Diagnosis and Management of Delirium near the End of Life

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Delirium is a common and distressing symptom that constitutes a significant challenge for end-of-life care. However, reliable techniques are available for the diagnosis of delirium, and effective therapies exist as well. This consensus paper uses a case-based format that begins with an overview of the definition and presentation of delirium. Next, strategies for diagnosis are suggested, with attention to the unique challenges that clinicians face in pursuing a diagnostic work-up for patients near the end of life. The paper concludes with a review of therapeutic options.

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Patients near the end of life may face a variety of distressing symptoms. Of these, perhaps none is as detrimental to quality of life and is as difficult to diagnose and manage as delirium. Delirium is characterized by a disturbance of consciousness, cognition, and perception, with a course that may wax and wane over a period of hours (1).

Delirium occurs in 28% to 83% of patients near the end of life, depending on the population studied and the criteria used (2–6). This syndrome is a challenge for physicians for several reasons. First, it may frighten patients and may cause as much distress as do pain and other symptoms. Second, families may regret the premature separation from a patient who can no longer communicate. Third, delirium may also be a predictor of approaching death for some patients (7). Fourth, delirium robs patients of valuable time and curtails opportunities to make final choices and plans. For all of these reasons, delirium can be a daunting obstacle to good end-of-life care if not addressed appropriately.

The skills required for diagnosing and managing delirium at the end of life can be a part of every clinician's repertoire. Prompt recognition and appropriate treatment of delirium can improve patient comfort, optimize quality of life, and enhance the leave-taking process for the patient and family. In this paper, we present strategies for the diagnosis and management of delirium, beginning with a description of a patient with mental status changes. We then describe steps for diagnosing delirium and evaluating potential causes, and conclude by discussing strategies for prevention and treatment.

Diagnosis of Mental Status Changes

In evaluating mental status changes near the end of life, as with other clinical decisions, the patient's and family's goals for care are of central importance. Some patients may wish to preserve their ability to communicate, while others focus on comfort, perhaps at the expense of alertness. For the former patients, diagnostic evaluation and treatment would be appropriate, but for the latter, any diagnostic or therapeutic interventions will be more circumscribed.

Mrs. Ghoduay is a 42-year-old woman with ovarian cancer metastatic to the peritoneum, liver, and lung. She has become increasingly agitated over the past week, and her husband, daughter, and nurse believe that these changes are due to pain. However, increases in her opioid dose have produced unacceptable sedation, and she is admitted for evaluation. Her physician, Dr. Marks, finds her to be somnolent and unresponsive to direct questioning. He is unable to assess her pain or other symptoms.

Often delirium is obvious, but up to half of delirium episodes are not noted by clinicians (8, 9). Delirium may be missed because the constellation of features that define it—acute onset, inattention, altered level of consciousness, and cognitive impairment—are not al-

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ways readily apparent. Indeed, their detection often requires a careful history and bedside evaluation.

To diagnose delirium, clinicians must first have an accurate picture of the patient's baseline status. Therefore, Dr. Marks can first inquire about Mrs. Ghoduay's mental status at several points in the past. This may require tenacious detective work and questioning of several family members. For instance, Dr. Marks could ask for specific examples of Mrs. Ghouduay's interactions with friends and family a day ago, or a week ago. He might also assess her ability to participate in conversations, or to recognize family. This line of questioning can be valuable in identifying the typical fluctuating course of mental status changes that is seen in delirium.

Further questioning confirms that Mrs. Ghoduay's decline in mental status began approximately 1 week before admission. Since then, her mental status has fluctuated dramatically, with periods of lucidity punctuated by episodes of somnolence and agitation. During that period, her morphine dosage was increased from 100 mg/24 hours to approximately 400 mg/24 hours, including rescue doses. Her oral intake has been limited. On the basis of this information, Dr. Marks believes that delirium is a possible cause of her somnolence.

Evaluating Possible Delirium

Delirium is a clinical diagnosis made at the bedside. To determine whether Mrs. Ghoduay's mental status changes are due to delirium, Dr. Marks will need to rely almost entirely on clinical skills to identify the two features of delirium: cognitive impairment and deficits in attention. Of the tests to assess cognitive function, the Mini-Mental State Examination (MMSE) (10) has the advantage of general availability and familiarity to most clinicians and, in most settings, is recommended as the test of choice. To assess attention, the MMSE's immediate repetition of three objects and a backwards-spelled word ("d-l-r-o-w") items can be very useful. Corroboration can be sought in the digit span test, in which inability to repeat at least five numbers forward without errors indicates inattention (11).

These tests of cognition and attention support a diagnosis of delirium, but they are not themselves diagnostic. In addition, Dr. Marks can also use one of several instruments that have been developed to distinguish delirium from other causes of altered mental status (**Table 1**). The most widely used include the Confusion Assessment Method (**Table 2**) (12), which systematizes bedside observations; the Memorial Delirium Assessment Scale (13); the Delirium Rating Scale (DRS) (14, 15); and the Delirium Symptom Interview (DSI) (16). Each has its own strengths and limitations, and the choice among them depends on the goals of use (**Table 1**).

Mrs. Ghoduay's mental status seems to improve shortly after admission. Her MMSE score is 16, but she cannot perform the serial sevens task or spell "world" backwards. She is able to repeat only two digits in the digit span test on several occasions. Throughout the interview, Mrs. Ghoduay is easily distracted and often appears to drift off to sleep. Later the same day, she can be aroused only with difficulty, and attempts to repeat the same tests are unsuccessful.

Characterizing Delirium and Identifying Causes

On the basis of the results of formal testing and Mrs. Ghouduay's fluctuating clinical course, Dr. Marks believes that Mrs. Ghoduay's mental changes are most likely due to delirium. Delirium may present as one of three major types: hyperactive, hypoactive, or mixed. Hyperactive or "agitated" delirium is characterized by agitation and hallucinations and is often readily apparent. In contrast, hypoactive or "quiet" delirium presents as a decreased level of consciousness with somnolence and can be mistaken for sedation due to opioids or obtundation in the last days of life. Finally, delirium of mixed type, alternating between agitated and quiet forms, may also be difficult to recognize. Of these, Mrs. Ghoduay's presentation is most consistent with quiet delirium.

Even when delirium is recognized, a cause is often elusive. Although delirium can be due to a single cause, a multifactorial etiology is most common in the palliative care setting (4). Therefore, once delirium is diagnosed, possible causes should be sought in the medication history, physical examination, and laboratory tests. The clinician's task is to identify potential causes that are easily treatable and offer the best chance of improved quality of life (4, 17) (**Table 3**).

Medication History

Available data suggest that medication effects are the most common cause of delirium both in the general patient population (8) and in patients near the end of life (4). Several medications commonly used in the pal-

Table 1. **Available Instruments Used To Evaluate Delirium***

Description	Domains	Validation	Reference Standard	Reliability	Feasibility
Confusion Assessment Method (12)					
Nine operationalized criteria from DSM-III-R scored according to CAM algorithm. Shortened version uses four criteria. Based on observations made during interview with MMSE, by trained lay or clinical interviewer.	1. Onset/course 2. Attention 3. Organization of thought 4. Level of consciousness 5. Orientation 6. Memory 7. Perceptual problems 8. Psychomotor behavior 9. Sleep–wake cycle	Sensitivity = 0.94 − 1.0 (26 delirious patients); specificity = 0.90 − 0.95 (30 controls without delirium) Convergent agreement with four other cognitive measures Ability to distinguish delirium and dementia verified	Geriatric psychiatrists' diagnoses based on clinical judgment and DSM-III-R criteria	Inter-rater: κ = 1.0 overall	Observer-rated: 10–15 minutes for cognitive testing and completion of rating
Delirium Rating Scale (14, 15)					
10-item rating, with additive score 0–32, designed to be completed by a psychiatrist after complete psychiatric assessment. Can be used to rate severity.	1. Onset/course 2. Cognitive status 3. Perceptual problems 4. Delusions 5. Psychomotor behavior 6. Emotional lability 7. Sleep–wake cycle 8. Physical disorder	No overlap in scores between delirious group (n = 20) and 3 control groups: demented (n = 9), schizophrenic (n = 9), and normal (n = 9) Convergent agreement with two other cognitive measures Ability to distinguish delirium and dementia verified	Consult-liaison psychiatrist's diagnosis based on DSM-III criteria	Inter-rater: Intraclass correlation coefficient = 0.97	Observer-rated: based on lengthy interview and detailed assessment (time not specified)
Delirium Symptom Interview (16)					
Includes interview with brief cognitive assessment and rating scale for 7 symptom domains of delirium, by trained lay or clinical interviewer.	1. Course 2. Organization of thought 3. Level of consciousness 4. Orientation 5. Perceptual problems 6. Psychomotor behavior 7. Sleep–wake cycle	Sensitivity = 0.90; specificity = 0.80 (30 "cases," 15 noncases, 3 borderline, 2 disagreements by psychiatrist and neurologist) Ability to distinguish delirium and dementia not tested	Psychiatrist's and neurologist's assessments based on presence of any 1 of 3 "critical symptoms" (disorientation, disturbance of consciousness, or perceptual disturbance)	Inter-rater: κ = 0.90 overall	Observer-rated in part; ≥15 minutes for interview, plus additional time for completion or rating (not specified)
Memorial Delirium Assessment Scale (13)					
10-item scale, with additive score 0–30 using cognitive testing and behavioral observations, by experienced mental health professionals. Designed for rating delirium severity, not screening or diagnosis.	1. Level of consciousness 2. Orientation 3. Memory 4. Digit span 5. Attention 6. Organization of thought 7. Perceptual problems 8. Delusions 9. Psychomotor behavior 10. Sleep–wake cycle	Sensitivity = 0.82; specificity = 0.75 (with score of 10; n = 33: 17 with delirium, 8 with dementia, 8 with other psychiatric problems)	Consult-liaison psychiatrist's diagnosis using Delirium Rating Scale, MMSE, and Clinician's Global Rating of delirium severity	Inter-rater: intraclass correlation coefficient = 0.92 overall	Observer-rated in part; ≥10 minutes for administration

* CAM = Confusion Assessment Method; DSM-III = *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition; DSM-III-R = *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition revised; MMSE = Mini-Mental State Examination.

liative care setting deserve attention (**Table 3**). Opioids can cause both substantial alterations in mental status (18) and more subtle, temporary changes in cognition and attention (19, 20). These changes may become pronounced in the setting of renal failure, particularly with opioids, such as morphine, that have active metabolites (21, 22). Meperidine is associated with a higher risk for delirium because its active metabolite, normeperidine, is cleared slowly (23). Other than meperidine, few data suggest that one opioid is associated a higher incidence of delirium than others.

A variety of other medications might also contribute

Table 2. **Confusion Assessment Method (12)***

Feature 1. Acute onset and fluctuating course
This feature is usually obtained from a family member or nurse and is shown by positive responses to the following questions: Is there evidence of an acute change in mental status from the patient's baseline? Did the (abnormal) behavior fluctuate during the day, that is, tend to come and go, or increase and decrease in severity?

Feature 2. Inattention
This feature is shown by a positive response to the following question: Did the patient have difficulty focusing attention, for example, by being easily distracted or having difficulty keeping track of what was being said?

Feature 3. Disorganized thinking
This feature is shown by a positive response to the following question: Was the patient's thinking disorganized or incoherent, such as rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?

Feature 4. Altered level of consciousness
This feature is shown by any answer other than "alert" to the following question: Overall, how would you rate this patient's level of consciousness: alert (normal), vigilant (hyperalert), lethargic (drowsy, easily aroused), stupor (difficult to arouse) or coma (unable to arouse)?

* Scoring: The diagnosis of delirium requires a present/abnormal rating for features 1 and 2 and for either 3 or 4.

to Mrs. Ghoduay's delirium (**Table 3**). These include sedative drugs, such as benzodiazepines and other sleeping medications; gastrointestinal drugs, such as cimetidine, ranitidine, and metoclopramide; and many nonsteroidal anti-inflammatory agents, corticosteroids, and medications with prominent anticholinergic effects, such as diphenhydramine, hydroxyzine, scopolamine, and amitriptyline (17, 24, 25).

In evaluating medications, it is also important to consider over-the-counter medications, including many of those described above and in **Table 3**. In addition, the use of complementary medications is common, and clinicians may be unaware of their use (26, 27). The side effects and interactions of these agents are poorly understood, and clinicians may wish to screen for these medications as a routine part of a delirium evaluation.

When medications are identified as precipitating factors, most can be switched (for example, cimetidine and ranitidine) or their dose can be tapered (for example, corticosteroids). Similarly, in the case of delirium due to opioids, it is sometimes possible to enhance pain relief while improving mental status by rotating to a different opioid, at a reduced equianalgesic dose (18, 28, 29). Although no randomized clinical trials support opioid rotation, expert opinion suggests that this practice can be useful.

Careful medication review often identifies several likely contributing medications, some of which may have an important role in pain and symptom management. Because delirium is multifactorial, it is unrealistic to expect that a single medication change will completely resolve the delirium. Instead, clinicians can limit the number of medications whenever possible and substitute agents with more benign side effect profiles.

Physical Examination

Information from a careful history should be supplemented by a physical examination. Some of the most important findings from a physical examination are those indicating that a patient is actively dying, such as hypotension and periods of apnea. The clinician should be alert for other signs on physical examination, including fever, focal or lateralizing neurologic findings, frontal release signs, or asterixis. Although the predictive value of these findings in dying patients is not known, their presence can help to guide the diagnostic evaluation.

Clinicians can also identify volume depletion, which

Table 3. **Contributors to Mental Status Changes near the End of Life**

- Medical contributors
  - Infection
  - Brain metastases
  - Hepatic encephalopathy
  - Renal failure
  - Hypercalcemia
  - Hyponatremia
  - Disseminated intravascular coagulation
  - Hypoxemia
  - Volume depletion
  - Infections (e.g., urinary tract infections, pneumonia)
  - Atelectasis with hypoxemia
  - Immobilization
- Psychosocial contributors
  - Depression
  - Vision/hearing impairment
  - Pain
  - Emotional stress
  - Unfamiliar environment
- Medications commonly used at the end of life
  - Opioids
  - Corticosteroids
  - Metoclopramide
  - Benzodiazepines
  - Hydroxyzine
  - Diphenhydramine
  - Nonsteroidal anti-inflammatory drugs
  - H_2-Blockers
  - Tricyclic antidepressants
  - Scopolamine

may be a common cause of delirium near the end of life (4). However, the treatment of volume depletion need not be automatic. Fluid replacement using a nasogastric tube or intravenous catheter may impose additional burdens on the patient and his or her family. Other, less invasive interventions, such as hypodermoclysis (30), pose fewer burdens. Nevertheless, all of these may prolong the patient's life, which may not be consistent with his or her goals.

Dr. Marks concludes that Mrs. Ghoduay's delirium is caused in part by diphenhydramine that was prescribed for sleep. In addition, he believes that her delirium might be potentiated by high doses of morphine. However, her family is not satisfied with this description. They are concerned about possible metabolic derangements or metastases that might be treatable, and they urge Dr. Marks to pursue a more aggressive valuation.

Laboratory Evaluation

The decision to search more aggressively for causes of delirium depends on the patient's and family's goals for care, the burdens of an evaluation, and the likelihood that a specific remediable cause will be found. In patients who are actively dying, altered mental status is common (4, 31), and these changes may be resistant to treatment. When death seems to be imminent, it is appropriate to forgo evaluation beyond a history and physical examination and to provide both pharmacologic and nonpharmacologic interventions to ameliorate the symptoms of agitated delirium.

When further evaluation is indicated, data are not available to dictate a "standard" algorithm. Nevertheless, metabolic causes of delirium may be found in up to 18% of terminally ill patients with cancer (4). Therefore, a targeted laboratory assessment might include complete blood count with differential, electrolytes, blood urea nitrogen, creatinine, calcium, magnesium, phosphorus, glucose, urinalysis, and oxygen saturation.

The burden of the evaluation, as well as the potential value of the findings and the goals of treatment, should guide decisions about laboratory or radiologic assessment. The clinical setting must also be considered, since treatment of metabolic derangements may be difficult in the home care setting. Therefore, each diagnostic and therapeutic decision requires careful discussion with the patient and his or her family. Evidence of the patient's prior wishes may be particularly helpful in this regard, and clinicians may wish to discuss treatment options for delirium as part of advance care planning.

Laboratory studies reveal new renal insufficiency but no electrolyte disorder that could explain Mrs. Ghoduay's mental status changes. Oxygen saturation is 93% on 2 L of oxygen delivered by nasal cannula.

Dr. Marks recommends a change from morphine to hydromorphone at a reduced equianalgesic dose. With this new regimen, Mrs. Ghouduay's level of consciousness still waxes and wanes, and her attention span is short. She cannot maintain a conversation and drifts off to sleep frequently. Her MMSE scores range from 10 to 15 over 2 days, and her digit span test score is between 0 and 2. Her family would like to know if there are other options for symptomatic treatment, even though they realize a cause has not been identified.

Prevention and Treatment

Delirium is often a distressing symptom, as are pain, dyspnea, and nausea. Like these symptoms, delirium is also treatable. Because patients' time is limited, it is reasonable to treat delirium before, or in concert with, a diagnostic evaluation. As in pain management, relief of suffering need not be delayed by a search for a cause. Instead, the decision to treat the symptoms of pain or delirium can be made independently of the results of the etiologic evaluation.

The decision to intervene also depends on the degree to which delirium is distressing. For instance, although many hallucinations are disturbing, hallucinations of deceased friends or relatives may sometimes be comforting. In the latter case, families can be reassured. Nevertheless, the course of delirium is highly variable, and clinicians must be alert to the abrupt development of more distressing manifestations of delirium.

Finally, a clinician's choice among therapeutic options will often be made with insufficient data. At the very least, clinicians may find it necessary to base treatment decisions on data from very different patient populations. For this reason, the data discussed below offer a guide for the treatment of delirium at the end of life, but they must be weighed carefully against clinical experience and the results of future research.

Table 4. **Nonpharmacologic Management of Delirium***

Targeted Risk Factor	Standardized Intervention Protocol
Cognitive impairment	Orientation protocol: includes orientation board with name of care team members and day's schedule; reorienting communication Therapeutic activities protocol: provides cognitively stimulating activities 3 times daily, such as current events discussions, structured reminiscence, or word games
Sleep deprivation	Nonpharmacologic sleep protocol: at bedtime, provides warm drink (milk or herbal tea), relaxation tapes or music, and back massage Sleep enhancement protocol: reinforces unit-wide noise reduction strategies (e.g., silent pill crushers, vibrating beepers, reduction in hallway noise) and adjusts schedule to allow sleep (e.g., rescheduling medications, vital sign checks, procedures)
Immobility	Early mobilization protocol: provides ambulation or active range-of-motion exercises 3 times daily; minimizes use of immobilizing equipment (e.g., bladder catheters, physical restraints)
Visual impairment	Vision protocol: offers visual aids (e.g., eyeglasses, magnifying lenses) and adaptive equipment (e.g., large illuminated phone dials, large-print books, fluorescent tape on call bell); daily reinforcement of use of adaptations
Hearing impairment	Hearing protocol: supplies portable amplifying devices, earwax disimpaction, and special communication techniques; daily reinforcement of use of adaptations
Dehydration	Dehydration protocol: provides early recognition and volume repletion measures (i.e., encouraging oral fluids)

* Data obtained from Inouye et al. (32).

Prevention and Nonpharmacologic Treatment

All patients near the end of life can be considered at high risk for delirium, and clinicians should consider preventive strategies that have been proven effective in other settings (32). For instance, protocols designed to encourage cognitive activity and to help patients orient to place, time, and environment can be useful. Sleep can be improved by a combination of nonpharmacologic interventions, such as relaxation and breathing techniques; quiet music at bedtime; and reductions in environmental light, noise, and other factors that may awaken the patient at night. These strategies also minimize the need for sedative medications, which are a significant cause of delirium (17, 25).

Immobility can be ameliorated in some patients by encouraging time out of bed and active range-of-motion exercises, as well as by limiting the use of catheters, restraints, or continuous intravenous infusions. All of these factors are potentially modifiable and can be considered not only for prevention but also as potential targets for the nonpharmacologic treatment of delirium (**Table 4**). These interventions will need to be adapted to the needs of patients near the end of life to make them consistent with patients' goals for care; they will not be appropriate for all patients. However, because these interventions can be initiated by family members in the home, they may have a particularly valuable place in end-of-life care by allowing families to take an active role in maintaining the patient's comfort.

Pharmacologic Treatment

Several nonpharmacologic interventions are initiated, including careful attention to the lighting in Mrs. Ghoduay's room and orientation cues. In addition, Dr. Marks recommends initiating a trial of intravenous haloperidol, and he explains the efficacy of this drug as a neuroleptic agent. However, Mrs. Ghoduay's family is reluctant to agree to this plan, arguing that she is not "crazy." The housestaff caring for her also object. They are concerned that haloperidol is not effective for a quiet delirium such as Mrs. Ghoduay's and that, if anything, it will only make her more sedated.

In most cases, the goal of pharmacologic treatment of delirium should be to bring patients closer to their baseline mental state, not to sedate them or to suppress agitation. Several agents are available (**Table 5**), but few data exist to guide their use in the treatment of patients near the end of life. Therefore, clinicians may prefer to turn first to a medication such as haloperidol, which one randomized, controlled trial has shown to be superior to benzodiazepines for both hyperactive and hypoactive de-

Table 5. **Pharmacologic Management of Delirium: Usual Starting Doses***

Predominantly neuroleptic effects
Haloperidol, 0.5–1 mg every 30 minutes orally (0.5–1 mg every 30 minutes subcutaneously or intravenously, titrate to effect, usual maximal dose not to exceed 3 mg/24 hours)
Olanzapine, 2.5–5 mg orally once daily
Risperidone, 0.5 mg orally twice daily
Predominantly sedative effects
Lorezepam, 0.5–1 mg every 4 hours orally, subcutaneously, or intravenously
Propofol, 10-mg bolus followed by 10 mg/h intravenously
Midazolam, 1–2 mg/h subcutaneously or intravenously

* Titrate dose to effect in all regimens.

lirium in patients with AIDS (33). Haloperidol also has the advantages of a fairly wide therapeutic window, availability in both parenteral and oral preparations, and minimal risk for respiratory depression. Benzodiazepines such as alprazolam have demonstrated some benefit in controlling agitation in elderly patients (34), but they do not necessarily improve cognitive function as does haloperidol (33). Although haloperidol is generally safe and effective, adverse events include dystonia and an initial worsening of delirium symptoms, particularly agitation.

Other agents are available, but even fewer data are available to support their use. Some clinicians have found newer neuroleptic agents to be effective, although most data come from the treatment of agitation in elderly patients. These agents include methotrimeprazine, risperidone, clozapine, and olanzapine (35–38) (**Table 5**). These agents may offer some advantages over haloperidol, such as diminished extrapyramidal effects (olanzapine and risperidone) (39–41) and analgesia (methotrimeprazine) (42). However, these studies are generally designed to test the effectiveness of a medication in treating agitation or aggression in cognitively impaired elderly patients, not delirium in patients near the end of life. Moreover, the time to response in these studies is as long as 12 weeks (36), a time frame that is not relevant to patients near the end of life. Therefore, without extensive experience in this patient population, clinicians may prefer to reserve these agents for use when haloperidol is ineffective.

In some cases, several agents are ineffective and patients require sedation. Several authors have suggested that sedation is required in 9% to 26% of patients with delirium near the end of life (31, 43). Because sedation may pose its own risks, such as decreased interaction with family or respiratory depression, the decision to use sedation should be weighed carefully. When patients and their families experience considerable distress, some have suggested that for these patients the benefits of sedation may outweigh the risks (44). However, a true consensus about this practice has not yet emerged (45).

If sedation is warranted, clinicians can choose an agent that is short acting and that can be easily and rapidly titrated to effect. These agents include lorazepam, midazolam, and propofol (**Table 5**). Lorazepam can be administered by oral, intravenous, and subcutaneous routes and is widely used. However, it has the longest duration of action of these drugs. Propofol offers the advantage of rapid titration (46, 47). It is expensive, however, and its use is often restricted to the intensive care unit or to anesthesiologists. Midazolam may be easier to titrate in some settings (47, 48), but its use may be restricted as well. It is sometimes given subcutaneously, but there are limited data to guide dosing by this route. Without convincing data to support one of these agents, the choice among them will be based on availability and on the requirements of the specific clinical situation.

Conclusion

The identification and management of delirium are essential to good end-of-life care. Nevertheless, this may not be easy. The challenge of providing good treatment for delirium, as with many other symptoms near the end of life, is made more difficult by the paucity of data to guide management decisions. The available data come from other patient populations, particularly elderly patients, and may not apply to patients with a variety of diagnoses who are nearing the end of life.

Therefore, research is needed to identify risk factors for delirium that are specific to patients near the end of life. Data are also needed to identify patients for whom laboratory assessment and imaging are likely to be helpful. Finally, data on the prevention and treatment of delirium in this setting should be gathered. Although this research raises ethical problems related to informed consent, these difficulties are surmountable (33, 49).

Until more data are available, clinicians must nevertheless be able to identify and manage effectively most cases of delirium using the assessment techniques and interventions outlined in this paper. When delirium occurs, its impact on the patient's quality of life should be assessed. If it is distressing to the patient, treatment should be initiated in parallel with an evaluation. By safeguarding or restoring cognitive function in these ways, clinicians can lay the foundation for high-quality end-of-life care.

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I can chart my father's last years by the medical apparatus that attached itself to his existence. The first, the machine that blew a fog of medication into his lungs, sat at his bedside with some innocence. A bland metal-gray in tone and not much larger than a typewriter, the device awaited him several times a day, took in his puffs of exertion and traded out its mysterious mist, sent him away breathing less hard. But next to come were the dark-green oxygen tanks, huge as battleship shells, and their conveyor-like pace to his bedside was the tempo of doom for him.

Ivan Doig
This House of Sky: Landscapes of a Western Mind
New York: Harcourt Brace Jovanovich; 1978:291

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