - YES

**Lewy-Body Dementia**

- YES

**Fronto-temporal Dementia**

- YES

**Alzheimer’s Dementia**

- YES

**Mixed Dementia**

- YES

**IDENTIFY** behaviour to be targeted

**Treatment—see Table 3**
Screaming in Dementia

risk of extrapyramidal symptoms (EPS), particularly in older adults. In patients with Lewy Body dementia, antipsychotics can give rise to disastrous medical consequences—such as tardive dyskinesia and possibly sudden death—and therefore must be avoided altogether. There is evidence that antipsychotics have inhibitory effects on cholinergic, dopaminergic, and histaminergic neurotransmission and thus could give rise to deleterious cognitive effects in some older patients.28

Atypical antipsychotic medications have been used to manipulate the serotonin system and modulate vocalizations.21 The newer atypical antipsychotics are a heterogenous group of drugs that may be associated with lower risk of EPS and are better tolerated.

Frenchman and Prince29 reported their experience in a retrospective study of 186 patients with diagnoses of Alzheimer’s dementia, senile dementia (not otherwise specified), or organic brain syndrome. In all, 60 were treated with risperidone (mean 1mg/day), 83 with haloperidol (mean 2mg/day), and 43 with thioridazone (mean 33mg/day). The target behaviours were violence (74), shouting (31), delusions (26), paranoia (19), pacing (3), and mixed behaviours (33).

**Figure 2b: Behavioural and Psychological Symptoms (BPS)—Their Recognition, Diagnosis, and Management, Part Two**

<table>
<thead>
<tr>
<th>Behavioural and psychological symptoms: assess nature, frequency, severity, etc.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sudden Onset</strong></td>
</tr>
<tr>
<td>NO</td>
</tr>
<tr>
<td>see Figure 2a</td>
</tr>
<tr>
<td>YES</td>
</tr>
</tbody>
</table>

**Convincing evidence of confusional state**—short history, abrupt onset, severity fluctuates, lucid periods, recent memory loss, agitation, mood lability, psychiatric phenomena, distractability, etc.

**Patient with no pre-existing dementia**

**Assess history, physical neurological examinations, psychometric testing, laboratory testing, etc.**

**Acute Confusional State**

**Patient with pre-existing dementia**

**Determine cause**

- Infection—chest, urinary tract drugs, liver, renal, cardiac failure, etc.
- Any focal neurological signs

**Infective, toxic, and metabolic confusional syndromes**

- Intracranial infection—cerebral tumour, normal pressure hydrocephalus, etc.

**Head Injury**

**History of accident**

**History of epilepsy**

**Alcohol binge withdrawal**

**Management: Assess risk, treat underlying cause or causes, pharmacological intervention if required**
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**Table 3: Profile of Common Medications Used in Screaming**

<table>
<thead>
<tr>
<th>Drug category</th>
<th>Associated symptoms with screaming</th>
<th>Daily dose (oral) mg*</th>
<th>EPS</th>
<th>AC</th>
<th>Sedation</th>
<th>Side Effects</th>
<th>Weight loss</th>
<th>Agranulocytosis</th>
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<tbody>
<tr>
<td><strong>Antidepressants</strong></td>
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<tr>
<td>SSRIs</td>
<td>Depression, agitation, aggression</td>
<td></td>
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<tr>
<td>Sertraline</td>
<td>25–100</td>
<td></td>
<td></td>
<td></td>
<td>++</td>
<td>No data</td>
<td>No data</td>
<td>0</td>
</tr>
<tr>
<td>Citalopram</td>
<td>10–20</td>
<td></td>
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<tr>
<td>Fluoxetine</td>
<td>10–40</td>
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<tr>
<td>Trazodone (SARI)</td>
<td>50–200</td>
<td>0</td>
<td>0</td>
<td></td>
<td>++</td>
<td>No data</td>
<td>No data</td>
<td>0</td>
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<tr>
<td><strong>Antipsychotics</strong></td>
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<tr>
<td>Atypical</td>
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<tr>
<td>Clozapine</td>
<td>Psychosis</td>
<td>200–600</td>
<td>+</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>Agitation</td>
<td>5–20</td>
<td>+</td>
<td>++</td>
<td>+++</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Risperidone</td>
<td>Aggression</td>
<td>2–6</td>
<td>++</td>
<td>0</td>
<td>++</td>
<td>+++</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>Violence</td>
<td>300–750</td>
<td>+</td>
<td>+</td>
<td>+++</td>
<td>++</td>
<td>++</td>
<td>+</td>
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<tr>
<td>Typical</td>
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<tr>
<td>Haloperidol</td>
<td>2–7.5</td>
<td>+++</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
<td>++</td>
<td>+</td>
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<tr>
<td><strong>Anticonvulsants</strong></td>
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<td></td>
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<tr>
<td>Valproic acid</td>
<td>Aggression</td>
<td>100–1,000</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>Agitation, Hostility</td>
<td>200–600</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hepatotoxicity, sedation, drug interaction, and ataxia</td>
</tr>
<tr>
<td><strong>Cholinesterase Inhibitors</strong></td>
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<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Donepezil</td>
<td>Psychosis</td>
<td>5–10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Predominantly gastrointestinal: nausea, anorexia, cramps Nightmares</td>
</tr>
<tr>
<td>Rivastigmine</td>
<td>Aggression</td>
<td>1.5–6 b.i.d.</td>
<td></td>
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<tr>
<td>Galantamine</td>
<td>Depressive symptoms, activity disturbance</td>
<td>4–12 b.i.d.</td>
<td></td>
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<tr>
<td><strong>Other</strong></td>
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<tr>
<td>Beta-blocker</td>
<td></td>
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<td></td>
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<td></td>
<td>Hypotension, bradycardia, unapparent hypoglycemia in diabetes</td>
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<tr>
<td>Propanolol</td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td>Exacerbation of cognitive impairment, risk of falls</td>
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<tr>
<td>Benzodiazepines</td>
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</table>

0=normal; + = minimal; ++ = mild; +++ = moderate; +++ = severe; EPS = extrapyramidal symptoms; AC = anticholinergic; SSRI = selective serotonin reuptake inhibitors; SARI = serotonin-2 antagonists/serotonin reuptake inhibitor

* The higher dose shown is the target dose likely to be effective for a typical patient.

Target behaviours improved in 94% of patients given risperidone, 65% given haloperidol, and 67% given thioridazine. Treatment failures were more frequent on patients started on haloperidol and thioridazine as compared with risperidone; extrapyramidal side effects were reported in 7% of patients taking risperidone, 22% taking haloperidol, and 18% taking thioridazine. Safe and effective doses were readily achieved with risperidone.

**Anticonvulsants**

Older patients with dementia and behavioural agitation improved when treated with sodium valproate, and the proposed mechanism of action was via the alteration of the gamma-aminobutyric acid (GABA) system and not the serotonergic system.21

The use of carbamazepine in BPSD was positive in a study of its efficacy for agitation and aggression in dementia. Carbamazepine and valproic acid are increasingly used for BPSD, particularly in patients with cerebrovascular disease and dementia.30

**Acetylcholinesterase Inhibitors**

Cholinesterase inhibitors are generally prescribed in Alzheimer’s disease to slow the cognitive decline but have also been found to be effective in reducing agitation.35 In Lewy Body dementia, the psychosis and behavioural disturbances rapidly respond to cholinesterase inhibitors.22,23 In a single case study of a patient with Lewy Body dementia with severe visual hallucinations and behavioural problems, introduction of donepezil was associated with cognitive improvement and complete remission of behavioural symptoms.31

It has been postulated that cholinergic agents may have psychotropic activity through actions in the paralimbic cortex of the frontal and temporal lobes.24 A large double-blind placebo controlled trial examined the prevalence of behavioural symptoms in moderate to severe dementia and the effect of treatment with donepezil. The common symptoms were aberrant motor behaviour (53%), depression/dysphoria (52%), agitation/aggression (45%), and apathy/indifference (67%). At the end of 24 weeks on donepezil, the behavioural symptoms had improved.32

Similar results were seen in another study with donepezil in which treatment had a mildly positive effect on emotional/behavioural symptoms in Alzheimer’s Disease.33 However, more evidence for this use of cholinesterase inhibitors is needed and responses thus far have been variable.

**Benzodiazepines**

Physicians should be cautious in prescribing benzodiazepines for older patients as they can lead to increased confusion and falls, although they are sometimes useful in managing anxiety and agitation.

**Beta-Blockers**

Many of the patients who exhibit noise making have features attributable to orbital-frontal cortical dysfunction and the use of beta-adrenergic blocking agents (beta-blockers) has been found to be effective. Shankle et al.25 suggested low-dose propranolol in such patients.

**Evidence-Based Approach to Pharmacotherapy**

The decision to initiate drug therapy in dementia patients with behavioural disorders that can occur across multiple diagnoses is based on an assessment of the severity and nature of the symptoms, the type of dementia, and the failure to respond significantly to nondrug interventions. Some drug classes should be favoured for initiation in certain symptom clusters (subs syndromes) because they have evidence of benefit in coexisting conditions. The next step would be to select the individual drug within the class; this will be influenced by known efficacy and side effect profile.

Because of the multiple changes occurring with age, older adults need to be prudently treated following the admonition: “start low, go slow,” with even more caution in the very old. The management algorithm and profile of the common medications used are provided in Table 3 and Figure 3. These are individualized choices based on clinical data, but may not have the support of RCTs. If the response to a drug in the class is inadequate, substitute a drug from a different class; this will be influenced by known efficacy and side effect profile.

Because of the multiple changes occurring with age, older adults need to be prudently treated following the admonition: “start low, go slow,” with even more caution in the very old. The management algorithm and profile of the common medications used are provided in Table 3 and Figure 3. These are individualized choices based on clinical data, but may not have the support of RCTs. If the response to a drug in the class is inadequate, substitute a drug from a different class; this will be influenced by known efficacy and side effect profile.

**Conclusion**

Screaming in dementia is viewed as a common behavioural disturbance pre-
Screaming in Dementia

Identify the target behaviours plus coexisting behaviours

Ascertain type of dementia (AD, MID, FTD, LBD)**

Identify medical precipitants (medications, psychiatric conditions, and environment)

NONPHARMACOLOGIC INTERVENTIONS

Figure 3: Algorithm for the Management of Screaming

NR=No response; **AD: Alzheimer’s Disease; MID: Multi-infarct Dementia; LBD: Lewy Body Dementia; FTD: Frontotemporal Dementia

Preferred medications in the subsyndromes: anticonvulsants: sodium valproate/carbamazepine; antidepressants: citalopram/sertaline; antipsychotics: risperidone/olanzapine; cholinesterase inhibitors: donepezil/rivastigmine/galantamine
senting with difficulties in its management, as the neural mechanisms underlying screaming in dementia are still obscure. This article does not illuminate the issue but does indicate that screaming with verbal outbursts, agitation, aggression, and wandering is said to be a continuum of hyperactive behaviour.

Screaming is often associated with other behavioural disturbances, and the target behaviours are agitation/aggression, wandering, psychotic manifestations, and depressive symptoms. It would be useful to consider screaming as part of a symptom cluster, for treatment may thus be directed towards subsyndromes rather than towards the individual behaviour.

There is no single approach that will solve the problem. Instead, a multimodal intervention initiating in nonpharmacological and followed by pharmacological treatments is needed. The former remains the mainstay of management. Although reported as effective, it lacks the support of controlled trials. However, these non-drug interventions do improve the quality of life of patients in long-term care.

Medication is often required as an adjunct therapy. In treating screaming, certain drug classes are preferred for initiation in the different subsyndromes because there is evidence of benefit in particular coexisting symptoms. Other influencing factors are the severity of the screaming and the type of dementia. The drug classes commonly used are antidepressants, anticonvulsants, antipsychotics, and cholinesterase inhibitors. There is no single effective therapy, and treatment of screaming in dementia remains a challenge.

No competing financial interests declared.

References