# Chronic Brain Failure

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**KEYWORDS**

• Dementia • Neurocognitive • Neuropsychiatric • Emergency • Agitation

**KEY POINTS**

- Chronic brain failure is also known as dementia or major neurocognitive disorder. Like other organ failure syndromes, its impact on quality of life can be mitigated with proper management.
- In addition to cognitive symptoms such as memory loss, chronic brain failure involves neuropsychiatric symptoms such as psychosis, mood lability, and agitation.
- The symptoms of chronic brain failure can mimic those of acute brain failure (delirium), but there are major distinctions to help guide diagnostic thinking.
- Neuropsychiatric symptoms are best managed nonpharmacologically. Medications to treat agitation should be used only as a last resort.
- The use of logic and reason are rarely successful when attempting to redirect someone with advanced dementia. Interactions that offer a sense of choice are more likely to succeed.

## CASE

*Pertinent history*: A 78-year-old man with a history of Alzheimer's disease (AD) was sent to the emergency department (ED) after becoming aggressive toward staff at his assisted living facility (ALF). Over the prior week, he had been leaving the facility repeatedly, accusing staff of stealing his belongings and poisoning his food. On the day of presentation, he struck a staff member while she was attempting to redirect him. He has had no recent medical illness.

*Past medical history:* Hypertension, osteoarthritis, and benign prostatic hyperplasia. AD diagnosed by his primary care provider approximately 5 years ago. Currently, he receives his primary care through the ALF's nurse practitioner.
*Surgical history:* Left total hip arthroplasty 6 months prior
*Medications:* Hydrochlorothiazide 25 mg twice daily, tamsulosin 0.4 mg daily, trazodone 25 mg nightly, quetiapine 100 mg nightly (started 1 week prior), lorazepam topical gel 2 mg twice daily as needed for anxiety/agitation (started 2 days prior)
*Family history:* Mother died of AD at age 80, father died of myocardial infarction at age 54

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*Social history:* 10th grade education, retired assembly-line worker, divorced, one daughter who lives out of state and is designated as his power of attorney; 30 pack-year history of smoking

*Pertinent physical examination:* Temperature: 37.2°C; blood pressure: 141/93; heart rate: 110; respiratory rate: 20; oxygen saturation by pulse oximetry: 99% on room air

*General:* Alert, thin, disheveled, smelling of urine. Oriented to self only. Minimally cooperative, attempting to leave ED, states that there is nothing wrong with him.

*Skin:* No rashes or petechiae

*HEENT*: Pupils equal, round, reactive to light, mucous membranes dry, conjunctivae pale

*Neck*: Full range of movement (ROM), neck veins flat

*Cardiovascular*: Regular rhythm, tachycardic, no murmurs/rubs/gallops, distal pulses equal bilaterally

*Pulmonary*: Rate increased, lungs clear with no wheezes, rales, or rhonchi

*Abdominal*: Soft, nontender, nondistended, normal bowel sounds

*Musculoskeletal*: Normal pulses throughout, full ROM of all extremities, no peripheral edema

*Neurologic*: 5/5 strength and normal sensation throughout, gait slightly unsteady

*Psychiatric*: Dysphoric mood, irritable affect, paranoid delusions, no hallucinations

Montreal Cognitive Assessment (MoCA) = 11/30

*Diagnostic testing*: A delirium workup was initiated.

| | |
|---|---|
| WBC | 6.6 k/μL |
| Hgb | 13.2 g/dL |
| Hct | 39.5% |
| Plt | 190 k/μL |
| Na | 128 mmol/L |
| Potassium | 4.2 mmol/L |
| Cl | 97 mmol/L |
| $CO_2$ | 19 mmol/L |
| Glucose | 139 mg/dL |
| BUN | 20 mg/dL |
| Creatinine | 1.51 mg/dL |

*Abbreviations:* BUN, blood urea nitrogen; Cl, chloride; $CO_2$, carbon dioxide; Hct, hematocrit; Hgb, hemoglobin; Na, sodium; Plt, platelet; WBC, white blood cell.

*Electrocardiogram*: Sinus tachycardia, normal axis and intervals, no ST elevation or depression, T waves normal

*Chest radiograph*: Normal

*Head CT*: There is global parenchymal volume loss. The ventricles are enlarged commensurate with parenchymal loss. No acute intracranial process is identified.

*Clinical course*: The patient was thought to be at high risk for falls given his agitation and minimal cooperation with staff. He was also physically threatening. He repeatedly asked for his wife and was told that she was “out of town.” After several failed attempts to administer oral medication for acute agitation, the patient received

olanzapine 5 mg intramuscularly. He remained agitated after 15 minutes so was assigned a one-to-one staff companion for constant monitoring. After approximately 40 minutes, he was awake and alert, remaining seated in a recliner.

The patient was a poor historian, so collateral history was obtained from a nurse at the ALF. She reported that she had known the patient since he moved there 2 years ago. He had some moderate memory loss that worsened precipitously 6 months ago following an extended hospitalization for a hip replacement with postoperative delirium. He was ordinarily quiet and "a loner," but over the past few months he had become more anxious and irritable. He was frequently restless, especially late in the day, and began accusing staff of stealing clothing and personal items from his room. He had difficulty falling asleep, and awoke several times throughout the night, usually to urinate. Within the past few weeks, he was sleeping very little overall. He normally had a good appetite but was eating and drinking less over the past few weeks, accusing staff of poisoning his food. The ALF medical provider had prescribed quetiapine and lorazepam gel without any benefit. The patient had wandered away from the ALF several times recently, but always returned willingly. On the day of presentation, however, he struck a staff member who attempted to direct him back inside the building.

The nurse reported that she had consulted with the ALF Director; they felt that the patient required a "higher level of care" and thus would be discharged from their facility.

Several attempts to contact the patient's daughter were unsuccessful.

## INTRODUCTION AND BACKGROUND

- Chronic brain failure is a complex, acquired syndrome characterized by progressive and usually irreversible decline in multiple areas of higher mental function, resulting in a gradual loss of independent daily living activities. This loss of independent function has a wide-ranging impact on individuals, families, and health care systems.[1]
- Chronic brain failure is an umbrella term, used to describe a class of disorders more commonly known as dementias and major neurocognitive disorders (these terms are used interchangeably in this article, reflecting their use in the literature). These terms emphasize consequences rather than causes; the syndrome can result from a number of different disease processes.
- Chronic brain failure is predominantly, although not exclusively, a disorder of later life, typically affecting persons older than 65 years.
- Older adults with dementia are frequent ED visitors who have greater comorbidity, incur higher costs, are admitted to hospitals at higher rates, return to EDs at higher rates, and have higher mortality after an ED visit than patients without dementia.[2] They are also associated with higher rates of delirium, falls, behavioral problems, and physical decline.[3]
- Patients with dementia are viewed as particularly challenging to ED clinicians.[4] Although most patients do not present with a chief complaint of dementia, their dementia and related symptoms often overshadow the primary reason for the ED visit.
- Chronic brain failure is distinct from acute brain failure (ie, delirium); the initial diagnosis cannot be made if delirium is present. Nevertheless, dementia and delirium are frequently encountered in tandem. As illustrated by the preceding case example, dementia is a risk factor for delirium, and delirium can accelerate the progression of dementia.

- Diagnosis and management of major neurocognitive disorder spans multiple medical specialties, including neurology, psychiatry, geriatrics, and general internal medicine, yet no one specialty clearly "owns" the diagnostic class. This can complicate treatment and disposition planning.
- The nature of their symptoms makes people with dementia more dependent and vulnerable, both socially and in terms of physical and mental health.

### *Prevalence/Incidence*

- Within the United States, approximately 5.7 million people are living with dementia.[5]
- As the population worldwide continues to age, the number of individuals at risk will also increase, particularly among the very old.
  - The prevalence doubles every 5 years after the age of 65, with more than 20% of 80-year-olds having moderate-to-severe dementia.[6]
- AD is the most prevalent subtype of dementia, accounting for 60% to 80% of cases.[5]
- ED-based studies of cognitive impairment report that up to 70% of older adults seen with cognitive impairment have undiagnosed dementia.[7]

### *Physiology/Pathophysiology*

The clinical syndrome of major neurocognitive disorder can be caused by several different pathophysiological processes that alter or damage nerve cells and synapses in the brain. The diagnostic category is broken down into corresponding subtypes based on clinical, genetic, and neuropathological features. The most common of these is AD, followed by vascular dementia, dementia with Lewy bodies, and frontotemporal dementia (**Table 1**). Less common causes include Huntington disease, normal pressure hydrocephalus, Parkinson disease, traumatic brain injury, substance/medication use, human immunodeficiency infection, and prion disease.[8]

The clinical symptoms and pathophysiological processes of these diseases overlap significantly. For example, research indicates that at least one-third of AD cases are complicated by some degree of vascular pathology.[9] Determining what specific subtype of major neurocognitive disorder a patient has can be important for prognosis and long-term treatment planning. For the emergency physician evaluating a patient with cognitive impairment in the ED, however, it is more important to differentiate between acute/potentially reversible causes and chronic/irreversible ones.

### *Clinical Characteristics*

Dementia subtypes can be difficult to diagnose clinically because of their multifactorial causes, overlapping symptoms, and inconsistent clinical presentations. Within the category, the most commonly used diagnosis code is Dementia Not Otherwise Specified,[10] which likely reflects these diagnostic challenges.

Fortunately, patients usually present to the ED with an existing diagnosis of dementia; the initial diagnosis is not commonly made by emergency physicians. Nevertheless, an understanding of the clinical characteristics of dementia can aid the emergency physician in managing the patient with chronic brain failure.

Although the hallmark of dementia is memory loss, symptoms typically involve multiple domains of brain function:

1. Complex attention
   - Experiences difficulty processing multiple stimuli (television, radio, conversation)
   - Gets easily distracted by competing events in the environment

**Table 1**
**The most common causes of major neurocognitive disorder**

| Disease | Pathology | Time Course | Clinical Features |
|---|---|---|---|
| Alzheimer's disease | Characterized by plaques, tangles, and neuronal loss. | Insidious onset. Slow progressive cognitive and functional decline. | Almost always includes neuropsychiatric symptoms in later stages. |
| Vascular dementia | Results from cerebrovascular disease. | Stepwise progression with variable rates of decline. | Often associated with focal neurologic signs such as spasticity, hemiparesis, and extrapyramidal signs. Apathy and depression are common. |
| Dementia with Lewy bodies | $\alpha$-Synuclein aggregates in neurons. | Progressive cognitive decline. | Includes fluctuating cognition, visual hallucinations, parkinsonism, rapid-eye movement sleep disorder, and hypersensitivity to antipsychotics. |
| Frontotemporal dementia (includes Pick disease) | Focal degeneration of frontal and temporal lobes. Involves hyperphosphorylated tau protein inclusion bodies. Knife-edge atrophy on MRI. | Typically a more rapid rate of decline than with Alzheimer's disease. | Progressive change in personality, behavior, and language. Motor impairment syndromes co-occur. |

- Is unable to perform mental calculations

2. Executive functioning
   - Abandons complex projects
   - Needs to focus on one task at a time
   - Needs assistance with activities of daily living and making basic decisions
3. Learning and memory
   - Repeats self in conversation, often within the same conversation
   - Cannot keep track of short lists of items such as shopping lists or plans for the day
   - Requires frequent reminders to orient to task at hand
4. Language
   - Experiences significant difficulties with expressive and/or receptive language
   - Often uses vague, general phrases such as "that thing" and "you know what I mean"
   - Prefers general pronouns rather than names
   - With severe impairment, may not recall names of friends and family, and may lose fluent language
5. Perceptual-motor

- Struggles with previously familiar activities (using tools, driving a car)
- Gets lost in familiar environments
- Often gets more confused at dusk, when shadows and lowering levels of light change perceptions

6. Social cognition
   - Exhibits behavior clearly out of acceptable social range
   - Demonstrates lack of sensitivity to social standards of modesty in dress and restraint in political, religious, or sexual topics of conversation

In addition to impairments in the preceding cognitive domains, dementia can involve noncognitive neuropsychiatric symptoms, which include the following:

1. Mood disturbance (depression, irritability)
2. Psychosis (hallucinations, delusions)
3. Agitation
4. Aggression
5. Apathy
6. Sleep disturbance
7. Disinhibition

Despite being almost universally present in chronic brain failure, noncognitive neuropsychiatric symptoms have not been included in the diagnostic criteria for dementia in the current classification system (**Box 1**).

**Box 1**
**Major neurocognitive disorder, DSM-5 diagnostic criteria**[8]

A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on
   1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a significant decline in cognitive function; and
   2. A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

B. The cognitive deficits interfere with independence in everyday activities (ie, at a minimum, requiring assistance with complex instrumental activities of daily living such as paying bills or managing medications).

C. The cognitive deficits do not occur exclusively in the context of a delirium.

D. The cognitive deficits are not better explained by another mental disorder (eg, major depressive disorder, schizophrenia).

## ASSESSING PATIENTS

### *Rule Out Delirium*

Delirium can be life-threatening, and patients with dementia are at increased risk for delirium.[12] The acute symptoms of delirium may be similar to the chronic symptoms of dementia. Therefore, the emergency physician must rule out delirium, starting by determining whether a patient's cognitive impairment is acute or chronic.

- Delirium is a syndrome characterized by the following:
  - Disorientation (confusion, inability to name time or place)

- Impaired attention (easily distracted, unable to complete simple tasks)
- A fluctuating state of consciousness (periods of alertness alternating with somnolence)

- Delirium usually results from *acute* conditions, such as the following:
  - Medication adverse effects
  - Acute medical disorders, such as infections or metabolic abnormalities
  - Substance intoxication or withdrawal
- Delirium can include neurocognitive symptoms, such as the following:
  - Depressed mood and affect
    - Slowed movement and appearance
    - Limited range of emotional expression
  - Psychosis
    - Delusions (firmly held false beliefs, such as paranoia)
    - Hallucinations (the perception of an external stimulus, such as a sound or a vision, without an actual external stimulus)
  - Agitation
    - Excited behavioral activity, usually accompanied by fear, anger, or extreme anxiety

Distinguishing between acute and chronic etiologies of neurocognitive impairment can be challenging.[13] The major clinical distinctions between delirium and dementia are summarized in **Table 2**.

It is rare for geriatric patients to present with a new-onset primary psychotic disorder, such as schizophrenia. In an older patient with no prior history of neuropsychiatric symptoms, a medical cause should be assumed until proven otherwise.[14]

### *Assess for Pain*

The inability to successfully communicate pain in severe dementia is a major barrier to effective treatment.[15] The behavioral indicators of pain have long been recognized, and many clinical tools have been developed to assess pain in older patients, including those with dementia. Behaviors including agitation and aggression can be considered as signs of pain.[16] However, in patients with major neurocognitive disorder, these signs may be interpreted as neuropsychiatric symptoms of dementia rather

**Table 2**
**Major distinctions between delirium and dementia**

| Characteristic | Delirium | Dementia |
|---|---|---|
| Symptom onset | Rapid, over hours or days | Gradual, over months or years |
| Disease course | Transient, usually limited | Chronic, persistent |
| Attention | Impaired, distracted | Usually intact, but may be impaired in advanced disease |
| Consciousness, sensorium | Altered, fluctuating | Usually alert and stable |
| Hallucinations | Often present, typically visual | Usually absent, but can be present in advanced disease |
| Prognosis | Usually reversible | Progressively deteriorating, no known cure |

*Adapted from* The Geriatric Emergency Department Guidelines, 2013.

than of pain.[17] In order to avoid missing this diagnosis, pain should always be considered as a potential precipitant in any patient with dementia who presents with neuropsychiatric symptoms.

### *Consider other Diagnoses*

After the patient has been assessed for delirium and pain, other reversible causes of dementia should be considered. Dementia of relatively recent onset has a higher likelihood of a potentially reversible etiology, underscoring the importance of obtaining a careful history. Some of these conditions include the following:

1. Medications that may affect mentation (sedatives, some anticholinergics, and hormone replacement)
2. Major depressive disorder (the term "pseudodementia" refers to neurocognitive impairment that results from major depression)
3. Normal pressure hydrocephalus
4. Vitamin B12 and folate deficiency
5. Thyroid disease
6. Benign tumors
7. Subdural hematoma
8. Infectious diseases (eg, syphilis)

It should be noted that the concept of "reversible dementia" is controversial in the literature. Some clinicians consider these conditions to be a form of delirium. One study estimated that only 1.5% of all dementias are reversible,[18] and routine screening for uncommon reversible causes of dementia is considered by some to be low yield.[19]

### *Clinical Tools and Scales*

Despite its prevalence in the ED, neurocognitive impairment frequently goes unrecognized.[20] This has led some investigators to call for greater use of cognitive assessment tools in the ED, including the use of dementia screens after delirium has been ruled out.[21]

The Mini Mental State Examination (MMSE) and the MoCA are the most widely used brief screening tools for cognitive impairment, but because they require approximately 15 minutes to administer, they are impractical for routine use in the ED.

The Abbreviated Mental Test 4 (AMT-4), Short Blessed Test, Brief Alzheimer's Screen (BAS), the Six Item Screener, and Ottowa 3DY (O3DY) are some of the ultrabrief screening instruments developed to identify geriatric patients with cognitive dysfunction in the ED.[22] The AMT-4 has been shown to be the most accurate ED screening instrument to rule in the diagnosis of dementia, whereas the BAS is the most accurate screener to rule it out.[23]

The ADEPT tool was developed as an easy-to-use, point-of-care tool to assist emergency physicians in the care of older patients with confusion and agitation.[11] It is an open-access, Web-based tool available on the American College of Emergency Physicians emPOC mobile device app. It was designed to be used by clinicians on shift. To help ensure thorough consideration of the multiple etiologies of neurocognitive symptoms in older patients, it highlights 5 core concepts: assess, diagnose, evaluate, prevent, and treat (**Box 2**). However, none of the preceding tools can be used to definitively diagnose dementia, and none of them differentiate the severity of cognitive impairment. Many require special training to administer, involve complex calculations in the scoring process, assume a cooperative patient, or have other barriers to their widespread use in the ED setting. In addition, some cognitive assessment instruments may not be valid across lower socioeconomic and limited health literacy

populations.[24] Further refinement of clinical instruments and the application of technology to the assessment process may accelerate the use of standardized tools to reduce the rate of unrecognized neurocognitive impairment in the ED.

**Box 2**
**Principles of the ADEPT tool**

ASSESS
- Perform a thorough evaluation to determine the underlying cause.
- The history, medication review, and collateral information are crucial.
- Perform a thorough physical examination.

DIAGNOSE
- Screen for delirium in any agitated or confused older patient.
- Screen for underlying major neurocognitive disorder (dementia).

EVALUATE
- Perform a thorough, focused medical workup for agitation or confusion.
- General tests for most patients
- Specific, targeted testing and evaluation

PREVENT
- Individual patient measures to prevent or manage delirium
- Hospital and systems-based measures to prevent or manage delirium

TREAT
- Take a multimodal approach to treatment.
- Use verbal de-escalation principles.
- If needed, start with oral medications.
- Carefully consider the use of intramuscular or intravenous medications.
- Avoid benzodiazepines if possible unless in withdrawal.
- Be cautious to prevent harm and minimize side effects.

*From* Shenvi C, Kennedy M, Austin CA, Wilson MP, Gerardi M, Schneider S. Managing Delirium and Agitation in the Older Emergency Department Patient: The ADEPT Tool. Ann Emerg Med. 2020;75(2):136-145.

## TREATING PATIENTS

Emergency physicians frequently struggle with management of the acutely agitated patient with dementia. Because of the increased risks associated with use of medications in older patients, clinicians should consider using nonpharmacological interventions as first-line therapy.

The best initial approach is to prevent agitation before it starts. This begins with identification of any potentially modifiable precipitants to agitation.[25] These include the following:

- Delirium
- Medication side effects
- Pain
- Physical needs (hunger, need for toileting)
- Emotional needs (separation from family, need for support)
- Environmental factors (noise, overcrowding, understimulation)
- Caregivers (including family) who are inflexible in their approach to the patient

Preventive measures include the following:

- Gentle verbal redirection, tailored to the patient's personality and emotional state

- Frequent reorientation to their surroundings
- Staff members reintroducing themselves at each encounter
- Well-lit rooms to minimize misperceptions
- One-to-one bedside companions
- Manual activities (coloring, folding, jigsaw puzzle)

## TREATMENT OF ACUTE AGITATION

No drug is approved by the Food and Drug Administration (FDA) to treat agitation associated with dementia, and medications should be used only as a last resort. Antipsychotics are the most frequently prescribed medications for this purpose,[26] despite an FDA black-box warning of increased mortality when used in older adults with dementia (odds ratio 1.7).[27] Other serious side effects include the following: extrapyramidal symptoms (EPS), sedation, tardive dyskinesia (TD), gait disturbances, falls, anticholinergic side effects, and cerebrovascular events.

### *Antipsychotics*

Despite the FDA warning, most clinicians recognize that antipsychotics are effective psychotropic agents for controlling severe agitation, aggressive behavior, and psychosis. Second-generation antipsychotics (SGA) have the best evidence to support their use in the treatment of agitation associated with dementia. Evidence suggests that risperidone and olanzapine are useful in reducing aggression, and that risperidone reduces psychosis.[28] Haloperidol, a first-generation antipsychotic, has been shown to be useful in reducing aggression,[29] but is more likely to cause EPS and TD than the SGAs. Despite their modest efficacy, none of these drugs should be used routinely to treat patients with dementia with aggression or psychosis unless there is severe distress or risk of physical harm, and only after case-by-case consideration of benefits versus risks.

Special care should be taken when using antipsychotics in patients with Lewy body dementia or Parkinson dementia. These diseases both involve dopamine dysfunction. Because all antipsychotics have some degree of dopamine blockade, their use could worsen or precipitate extrapyramidal symptoms.[30] Antipsychotics with the least amount of dopamine blockade, such as quetiapine, may be better tolerated by these patients. A newer antipsychotic, pimavanserin, may reduce psychosis symptoms without worsening motor function and is FDA approved for use in Parkinson disease.[31]

### *Benzodiazepines*

Care must be taken when prescribing benzodiazepines to older persons. The half-life of benzodiazepines may be increased dramatically in late life, with diazepam having a half-life nearing 4 days in persons in their 80s.[32] Older persons are also more susceptible to the potential side effects of benzodiazepines, such as memory impairment, fatigue, drowsiness, motor dysfunction, and falls. Some investigators argue that short-acting benzodiazepines may be used temporarily for acute agitation or agitation associated with anxiety.[26] In general, however, they are best avoided.[33]

## TREATMENT OF CHRONIC SYMPTOMS

The following medications are not typically used on an as needed basis for control of acute neuropsychiatric symptoms, but they are frequently prescribed to help manage chronic symptoms.

### *Cholinesterase Inhibitors*

Donepezil, galantamine, and rivastigmine are cholinesterase inhibitors (ChEI) that are FDA approved for use in mild to moderate AD but are commonly used in nearly all dementia subtypes. When taken daily, they can enhance cognition, reduce behavioral changes, and delay functional decline in persons with major neurocognitive impairment; however, they do not affect the progression of disease. Common side effects include nausea, vomiting, and diarrhea.

### *Memantine*

Memantine is an N-methyl-D-aspartate (NMDA) receptor antagonist that can also have beneficial effects on cognition, behavior, and function in dementia. It is frequently prescribed in combination with a ChEI. It generally has few adverse effects.

### *Antidepressants*

Depression is a common neuropsychiatric symptom of dementia, and use of antidepressants among patients with major neurocognitive disorder is widespread. Despite limited evidence for the efficacy of these medications in treatment of depression in patients with dementia, some investigators and clinical organizations recommend their use.[34] Selective serotonin reuptake inhibitors, such as sertraline and citalopram, are most commonly used because of their favorable side-effect profile. Trazodone, an antidepressant with mixed serotonin, histamine, and $\alpha$-adrenergic activity, is sometimes used to treat agitation in chronic brain failure, despite little supporting evidence.[35] Trazodone and mirtazapine are often prescribed for sleep.

## DECISION-MAKING CAPACITY

The cognitive deficits of chronic brain failure often impair a patient's decision-making capacity and ability to provide informed consent. Before administering any medication or treatment, an effort to obtain informed consent should be made to the extent possible. Many patients have a designated decision-maker, such as a guardian or power of attorney; however, all patients with dementia cannot be assumed to have impaired capacity. To demonstrate decision-making capacity, the patient must (1) communicate a consistent choice, (2) understand the relevant information, (3) appreciate the current situation and its consequences, and (4) manipulate information rationally.[36] Decision-making capacity is situation-specific, and some patients who carry a diagnosis of major neurocognitive disorder may still be able to meet those 4 criteria in a given situation.

Treatment decisions and disposition planning for patients with advanced dementia should take into consideration the severity of their neurocognitive impairment and ideally should be guided by the goals of care for that individual. Unfortunately, the goals of care are often unclear when making disposition plans; 56% to 99% of older adults do not have advance directives available at ED presentation.[37]

## DISPOSITION

Despite the potential to optimize a patient's quality of life during the disease course, dementia remains a progressively debilitating disorder that ultimately results in death. Of persons in the United States who die with dementia, approximately 16% die in hospitals.[38] In one study, 19% of nursing home patients who died with advanced dementia had a burdensome transition near the end of life (hospitalization in the last 3 days of

life, multiple hospitalizations in the last 90 days of life, or care in multiple nursing homes after hospitalization in the last 90 days of life).[39]

Patients with dementia are at increased risk of hospitalization, despite no true medical indication. This may be due to a decrease in community supports and resources to safely care for persons with major neurocognitive impairments.[40] It has also been associated with "caregiver burnout," which occurs when someone caring for a patient with dementia is overwhelmed by the agitation, psychosis, and other neuropsychiatric symptoms of the disease.[41] As seen in the preceding case example, an entire residential care facility also can be overwhelmed by the severity of a patient's symptoms to the point where they no longer feel capable of safely caring for that patient. Without any clear discharge options, many patients with dementia are admitted to inpatient medical services despite lacking medical necessity.[42]

Effective treatment of neuropsychiatric symptoms can help facilitate the patient's return to their home or care facility. This may include appropriate pain management. Surveys have indicated that the goal of care for most patients is comfort.[43] If available and appropriate, a palliative care consultation or hospice referral can be considered. Patients with advanced dementia who are enrolled in hospice have been shown to have a lower risk of dying in the hospital[44] and of being hospitalized in the last 30 days of life.[45] In addition, their families have greater satisfaction with care.[46]

## DISCUSSION

The term *chronic brain failure* has been used here in an attempt to better represent the condition to which it refers. The term emerged in the 1970s as an alternative to *dementia*, which was thought to be imprecise and, when used as a lay term, stigmatizing and potentially pejorative.[47] More recently, the term *neurocognitive disorders* has been officially adopted,[8] but it excludes the emotional, behavioral, and other noncognitive aspects of brain function that can be part of the syndrome. Like chronic heart and kidney failure, chronic brain failure describes a progressive loss of organ function that is typically irreversible but whose symptoms can be mitigated with proper management.

Unlike with other organ failure syndromes, however, little progress has been made to advance our understanding of dementia, despite tremendous research efforts. No new AD therapies have won federal approval since 2003, and AD clinical trials have had a 99% failure rate. Since the introduction of tacrine in 1993, only 5 drugs have been approved by the FDA to treat AD, and those merely alleviate symptoms, such as memory loss and confusion; they do not prevent, slow, or reverse the disease.[48]

Some promising developments have been made in nonpharmacologic approaches to chronic brain failure. Some of the most significant of these have been in the area of prevention: for example, linking aerobic exercise to lower rates of AD.[49] Others involve conceptualizing dementia as a spectrum syndrome, with interventions tailored to the degree of impairment, and to the personality and preferences of each individual patient. Interactions that give a patient a sense of choice and independence are more likely to be successful.[50] The diagnosis of dementia does not imply specific functional impairments, and many individuals with chronic brain failure live rich, active lives. Treatment goals should be focused on helping patients cope with the negative aspects of their illness so that they can live the best life that they can.

Chronic brain failure involves physical changes in the brain that can degrade a person's ability to modulate the behavioral expression of emotion. For example, a person with dementia who is receiving personal hygiene care might feel embarrassed or threatened and react aggressively. Fear, anxiety, and insecurity in someone with severe neurocognitive impairment are often expressed as paranoia and/or aggression.

Effective communication, therefore, requires an understanding of the patient's underlying emotional state and an adaptation to their internal experience of reality.

Use of logic and reason is rarely successful when attempting to redirect a patient with severe neurocognitive impairment. Efforts to alter their reality can create frustration and confusion and lead to unsafe behaviors. Should a patient become agitated, the clinician should identify what the patient sees as their own needs in the moment and adapt their approach to the underlying emotion. Distraction or redirection of the patient's thoughts can also be effective; for example, gently changing the subject or asking about something or someone from their past.

The use of deceit in dementia care, also known as "therapeutic lying," is widespread but controversial. One survey of nursing home staff showed that 96% of respondents (n = 112) across disciplines lied to cognitively impaired residents.[51] Despite this widespread practice, many caregivers feel uncomfortable or guilty about deceiving patients, even if it is perceived to be in their best interests.[52] It can be viewed as demeaning and disrespectful to the patient, and inconsistent with the principle of patient autonomy. In the preceding case example, the patient asked for his wife and was told that she was out of town; however, if he was told that she was dead each time he asked for her, he might experience recurrent grief. Thus, deception can be used to the patient's benefit. In the absence of clear ethical guidelines, the provider is left to use his or her clinical judgment in deciding whether to use deception as part of the management approach. When making that decision, consideration should be given to the patient's degree of memory impairment, and the deception should occur only respectfully and in the best interest of the patient.

The stress of caring for patients with chronic brain failure is well documented.[53] The neuropsychiatric symptoms of severe dementia have a profound physical and psychological impact on both professional and informal caregivers. Psychological distress and burnout are common.[54] This in turn can have negative effects on the caregiver's relationship with the patient and is the primary driver of long-term placement.[55] Emergency physicians can play a role in identifying caregiver burnout, encouraging healthy stress-release practices among caregivers, and directing them to appropriate resources, such as those offered by the Alzheimer's Association (www.alz.org) and the Family Caregiver Alliance (www.caregiver.org).

## DISCLOSURE

The author has nothing to disclose.

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