



Primary Care Physicians Guide to BPSD

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Table of Contents

Introduction	4
Key Messages	6
BPSD	10
Incidence and prevalence	10
Variation with type of dementia	10
Particular Symptoms	11
Hallucinations	11
Delusions	11
Misidentifications	11
Depressed mood and other affective symptoms	12
Apathy and amotivation	12
Anxiety symptoms	12
Aggression	12
Disinhibition	13
Negativism	13
Intrusiveness and shadowing	13
Etiology	13
Differentiate etiology to guide therapy	13
Always rule out delirium	13
Differentiating BPSD	14
Laboratory and Imaging Examinations	16
Management Strategy	17
Ethical Issues	17
Therapeutic approach	18
Non-pharmacological Management	18
ABCs of Behavioral Management Strategies	18
Some useful non-pharmacological techniques	19
Pharmacological Management	20
Typical (Conventional) Antipsychotics	21
Atypical Antipsychotics	22
Antidepressants	22
Cholinesterase inhibitors	24
Memantine	24
Benzodiazepines	24
Mood Stabilizers	25
Buspirone	25
Beta-Blockers	25
Estrogens and Anti-androgen Treatments	26
Antihistamines	26
Appendix: Scales in BPSD	27
References and recommended reading	29

INTRODUCTION

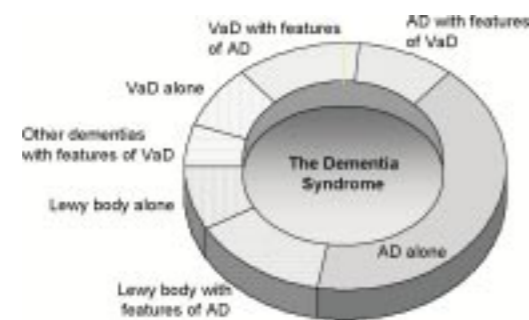
Although the predominant cause of the dementia syndrome is one of the several degenerative neurological diseases, it may also occur in other disturbances, some of which are reversible. Consequently, proper management of the syndrome requires case-by-case identification of the most probable etiology. In particular, those causes that may be reversible must be ruled out before treatment proceeds on the assumption of an underlying dementia.

Reversible causes of Dementia Syndromes

- Delirium (alone or with a dementia)
- Depression – Pseudo dementia
- Drugs
 - Alcohol
 - Recreational
 - Ethical medications

Notwithstanding the critical need to identify reversible cases of the dementia syndrome, the great majorities result from an underlying dementia. Although the majority of cases will be due to or have significant features of Alzheimer's disease (AD), it is still useful to recognize not only other types of dementia, but also the mixed nature of many cases.

The major dementias



Despite extensive research, the factor(s) precipitating neurological degeneration remain obscure and efforts to avert, arrest or slow progression of disease have to date had minimal success.

Cognitive deficits due to neurological degeneration remain the principal and most researched features of dementia. However, dementia is also characterized by clinically important behavioral and psychological symptoms (BPSD). There is increasing recognition that effective clinical management of the behavioral and psychological symptoms—BPSD—is central to the quality of life for both carers and patients.

BPSD are most distressing and disruptive for both patients and carers and if not effectively managed are most likely to lead to institutional care. Recognition of the correlation between progression of dementia

and manifest BPSD has also been used in conjunction with other characteristics for clinical staging of the disease. A list of common BPSD includes:

Common Behavioral and Psychological Symptoms of Dementia *

Behavioral	Psychological
Activity Disturbances	Affective Disturbances
Agitation, Restlessness Hyperactive Wandering Inappropriate activity Cognitive abulia	Anxiety Irritability Depressive symptoms Major Depression Emotional lability
Aggression	Apathy
Verbal Physical	Delusions and misidentification syndromes
Appetite and eating disorders	People are hiding or stealing things Paranoid, Suspiciousness
Disturbed diurnal rhythms Sleep / wake	Long-time home is not home Spouse / Caregiver
Socially improper behaviors	Is an impostor Is unfaithful Has abandoned him / her Dead relatives / acquaintances are alive
	Hallucinations
	Visual Auditory Olfactory Touch (haptic)

* Brief descriptions for most terms will be found in the Glossary



KEY MESSAGES

The following two-sided pull-out summarizes the main issues in the management of BPSD and can be used as an aide memoire and supplied to others engaged with patients and caregivers. Within the pull-out are specific references to pages in this guideline that give further information about related clinical issues and, in turn, refer to original publications that underpin the information and advice given here.

In general, primary practitioners and their staff engaged in care of patients with BPSD should remember:

- ▶ Behavioral and psychological symptoms of dementia (BPSD) are very common (up to 90% for some symptoms).
- ▶ BPSD cause significant distress to patients, their families and caregivers and if not effectively managed are likely to precipitate early institutional care.
- ▶ Interventions for BPSD are as or more effective as those for cognitive symptoms of dementia.
- ▶ All physicians and staff involved in "front-line" care of demented patients should be familiar with clinical manifestations and management of BPSD.
- ▶ Clinicians should try to find the cause(s) of BPSD before embarking on intervention.

▶ Non-pharmacological interventions should be considered for all severities of BPSD; however, the chances of their success may be greater in those with mild to moderate BPSD.

▶ In moderate to severe BPSD partially or unresponsive to non-pharmacological interventions alone, adjunct medication may be considered.

▶ When considering medication, physicians should first look for defined psychiatric syndromes, such as depression, psychosis, or anxiety to guide initial prescription.

▶ When selecting pharmacological interventions:

- Select to minimize interaction and side-effect potential and maximize chances of efficacy.
- Look for agents with efficacy confirmed in controlled clinical trials
- Start with 1/3 to 1/2 the usual adult starting dose
- Start with one drug at a time
- Monitor frequently for efficacy and side effects
- On the basis of effects, titrate dose as required or change selection
- Do not withdraw previously started nonpharmacological interventions

Primary Care Management of the Behavioral and Psychological Symptoms of Dementia (BPSD)

The dementia syndrome is generally associated with neurodegenerative diseases, but may also occur due to major depression (pseudodementia), drug or alcohol abuse and some pharmaceuticals. It is critical for proper clinical management that the probable cause is identified.

BPSD occur also in other disorders, particularly psychoses and affective disease, and again determination of the underlying cause is critical for proper management

- All physicians and staff involved in "front-line" care of demented patients should be familiar with the common manifestations of BPSD and their clinical management.
- BPSD may be the presenting symptom in dementia. Primary care physicians should be alert to this possibility in patients at risk due to age or family history.
- BPSD are very common (in up to 90% of cases of dementia) are most distressing to patients, caregivers and families. If unmanaged, often induce excess disability and precipitate institutional care.
- Interventions in BPSD are often more effective and helpful than those in cognitive symptoms of dementia.
- All physicians and staff involved in "front-line" care of demented patients should be familiar with identification and clinical management of BPSD.
- Non-pharmacological interventions should be considered first with mild or moderate BPSD.
- In moderate to severe BPSD or cases partially or unresponsive to non-pharmacological interventions alone, adjunct medication should be considered.
- When prescribing, physicians should:
 - Use defined psychiatric syndromes (depression, psychosis, anxiety) to guide initial treatment.
 - Select medications to minimize interaction and side-effect potential and maximize chances of efficacy.
 - Look for evidence of efficacy in controlled clinical trials
 - Start with 1/3 to 1/2 the usual adult starting dose
 - Monitor frequently for efficacy and side effects. Titrate slowly on the basis of effects on target symptom(s) and adverse effects. If adequate dosage for an adequate time fails to yield desired result or is associated with adverse effects, be prepared to change selection.

Some Behavioral and Psychological Symptoms of Dementia (BPSD)	
Behavioral	Psychological
Activity Disturbances	Affective Disturbances
Agitation, Restlessness	Agitation
Hyperactive	Anxiety
Wandering	Depressive symptoms / Major Depression
Inappropriate Activity	Emotional lability/ Elation
Cognitive Abulia	Irritable
Aggressive – verbally, physically	Apathy
Appetite and eating disorders	Delusions
Disturbed diurnal rhythms	People are hiding or stealing things
Wake/ Sleep disorder	Long-time home is not home
Sundowning	Spouse / Caregiver
Socially improper behaviors	Is an Impostor
	Is unfaithful
	Phantom boarder
	Mirror image is another person
	Visual
	Auditory
	Olfactory
	Touch (haptic)

General Giudelines for Management of BPSD		
Probable aetiology	severity	Suggested approach to management
Medical cause? (e.g., UTI,metabolic,constipation, etc)?		Usual clinical management considering patient's age
Delirium?		Rule out or manage underlying cause
Environmental cause or disturbing events?	Mild / Moderate	As practical, alter environment. Reassure in advance of event(s). Maintain calm, stable environment
	Severe	Consider low-dose prn pharmacotherapy
Identifiable psychiatric syndrome	Mild / Moderate	Use non-pharmacological methods. If necessary, augment therapy with drugs appropriate for syndrome, patient's age, condition and expected sensitivity to dose
	Severe	Pharmacological therapy augmenting non-pharmacological interventions
No identified or unmanageable cause, no specific syndrome?	Mild / Moderate	Try non-pharmacological interventions. Response inadequate, augment with minimal pharmacotherapy based on the nature of observed symptoms
	Severe	Non-pharmacological intervention augmented with empirical pharmacotherapy based on nature of observed symptoms

Guidelines for Use of Pharmacological Agents in BPSD	
Agent	Application (page reference for details)
Any agent	<p>Patients with dementia and elderly are often hyper-responsive and/or show less predictable effects of pharmacotherapy. Therefore:</p> <ul style="list-style-type: none"> • Always initiate treatment with 1/3 to 1/2 of the usual adult dose • Review frequently, titrate for desired and adverse effects • If results are sub-optimal, be prepared to switch to a different agent or class of agents
Antipsychotics: Typical Atypical	Psychotic symptoms—e.g., delusions, hallucinations, paranoia Agitated Aggressive
Antidepressants SSRIs, SNRIs, avoid anticholinergic TCAs	Depressive / depressed Anxious (Antidepressants with anxiolytic effects) Agitation, moderate / severe psychotic symptoms
Cholinesterase inhibitors	Cognitive deficits Variable findings, but possibly useful in some BPSD
Benzodiazapines	Premedication for anxiety-inducing events; sleep / wake problems Significant adverse effects; Long-term use discouraged
Mood stabilizers Anticonvulsants	Limited evidence of efficacy in BPSD and adverse effects recommend against use outside specialist care
Other drugs	<p>Buspiron: limited data, not available in Europe Gabapentin: limited data, possible use in anxiety, agitation Beta-blockers: limited data, not recommended outside specialist units Hormones: Not recommended outside specialist units Antihistamines: contra-indicated</p>



BPSD

Anyone treating the elderly will be familiar with the distress and despair felt by those caring for a formerly intelligent and accomplished individual who now wanders out of home at night, screams when bathed, or wonders when a long-dead parent or friend is coming to visit. Families may be irrevocably split attempting to cope with the grandmother who accuses her dutiful daughter-in-law of stealing her clothes and suffers from terrifying hallucinations. These symptoms are often a prominent and the most disruptive part of the dementia syndrome. All primary care staff involved in the management of patients with dementia should be familiar with the range of BPSD and their treatment

BPSD can be categorized in several ways, which for clinical purposes can be divided into behavioral and psychological. Likewise, the level of detail can be debated and the list on page 4 includes both "headline" symptoms, such as "delusions," together with some of the more common examples.

From a clinical point of view, another useful way to classify these symptoms is in terms of analogous psychiatric syndromes; e.g., delusions, hallucinations and paranoia can be viewed as psychotic symptoms while depressed mood, apathy and amotivation can be considered Depressive symptoms. Similarly, psychomotor agitation may be considered a symptom of anxiety or depression. Such considerations may be useful in choosing among options for intervention.

INCIDENCE AND PREVALENCE

During the course of dementia, one or more clinically significant BPSD are likely in up to 90% of patients.

The incidence and prevalence of specific BPSD vary widely with the type and stage of the dementing disease and whether patients have concurrent somatic comorbidity or are institutionalized. Consequently, reported findings may not apply to dissimilar patient groups.

VARIATION WITH TYPE OF DEMENTIA

The prevalence of particular BPSD has been found to differ among dementias of differing etiology. Eriksson (1996) found differences between vascular dementia (VaD) and Alzheimer's disease (AD) sufficient to warrant separate analyses. Patients with VaD appear more likely to experience depressive symptoms¹.

Fronto-temporal dementia (FTD) has been associated with higher incidences of many symptoms² including impulsivity³, compulsive behaviors⁴ and verbal outbursts⁵. Loss of emotions and insight, selfishness, disinhibition, personal neglect, gluttony and sweet food preference⁶, wandering, motor and verbal stereotypes, loss of pain, echolalia and mutism are symptoms that discriminate FTD from AD, whereas irritability, hyposexuality and hypersomnia did not discriminate⁷. Emergence of artistic abilities has been associated with left temporal involvement in fronto-temporal dementia.⁸ The anatomic distribution of asymmetric atrophy in FTD has been correlated with specific BPSD.⁹

In dementia with Lewy bodies (DLB), neuropsychiatric symptoms are among the defining features and are present in a much higher frequency than in other dementias. Visual hallucinations are prominent but hallucinations in other domains and delusions are also more common as are mood changes and insomnia with REM sleep behavior disorders^{10, 11}. Not only are visual hallucinations more common, they are also more persistent in AD¹². Extreme sensitivity to the side effects of neuroleptics is found in dementia with Lewy bodies (DLB)¹³.

Huntington's disease has been associated with hypersexuality, psychosis, and impulsivity.¹⁴ Parkinsonian dementia is associated with more prominent disturbances of nocturnal behavior¹⁵ and greater apathy.¹⁶

These differences may help to guide prognosis when the probable type of dementia has been established. However, as shown earlier, a mixed etiology is not uncommon and can be expected to blur any such differences, especially in comorbid VaD, DLB, and AD, which appears to be quite common¹⁷.

PARTICULAR SYMPTOMS

Hallucinations

Hallucinations—experience of a sensation without external input—are common in dementia¹⁸ and typically occur in the early phases of DLB¹⁹. Some studies suggest that hallucinations predict a more rapid rate of cognitive decline in AD and increase the risk of aggressive behavior^{18,20} although others fail to find such a relationship.

Somatic hallucinosis are syndromes associated with sensory deficits; i.e., the patient with impaired vision will report visual

hallucinations (Charles Bonnet Syndrome) while the patient with impaired hearing reports hearing music that is not real (musical hallucinosis). It is important to distinguish those hallucinations seen in other psychoses in order to direct the treatment appropriately²¹.

Delusions

Delusions—beliefs without foundation and contrary to reality—are frequent in patients with dementia and may arise from attempts to rationalize the effects of lost memory; e.g., a misplaced item has been "stolen."

Common delusions in dementia include beliefs that people are stealing things (experienced by 18-43% of patients with dementia) that one is being abandoned (3-18%), and that a spouse is unfaithful (1-9%)²². Delusions are often associated with physical aggression²³.

Misidentifications

Delusional misidentification syndromes affect about 30% of patients. Both the Capgras delusion — the belief that relatives have been replaced by impostors — and the Fregoli delusion — the belief that familiar people are disguised as others—have been described in patients with dementia²⁴.

A "phantom boarder" is the delusion of an intruder living in the patient's home. In the "mirror image," patients with AD often experience their image in the mirror as that of another person. Another common type of misidentification occurs when the person with dementia thinks that people on television are real or in the room with them.

The presence of such misidentifications has been correlated with several neuropathological and neuroanatomic markers.²⁵

Depressed mood and other affective symptoms

Depressive symptoms occur to some degree in 30% or more of patients with dementia^{26,27} and can be associated with significant disabilities, such as:

- Isolation, withdrawal
- Appetite and associated weight loss
- Sleep disturbances
- Loss of muscle tone and mobility
- Lack of self-care.

Depressive symptoms may also be associated with constant requests for help, complaining, and negativism.²⁸

Most standard diagnostic scales, with the exception of the Cornell Scale for depression in dementia²⁹, have not been validated in the elderly and the usual criteria for depression can be difficult to apply in patients with dementia, particularly in later stages. Given the prevalence of depressive symptoms in dementia, a high index of suspicion is clinically prudent, and a consequent trial of therapy may be warranted. However, in the face of uncertainty about either the diagnosis or the contribution of depression to BPSD, consultation with or referral to secondary care should be seriously considered.

Apathy and amotivation

Apathy—a lack of passion, emotion or excitement and indifference to appeals to feeling or interests—is one of the most common of the BPSD.³⁰ Lack of motivation is more common in AD than in frontal-lobe dementias,³ and is more common among men than women.³¹ Unfortunately, it may not be identified as a problem by caregivers, and is therefore less likely to prompt attempts at treatment. Nonetheless, there is evidence that apathetic symptoms are troublesome and distressing to caregivers³².

The inability to “get started” can be one of the earliest symptoms of dementia and well-informed caregivers can help initiate tasks that can then be completed by the patient. Failure to eat can be part of the constellation of apathy symptoms, and may be present in the absence of depressed mood, especially in advanced-stage dementia.

Anxiety symptoms

Anxiety is frequent among patients with dementia and may be associated with irritability, overt aggression, psychomotor agitation, and pathological crying³³. Behaviors such as pacing, chanting and repetitive tapping may reflect underlying anxiety. Refusal to allow necessary care such as bathing, dressing, and dental or podiatric care may reflect acute situational anxiety and may respond to measures that decrease anxiety, such as pre-medication with anxiolytics or listening to music³⁴.

As in the general patient population, the frequent finding of co-morbid anxiety and depression persists in those with dementia and symptoms of either should prompt specific evaluation for the other. Unrecognized pain can produce symptoms of anxiety such as irritability and aggression that will respond to pain relief.

Aggression

Aggressive behaviors probably prompt the most calls by caregivers for help from clinicians. Aggressive symptoms are often characterized as either physical or verbal.³⁵

Physical aggression is associated with more frequent delusions and more severe irritability³⁶. Prospective studies show an association between aggression and the need for institutionalization³⁷ and aggressive behavior has been found to be particularly common in FTD2.

Disinhibition

The disinhibition syndrome is associated with impulsive and inappropriate behaviors, emotional instability, poor insight and poor judgment. Symptoms include crying, euphoria, verbal aggression, physical aggression, self-destructive behavior, sexual disinhibition, intrusiveness, wandering, shoplifting, impulse buying and other unrestrained behaviors.

Negativism

Negativism is characterized by the rote refusal to do things, even when the patient does not understand what they are being asked to do. This can lead to stubborn, uncooperative behavior and resistance to care.

Intrusiveness and shadowing

Intrusiveness can be manifest by demanding, impatient clinging or pushing actions that attempt to force the caregiver into doing something involuntarily. Patients push themselves into situations without invitation or encroach on something possessed or enjoyed by another.

Shadowing is a variant of intrusiveness, where a patient closely follows and often imitates the actions (echopraxia) of another person.

ETIOLOGY

It is reasonable to expect that the better the cause of a disorder or event is understood, the better it can be managed. This is no less true for BPSD, although it is often difficult to identify specific triggers for BPSD. Efforts to find a cause or causes of BPSD are likely to be rewarded with better management.

- Start by ruling out medical conditions such as constipation, infection,

metabolic imbalance or pain that may not be articulated by the patient and unrecognized by caregivers.

- Seek and rule out comorbid conditions such as delirium, depression, anxiety or psychosis.

If any such conditions are found, not only does their discovery help to direct therapy, but it also helps caregivers to shunt blame for the symptoms away from the patient and collaborate more fully in treatment.

Medical conditions that can contribute to BPSD include stroke and Parkinson's disease, both predisposing to depression. Many painful conditions, such as arthritis or tooth decay, can contribute to insomnia, repetitive vocalizations and aggression.

Illnesses in older patients with dementia may lack characteristic features; e.g., appendicitis without abdominal pain, pneumonia without cough or fever, urinary tract infections without dysuria or fever. The initial, and often the only presenting symptom of such an acute illness, may be a change in behavior or mood.

Differentiate etiology to guide therapy

Always rule out delirium

The neurological damage of dementia markedly increases susceptibility to delirium, an often reversible, superimposed impairment precipitated by many factors including:

- Alcohol, drug or medication intoxication
- Infections, in particular pneumonia and UTI
- Dehydration and metabolic imbalance
- Sensory deprivation
- Psychological stress (e.g. absence of the primary caregiver)

A more comprehensive list will be found in Table 1.

Patients with a dementing illness are particularly susceptible to the ill effects of comorbid illness, drug side effects, and environmental stress that can lead to delirium. Dementia complicated with delirium may persist for several weeks and often includes symptoms similar to primary BPSD such as day-night reversal, psychomotor agitation or retardation.

It is therefore imperative to consider delirium in the differential diagnosis of BPSD. Delirium in the elderly with dementia may be a "quiet" phenomenon with slow onset in situations of metabolic illness, drug accumulation, malnutrition, or the like. Such "quiet" delirium is easily missed. A comprehensive evaluation of all possible sources of delirium including all medications, non-prescription, complementary or other alternative medicines is important.

Differentiating BPSD

Diagnosis of problems that may precipitate BPSD in patients with impaired ability to communicate due to their dementia will challenge the skills of any practitioner. Factors such as urinary urgency or nausea can precipitate BPSD and may be far from apparent when the patient cannot communicate effectively.

A patient using bilateral hearing aids presented with onset of marked psychomotor agitation and aggressive behavior. Closer examination found that both hearing aids were malfunctioning leading to extreme frustration and the consequent BPSD.

Table 1. Common Causes of Delirium by System

<p>Endocrine</p> <ul style="list-style-type: none"> • Hypercortisolemia • Hyperparathyroidism • Hypothyroidism • Pituitary insufficiency • Hypocortisolemia • Diabetes • Hyperthyroidism 	<p>Cardiovascular</p> <ul style="list-style-type: none"> • Bradyarrhythmias • Angina • Malignant hypertension • Tachyarrhythmias • Hypotension • Myocardial infarction 	<p>Gastrointestinal</p> <ul style="list-style-type: none"> • Constipation • Fecal impaction • Bowel obstruction • Perforated viscus • Hemorrhage • Hepatic failure • Diarrhea with dehydration or electrolyte imbalance • Bowel ischemia or infarction • Diverticulitis 	<p>Infection</p> <ul style="list-style-type: none"> • Generalized sepsis • Pneumonia • Scabies • Endocarditis • Decubitus ulcer infection • UTI, pyelonephritis • Meningitis • Herpes Zoster • Peritonitis • Cellulitis 	<p>Metabolic</p> <ul style="list-style-type: none"> • Renal Failure • Hyperglycemia • Hyponatremia • Hypercalcemia • Hypermagnesemia • Hepatic failure • Hypoglycemia • Hyponatremia • Hypocalcemia • Hypomagnesemia • Hyperosmolality • Hypercapnia • Fever • Hypoosmolality • Hypoxia • Hypothermia
<p>Neurologic</p> <ul style="list-style-type: none"> • Stroke • Subdural hematoma • Brain tumor • Epidural hemorrhage • Hydrocephalus • Concussion • Seizures • Cerebral vasculitis • Non-convulsive epileptic state • Meningitis 	<p>Pulmonary</p> <ul style="list-style-type: none"> • Hypoxia • Sleep apnea • Pneumonia • Hypercapnia • Pulmonary embolus • Pulmonary edema 	<p>Urinary Tract</p> <ul style="list-style-type: none"> • Uremia • Urinary tract infection • Urinary obstruction • Urinary tract stone 	<p>Miscellaneous</p> <ul style="list-style-type: none"> • Occult fracture, e.g. pelvic, hip • Drug intoxication or side effect, e.g. anticholinergics • Drug withdrawal, e.g. sedatives, alcohol, neuroleptics, nicotine, caffeine, antidepressants • Drug interactions • Sensory deprivation, i.e. due to visual and auditory impairment • Environmental change, e.g. hospitalization, travels, etc. • Foot blisters in patients with wandering. 	

Relatively common and treatable medical conditions such as fecal impaction, urinary retention, infections, intoxications, sleep deprivation, heartburn, or unsuspected angina can trigger the onset of BPSD.

Consider also the patient's living

circumstances. Factors as simple and apparently trivial as uncomfortable clothing may contribute to BPSD. In general, patients presenting with BPSD will require a very careful current history and physical examination to discover clues to the etiology of the BPSD. Laboratory tests can be very

useful in identifying contributory factors such as hypocalcemia, hepatic and renal failure, hyperglycemia, electrolyte abnormalities, hypoxia, or hyper- and hypothyroidism, any of which can cause or contribute to BPSD.

When evaluating BPSD it can be helpful to

use any of several scales that have been developed to provide consistent and reliable identification of specific BPSD. Some of these scales also classify the intensity and/ or frequency of symptoms, which will prove useful in assessing the effects of treatment over time. As well as those scales specific to BPSD, others specific for all common psychiatric syndromes that may be mimicked by BPSD, are also readily available. They will be helpful when specific BPSD such as depression, anxiety or psychosis has been identified and is the target of therapy. The Appendix contains a selected list of scales.

Laboratory tests that should be routinely considered when evaluating BPSD and ruling out treatable causes are listed in the next section. However, there may be cases where the experience and expertise of secondary care should be utilized to differentiate the etiology of BPSD and determine therapeutic strategy.

Many cases will require referral for specialist evaluation and suggestions for optimal therapy.

Finally, it is worth noting that recent and ongoing research may shed light on the biological basis of BPSD and provide tools to avert or control these distressing symptoms. Biochemical³⁸, neuroanatomic³⁹, metabolic^{40,41} and personality⁴⁴ correlates with BPSD have been identified and neuroscientists have begun to unravel associations between genetic variation of neuroreceptors and susceptibility to BPSD^{43,44,45}.

Laboratory and Imaging Examinations

A variety of tests can be useful when attempting to identify factors that may be contributing to BPSD in a patient with dementia.

- Complete blood count
- Urinalysis and culture
- Electrolytes, including calcium and magnesium
- BUN and creatinine
- Blood Sugar
- Thyroid stimulating hormone
- Chest X-ray
- Electrocardiogram
- Arterial blood gases

In particular circumstances, tests generally available in specialist units may be undertaken:

- Lumbar puncture if there is suspicion of meningitis
- Tumor markers if a cancer is present or suspected
- CT or other scans if there is suspicion of structural lesions
- EEG if there is suspicion of delirium

Clinical Management of BPSD

The basic objectives of therapy include:

- Maximizing functional independence and quality of life for patients
- Minimizing caregiver stress and distress and maximizing their ability to cope with and care for the patient

The armamentarium for this task encompasses non-pharmacological techniques such as:

- Manipulating the environment
- Training for resolution of aberrant behavior
- Using behavior management techniques
- Fostering reminiscence
- Providing cueing
- Structuring activities

and the use as necessary, of a range of pharmacological agents including:

- Anxiolytics
- Antidepressants
- Antipsychotics
- Anticonvulsants
- Sedatives, hypnotics

While available agents directed at cognitive symptoms provide at best only modest and temporary slowing in the rate of decline, BPSD are often more amenable to both non-pharmacologic and pharmacologic interventions⁴⁶. Consequently, given the distress caused by BPSD, their impacts on the quality of life of both patients and caregivers and the cost of care it is incumbent upon primary care practitioners to be able to:

- Recognize and quantify individual BPSD
- Diagnose the cause in many cases
- Devise a management plan
- Evaluate outcomes
- Adapt or modify therapy to obtain the optimum possible result

Alleviating BPSD has potential to improve the functional abilities of patients with dementia and clearly can improve their quality of life and that of their caregivers.

MANAGEMENT STRATEGY

The first step in management is to attempt to determine the etiology of any symptoms of dementia and, if possible, to determine which dementing illness is most likely. Although efficacy is limited, there are now approved treatments for cognitive impairment in Alzheimer's disease, e.g. the cholinesterase inhibitors, which have also been shown to have benefit on some behavioral symptoms (see below). Interventions for other primary progressive dementias may be approved in the future. Improved or preserved cognitive function due to such treatments may also have a positive effect on BPSD. In addition, the underlying dementia has implications for management of BPSD. For example, management of disinhibitory symptoms due to FTD may well differ from that used in AD.

In cases where dementia has been previously diagnosed, reversible causes of BPSD such as delirium or concurrent illness (see Table 1) should be ruled out. Remaining BPSD will often be related to common psychiatric syndromes such as depression, anxiety or psychosis where the diagnosis will indicate the appropriate therapeutic interventions.

The need for and level of intervention require careful consideration. Mild or relatively benign BPSD may not justify pharmacological intervention. In all cases the degree of distress the symptoms are causing to patients, their caregivers, and other people in their environment must be determined.

Behaviors that do not endanger anyone or infringe on others' rights may not need to be changed, even if annoying! Functional impairment that can be attributed to the BPSD has to be evaluated together with the difficulty and distress caregivers have in dealing with the symptoms. In some cases, caregivers may simply need reassurance that the BPSD are not serious and that immediate intervention is not advisable.

Behaviors may be productive even if they cause some problems for caregivers. For example, asking many questions or attempting to feed one's self should be considered assets rather than liabilities. Conversely, one must be alert for behaviors that caregivers may not consider problematic, but are detrimental to achieving care goals (e.g. refusal to drink or eat). Learn to look for and manage apathy, withdrawal and over-compliance (e.g. failure to report discomfort or pain).

Ethical Issues

If active therapy is considered necessary, informed consent to treatment must be

considered. This will be particularly important when therapy may involve off-label use of psychotropic or other medications. In at least some cases, there will be doubt whether or not the patient will comprehend proposed therapy well enough to give an informed consent. Because these patients are likely to be well known due to the duration of their dementia, hopefully the need for consent by a third party will have been anticipated with arrangements for guardianship or power-of-attorney. At some time during the course of dementia, capacity for informed consent will be beyond the remaining capabilities of the patient and will have to be granted by another. Preparation for that day will prove an asset for patient, caregiver (or other responsible person) and physician.

While such a legalistic approach may seem rather dogmatic, it is worthwhile to respect the right of the individual, with or without dementia, to agree or disagree with a proposed intervention however well intentioned it may be. Occasionally caregivers may wish for treatments that would render the patient with dementia less intrusive or bothersome at the cost of suppressing what little cognitive or motivational assets yet remain for the patient.

Therapeutic approach

Non-pharmacological and environmental strategies should be the first line of therapy when symptoms are not severe. If these fail to achieve a satisfactory outcome, pharmacological intervention therapy should be undertaken.

In the event that BPSD are dangerous or even life threatening, aggressive management is indicated and early initiation of pharmacologic treatments is both necessary and justified. For any occurrence of BPSD, the caregiver's capabilities and needs must be considered

and appropriate advice, education and support supplied.

NON-PHARMACOLOGICAL MANAGEMENT

Non-pharmacological and environmental interventions devised with the support of family and other caregivers will often manage BPSD effectively. Creativity is a necessary ingredient in such management.

ABCs of Behavioral Management Strategies

- Assess for and address factors that can be readily treated – unmet needs such as pain, wetness or hunger.
- Assess for and address delirium, depression.
- Anticipate situations and environments that predictably provoke anxiety and fear, and make every effort to modify them to minimize those effects.
- Acknowledge the demented patient's frustration and anger over the loss of capability and control. Assure that both patient and caregivers are aware of this understanding.
- Be consistent—maintain structure by following fixed routines.
- Caregivers benefit from education. Teach techniques for better communication with patients with dementia:
 - To use short, simple sentences
 - To give only one directive at a time
 - To make positive statements and avoid negative phrasing, for example, "Stay inside" rather than "Don't go outside"
 - To speak slowly and not to be afraid of repeating themselves frequently
 - To maintain eye contact when speaking
 - To use gestures and nonverbal cues such as exaggerating a smile or a nod

- To limit choices to minimize confusion
- To avoid confronting or correcting the patient unnecessarily
- To learn to interpret behaviors such as pulling at clothing that could indicate a need to use the toilet.

Some useful non-pharmacological techniques

- Distract the patient with a snack or an activity.
- Use gentle touch, soothing music, reading, or walks to dissipate anxiety and stress.
- Use symptom-appropriate approaches. For example, if there are symptoms of depression⁴⁷, consider interventions that engage patients in activities that are not beyond their abilities to encourage a sense of control and accomplishment, and minimize frustration. Depending on the stage of dementia, offering activities such as gardening, pet care, arts and crafts or the like can help treat the depressive symptoms. Similarly, anxiety-related symptoms can be minimized by reassurance, familiar environments, calming music and lighting.
- Anticipate situations that predictably provoke anxiety, such as baths, dressing and dental or podiatry appointments. Provide reassurance and approach the situation slowly and calmly.
- Educate family and other caregivers to explain what they are doing there, which often means reintroducing oneself many times a day.
- Learn to recognize certain behaviors. An agitated state or pulling at clothing, for example, could indicate a need to use the toilet.
- Try non-verbal reassurances such as a gentle touch or hug. Be liberal with

encouragement and compliments. The ability to respond to flattering comments seems often to be retained late into the dementing illness.

- Provide activities that help interest and stimulate patients with dementia without over stimulating them. Activities after dinner that keep the patient awake until later in the evening can help prevent early morning awakening and associated BPSD that result from the sleep phase advance associated with going to bed too early. Regular exercise—outdoors if possible—helps to dissipate anxiety and can help keep demented patients awake during the daytime and tired at night. Such strategies can minimize the day-night reversal that occurs in many demented patients.
- Carrying a doll can calm certain patients while exposure to pets has a similar effect on others. Beautician services such as hair styling and nail manicures can be very soothing. Participating in food preparation has the dual benefit of giving a sense of usefulness and stimulating appetite.
- Camouflaging exits with curtains or painting doors black can help to minimize wandering. Similarly, a wide, dark stripe on the floor in front of restricted areas can help to control movements. Conversely, a large sign with the patient's name together with a room filled with familiar objects can reassure the individual that he or she is in the right place.

Physical restraint should be limited to situations where all else has failed. But restraint may be necessary if the individual is physically unable to move about safely or is at risk of injury, cannot remember this. For example, amputees with dementia, hemiparetic patients or severely Parkinsonian patients with dementia are at high risk for injury. In these circumstances, the benefits of restraint may

outweigh the associated risks.

For physically well patients, restraints are to be avoided as much as possible. Restraints contribute to increased agitation, decreased mobility, contractures, pressure ulcers, and can lead to tragic accidents such as strangulations. The environment should be made as safe as possible for the patient with dementia and should allow those who are ambulatory the opportunity to wander safely without restraints.

Proxy consent from the person's legal guardian or equivalent should always be sought. In some jurisdictions other legal consent procedures apply. The use of physical restraints should be time-limited and reviewed frequently.

PHARMACOLOGICAL MANAGEMENT

When other methods have failed, medications may be indicated. Older people, especially those with dementia, often respond differently to medications than do younger adults. Consequently, medication should be started at lower doses. It is also noteworthy that responses to medication among the elderly are generally much more variable than in younger populations. Clinicians should consult a pharmacology text for a comprehensive account of adverse effects of medications discussed in Table 2.

Although many different classes of drugs have been used to treat BPSD, there are few large, randomized, controlled trials to establish their safety and efficacy for this indication. Consequently, use of many medications in BPSD is outside approved labeling and general practitioners should consider specialized advice before prescribing.

In the published studies that do show medication efficacy in BPSD, robust placebo

responses are often found as well. Several consensus statements have been published⁴⁸, and guidelines have been promulgated. Primary care physicians should attempt to become comfortable treating the majority of patients, while recognizing that for those patients who do not improve or for complicated cases, psychogeriatric or other secondary care consultation should be considered.

All front-line physicians should be familiar with the most commonly used classes of drugs applied in BPSD and develop a strategy that allows an appropriate choice of first line therapy for each patient. As noted repeatedly in this guide, proper choice will be facilitated by identifying presenting BPSD with analogous psychiatric syndromes. The ultimate goal is to find an efficacious drug that is administered in the lowest effective dose with the smallest burden of side effects.

The goal is to find a drug that is efficacious at the lowest possible dose with the smallest burden of side effects.

It is also necessary to consider that many elderly patients will be receiving several other medications for coexistent medical illnesses with consequent higher risk for drug-drug interactions. As dementias are progressive diseases with an evolving clinical picture it will be necessary to review prescriptions regularly. Because symptoms come and go it may prove possible to taper or even discontinue treatment without reappearance of the target BPSD⁴⁹. Review of the need for medication with periodic trial reduction should be undertaken every 3-6 months⁵⁰.

Table 2. Sample Psychotropic Dosing: Recommended Oral Doses*

Class/Medication	Starting Dose (mg/day)	Average Target Dose (mg/day)
Typical (conventional) High Potency Antipsychotics		
Haloperidol	0.25 – 0.5	1.0-2.0
Atypical Antipsychotics		
Risperidone	0.25 – 0.5	0.75-1.75
Olanzapine	2.5-5	5-10
Anxiolytics		
Lorazepam	0.5-1.0	1.5-2.0
Oxazepam	5-10	10-30
Buspirone	10	30
Trazodone	25-10	50-100
Antidepressants		
Citalopram	10-20	20
Mood Stabilizers		
Carbamazepine	100-200	400-600
Divalproex	125-250	500-750

*Prescribers should check product information for contraindications and possible adverse effects.

Typical (Conventional) Antipsychotics

By far the most common class of drug used for treatment of BPSD in the elderly is the antipsychotics. The older antipsychotics (sometimes referred to as neuroleptics) are most clearly indicated when true psychotic symptoms of BPSD such as delusions, hallucinations or paranoia are present. Clinical trials have confirmed efficacy in improving non-psychotic BPSD symptoms, as well⁵¹.

In the past, these drugs were often chosen in part for their sedative rather than antipsychotic properties. A more modern approach is to use the lowest dose that controls the target BPSD thereby minimizing sedation. A randomized, double-blinded study of the high-potency typical antipsychotic haloperidol⁵² found significant benefit at

doses of 2-3 mg/day. However, even at these doses, a subgroup of patients developed moderate to severe extrapyramidal signs and tardive dyskinesia, which is often irreversible, and no benefits were found with lower doses.

A meta-analysis of placebo-controlled studies in this class of drug did show modest (only 18% better) improvements compared with placebo. No one member of the class was found superior to the others⁵³. A more recent meta-analysis failed to demonstrate efficacy⁵⁴.

Side effects such as extrapyramidal disorders, sedation and tardive dyskinesia are more common in the elderly generally and these older, so-called "typical" or "conventional" neuroleptics such as haloperidol have a

cumulative incidence of tardive dyskinesia of approximately one-third of patients after one year⁵⁵.

Given the increased incidence of side effects at higher therapeutic doses, if a typical antipsychotic is chosen, treatment should be initiated with a very low dose and increased in small increments as needed to control the target BPSD.

For example, with a typical high potency drug such as haloperidol a good starting dose is 0.25-0.5 mg/day. If not efficacious after a week or so, slowly titrate upwards, and try not to exceed a total dose of 2.0 mg/day. Avoid the use of anticholinergic drugs to mitigate any extrapyramidal side effects, as their propensity to cause delirium, constipation and urinary retention outweighs the potential benefit.

Atypical Antipsychotics

Newer drugs, usually referred to as "atypical antipsychotics," include such drugs as risperidone, olanzapine and quetiapine. Members of this class can minimize the risk of extrapyramidal side effects and tardive dyskinesia, but are more costly than the conventional or "typical" antipsychotics.

A recent double-blind randomized study demonstrated that risperidone was more effective than placebo for both psychotic symptoms and more general measures of BPSD⁵⁶. An international study showed similar benefits of risperidone, including a significant effect on reducing physical aggression⁵⁷. An Australian and New Zealand study demonstrated superiority for risperidone over placebo in the treatment of aggression in nursing home residents with dementia⁵⁸. The incidence of tardive dyskinesia with this atypical antipsychotic was 2.6% - one tenth that found with the older agents. The dose

of risperidone that showed benefit with the least side effects was 1 mg per day. A useful regimen is to start at 0.5 mg/day, increasing by 0.5 mg/day in weekly steps if not effective, to a maximum of 2 mg/day.

Agitated nursing home patients treated with olanzapine benefited from doses of 5 and 10 mg daily, compared to placebo, but there was no difference between 15 mg daily and placebo⁵⁹. A useful starting dose is 5 mg/day of olanzapine, increasing to a maximum of 10 mg/day. There are as yet no data for quetiapine, ziprasidone, or aripiprazole in BPSD, and there are no published studies comparing the efficacy of atypical and typical antipsychotic agents or comparing the atypicals with each other.

Tiapride is an atypical antipsychotic derived from benzamine family and used as a treatment for alcoholism as well as several neurological conditions. One trial suggests equivalent efficacy to haloperidol compared with placebo in treating agitation in patients with dementia⁶⁰.

If an antipsychotic is used, it is desirable to designate "target symptoms." If control of hallucinations is the object of treatment, it is not then appropriate to increase the dosage if a behavior such as "refusal to cooperate with bathing" persists or is manifest when hallucinations have been eliminated. A useful starting dose is 5 mg/day of olanzapine, increasing to a maximum of 10 mg/day.

Antidepressants

One short-term randomized placebo controlled trial in hospitalized dementia patients has shown that an antidepressant, the selective serotonin reuptake inhibitor citalopram, had efficacy equal to that of an antipsychotic in reducing moderate to severe BPSD⁶¹. Benefit was seen in a matter of days; well before

antidepressant effects could be expected. There are no published long-term placebo controlled trials for the use of antidepressants for agitation.

Depression should be treated in patients with dementia and physicians should have a low threshold for initiating treatment. In general, the selective serotonin reuptake inhibitors (SSRIs) are the "first line" class of antidepressants.

BPSD may be manifestations of depression. A study of citalopram shows efficacy for measures of generalized well-being as well as for depressive symptoms in dementia⁶². Larger trials are in progress. Antidepressants can have benefit in early or mild dementias and in severe dementia when standard diagnostic criteria are of little use.

As anti-cholinergic properties may contribute to delirium, tricyclic antidepressants with such properties should be avoided. If other antidepressants fail and a tricyclic is indicated, choose nortriptyline as it has the least anticholinergic side-effects of all tricyclics.

When prescribing antidepressants for BPSD, bear in mind that:

- A common source of failure is inadequate dosing.
- Slow onset of action is a class property of all antidepressant drugs. Allow 4-6 weeks trial after the desired antidepressant dose is reached.
- An alternate SSRI can be tried if one fails, or an agent can be chosen with other or additional neurotransmitter reuptake inhibition and/or direct effects on receptors.
- Activating antidepressants, such as venlafaxine or bupropion may be useful when activation is needed for patients

with withdrawal or psychomotor retardation.

- Mirtazapine can be useful when anorexia is a problem—it often causes appetite stimulation as a side effect. It is also sedating which can be helpful.
- Trazodone is an antidepressant that is very sedating in antidepressant doses. It can be used in lower doses, where it has prominent anti-anxiety effects. Comparable efficacy to haloperidol was shown in treatment of agitation associated with dementia in one relatively large study⁶³, but its alpha1 blocking effects can cause orthostatic hypotension.

A few randomized, placebo-controlled trials are available to guide us. Trials of clomipramine⁶⁴, sertraline⁶⁵ and citalopram⁶² showed benefit of treatment compared with placebo. Other trials of fluoxetine⁶⁶ and sertraline⁶⁷, in the treatment of depression in dementia patients, showed no difference from placebo. These clinical trials tend to show high rates of placebo response as well and a study of non-pharmacological interventions also showed benefit compared with placebo⁶².

In general, the key to using antidepressants in the elderly—and patients with dementia in particular—is to use an agent in an adequate dose for an adequate duration. Although starting with a low dose and slowly titrating up is an appropriate strategy, one must continue titrating until a full therapeutic dose is achieved ("Start slow, go slow, but go and be persistent!"). Once depression is satisfactorily alleviated, past history will guide the decision as to whether treatment should continue indefinitely or whether the antidepressant dosage should be tapered after 6-12 months while monitoring for recurrence of symptoms.

Cholinesterase inhibitors

Alzheimer's disease and many other forms of dementia are associated with a prominent deficiency of acetylcholine. Cholinesterase inhibitors increase the residual acetylcholine in the brain and are indicated for treatment of cognitive impairment in patients with mild and moderate AD. The initial studies of cholinesterase inhibitors examined cognitive rather than behavioral end-points. Subsequent studies have shown benefit on cognition in severe AD, other dementias (LBD, VaD), and on behaviors^{68,69} particularly apathy, depression and anxiety⁷⁰.

A randomized trial of donepezil in nursing home patients with behavioral problems was negative for benefit for the behavioral symptoms⁷¹ while a trial of donepezil in community-dwelling patients with moderate-severe AD was positive for some BPSD, especially in those without concomitant psychoactive medications. A trial of rivastigmine in dementia with Lewy bodies patients showed reduction of apathy, anxiety, delusions and hallucinations⁷². Trials of galantamine in AD have also shown significant benefit for behavioral parameters^{73,74}. These effects on behavior are in addition to the well-documented benefits in cognitive function with agents such as donepezil, rivastigmine, and galantamine. Their cognitive effects can justify the use of any one of these agents. In situations where the decision to use such an agent is "on the borderline," the potential for a beneficial effect on BPSD can be a factor for consideration⁷⁵.

It should also be recognized that the cholinesterase inhibitors could have side effects, especially at higher doses. The side effects could be nausea, vivid dreams, headache and others that a dementia patient may find difficult or impossible to describe.

If deterioration of behavior is seen after starting one of these drugs, try stopping the cholinesterase inhibitor and re-evaluating—it may be causing agitation as a response to drug side effects that cannot otherwise be detected.

Memantine

Memantine, a non-competitive NMDA glutamate receptor antagonist, has been available in Germany for about 20 years for the treatment of organic brain disease. The drug has recently been approved in Europe and Australia for the management of cognitive impairment in moderately severe and severe AD and is being considered by the regulatory authorities in the United States for the same indication. Studies have suggested less emergent agitation with memantine^{76,77}.

Benzodiazepines

The benzodiazepine anxiolytics have been shown to be of some benefit, but all share the side effects of sedation, motor and cognitive impairment, and a propensity to cause withdrawal symptoms when discontinued. Delirium, paradoxical excitation, and falls may also occur.

Sleep architecture is also altered, with inhibition of both rapid eye-movement (REM) sleep and delta wave sleep. These side effects make benzodiazepines a poor choice for regular use in patients with dementia. They are best used for short-term management of sleep-cycle disturbances or anxiety states, or as planned premedication prior to predictably anxiety-provoking situations.

As pre-medication prior to frightening situations such as dental or podiatry care, intermediate-acting benzodiazepines can be used on occasion. This is the best use of benzodiazepines for BPSD, as it avoids the

risk of withdrawal phenomenon, which can actually precipitate or predispose to agitated behavior. As the half-life of the commonly used drugs results in significant motor impairment for a day or so after use, careful supervision after use is needed to prevent falls.

No benzodiazepine is more anxiolytic than any other and choice is primarily based on the pharmacokinetic profiles. The intermediate acting drugs, oxazepam and lorazepam, have a short half-life, and no active metabolites. These drugs have intermediate to slow onsets of action, and so when used as premedication, should be given at least two hours prior to the stressful event. Examples include oxazepam 10-15 mg given 3-4 hours before the procedure or doctor's visit, or lorazepam 0.5-1.0 mg given 2-3 hours prior to the procedure.

Mood Stabilizers

The efficacy of mood stabilizers on agitation and aggression such as carbamazepine, valproate, gabapentin or lithium in treating BPSD has been observed in several small studies⁷⁸.

These first two drugs are generally well tolerated, but somnolence and motor symptoms may limit their utility in some patients. Divalproex sodium has the advantages of fewer adverse effects and drug-drug interactions. Monitoring of hematology and hepatic transaminases has been recommended due to the potential for adverse reactions.

There are no studies of gabapentin, topiramate or lamotrigine for BPSD, although anecdotal benefit of gabapentin has been reported.

The narrow therapeutic window for lithium,

the frequency and severity of side effects, and the lack of strong data supporting its efficacy for BPSD make it a poor choice for primary care providers to use in this setting.

In general, the lack of convincing data demonstrating efficacy for the mood stabilizers for the treatment of BPSD should limit their use to situations where first line therapy has proven unsatisfactory.

Buspirone

Azapirones are a relatively new class of anti-anxiety agents. The currently available member of this class is buspirone, which, in Europe, is only available in Italy. These drugs work as partial serotonin (5-HT_{1A}) receptor agonists. There are no randomized, placebo-controlled trials demonstrating efficacy for BPSD. Their well-tolerated side-effect profile, which includes low sedation, minimal impairment of motor function and minimal withdrawal phenomenon, make them an appropriate choice when chronic anxiety symptoms warrant the regular administration of medication. They have a two to three week delay until full onset of action. Dosages for BPSD range from 10-80 mg/d in divided doses. Given the lack of data for this class of drug, consider an antidepressant as an alternative, as most antidepressants also have prominent anxiolytic properties.

Beta-Blockers

There are no large or recent studies, but in the past propranolol (80 to 560 mg per day) has been used after conventional therapy had failed. In one small study, low-dose propranolol monotherapy (10-80 mg/day) was also effective in reducing aggression⁷⁹. The agitated behavior was controlled without inducing general sedation or adverse side effects.

Medical contraindications include asthma, congestive heart failure, diabetes, peripheral vascular disease, or a history of depression. Due to the lack of supportive data and frequent contraindications, beta-blockers are not considered a first-line choice for managing BPSD.

Estrogens and Anti-androgen Treatments

Estrogens and anti-androgen treatment (e.g. medroxyprogesterone acetate and leuprolide acetate) have been proposed for the specific BPSD of sexually inappropriate behavior or extremely aggressive behavior in men. Evidence for their use remains anecdotal and there are many potential side effects. In general, they are used as a last resort. Legal restrictions to their use apply in some jurisdictions.

Antihistamines

Sedating and anticholinergic antihistamines such as diphenhydramine can result in worsening confusion and other symptoms of delirium. They may be perceived inappropriately as safer than "traditional" psychotropic drugs because they are available without prescription in many countries. In general, they are a very poor choice for managing BPSD.

Cross-cultural Aspects of BPSD

Although BPSD occur in all cultures that have reported experience, the impact of individual behaviors is very much influenced by culture. A commonly used example is wandering behavior, which can be very well tolerated in rural areas where the community is tolerant and willing to return demented patients when they stray. Conversely, disinhibition of sexual behavior may be very poorly tolerated in cultures where such behavior is strongly forbidden on religious or cultural basis.



Appendix: Scales in BPSD

This Appendix lists scales that non-specialists may find useful for the assessment of BPSD and the identification of depression and delirium. Some of the scales e.g., the MOUSEPAD require a degree of training.

*BEHAV-AD

Reisberg B, Borenstein J, Salob SP et al. Behavioral symptoms in Alzheimer's disease: phenomenology and its treatment. *J Clin Psychiatry* 1987;48:9-15.

Brief Agitation Rating Scale (BARS)

Finkel SI, Lyons JS, Anderson RL. A brief agitation rating scale (BARS) for nursing home elderly. *J Am Geriatr Soc* 1993;41:50-52.

Caretaker Obstreperous Behavior Rating Assessment (COBRA)

Drachman DS, Swearer JM, O'Donnell BF, et al. The Caretaker Obstreperous Behavior Rating Assessment (COBRA) scale. *J Am Geriatr Soc* 1992; 40: 463-480.

Cohen-Mansfield Agitation Inventory (CMAI)

Cohen-Mansfield J, Marx MS, Rosenthal AS. A description of agitation in a nursing home. *J Gerontol* 1989; 44: M77-M84.

Confusion Assessment Method.

Inouye SK, van Dyck CH, Alessi CA et al. Clarifying Confusion: the Confusion assessment Method, *Annals of Internal Medicine* 1990; 113: 941-948.

Cornell Scale for Depression in Dementia

Eliopoulos GS, Abrams RC, Young RC, Samoan CS. Cornell Scale for Depression in Dementia. *Biol Psychiatry* 1988; 23: 271-284.

Delirium Rating Scale.

Trzepacz PT, Baker RW, Greenhouse J. A symptom rating scale for delirium, *Psychiatry Research* 1988; 23: 89-87.

Global Assessment of Psychiatric Symptoms (GAPS)

Raskin A, Crook T, *Global Assessment of Psychiatric Symptoms (GAPS)*. *Psychopharmacol Bull* 1988; 24: 721-725.

Manchester and Oxford Universities Scale for the Psychopathological Assessment of Dementia (MOUSEPAD)

Allen NHP, Gordon S, Hope T, Burns A. Manchester and Oxford Universities Scale for the Psychopathological Assessment of Dementia (MOUSEPAD). *Br J Psychiatry* 1996; 169: 293-307.

Sandoz Clinical Assessment - Geriatric (SCAG)

Shader RL, Harmatz JS, Salzman C. A new scale for clinical assessment in geriatric populations: Sandoz Clinical Assessment- Geriatric (SCAG). *J Am Geriatr Soc* 1974; 22: 107-113.

*The Neuropsychiatric Inventory

Cummings JL, Mega, Gray K et al. The Neuropsychiatric Inventory: comprehensive assessment of psychopathology in dementia. *Neurology* 1994;44:2308-14.

* Denotes most widely-used

Glossary

Aggression

Unprovoked or disproportionate act of hostility that can be verbal or physical in action

Amotivation

Absence of the internal drive to accomplish tasks

Apathy

Lack of passion, emotion or excitement; indifference to appeals to feeling or interest

Delusion

A false belief that cannot be changed by rational argument

Depressed Mood

Sadness or loss of pleasure in activities that normally produces pleasure

Hallucination

A perception with no external cause

IPA Educational Programs for BPSD

At IPA's Seventh International Congress, the BPSD Task Force determined its mission statement to be:

"The promotion of research, training and dissemination of information on behavioral disturbances of dementia [now referred to as BPSD] to healthcare professionals and caregivers."

The goals of the BPSD educational programs are to:

- inform psychiatrists, neurologists, geriatricians, related healthcare providers and caregivers of the behavioral and psychological symptoms of AD and other dementias
- inform about the relationship between the symptoms and the course of the illness
- view the symptoms both individually and collectively in developing a specific plan for intervention
- describe what is known about current treatments and management
- describe and understand the specific needs of caregivers in relation to these symptoms.

Educational modalities being developed include:

- publication in journals
- slides
- an Internet web site. (The BPSD Educational Pack is available at IPA's web site, www.ipa-online.org)

For more information about IPA educational programs for BPSD, please contact:

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For a broader perspective and further clarification of dementia we suggest the following sources:

Highly Recommended Reading

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