Update on Delirium: Diagnosis, Management, and Pathophysiology

Barbara Kamholz, MD

Delirium, a condition marked by changes in clinical and mental status, has increasingly been understood as prevalent and dangerous, particularly among elderly or frail patients. It is caused by medical or surgical problems and medication burdens that are beyond the physiological tolerance of the patient. Delirium occurs in 15% to 60% of nursing home patients, 14% to 56% of inpatients, and up to 60% to 87% of patients in the intensive care unit (ICU). Delirium is now understood as an independent risk factor for death and dementia. About 22% to 76% of patients who are hospitalized with delirium die. Patients with delirium have more inpatient complications, they more frequently transition to dependent living settings, and they have significant rates of new dependency in instrumental activities in daily living (IADLs) and activities of daily living (ADLs), especially after episodes of delirium in the ICU. Deliriums that are more severe or persistent have worse outcomes. Delirium costs $38 billion to $152 billion per year. In ICUs alone, episodes of delirium average 39% higher ICU costs and 31% higher hospital costs, largely related to length of stay.

Despite its impressive clinical impact, however, rate of diagnosis lags far behind. Up to 95% of cases are missed. Yet delirium can be prevented or mitigated in healthcare settings that implement interdisciplinary programs across the healthcare spectrum to address it. Clinical programs that provide means to identify the earliest signs of delirium and that have effective methods to rapidly respond to its diverse problems are most likely to succeed in minimizing its devastating clinical impact.

DIAGNOSING DELIRIUM

The diagnosis of delirium is made complex by its variety of behavioral and cognitive presentations and the reality that all of its signs, including behavioral, affective, sensory, cognitive, and motor, can

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fluctuate within seconds. This accounts for much of the clinical confusion associated with delirium, yet it is important to recognize the earliest presentation of any aspects of the syndrome. The best outcomes are achieved with early recognition.10,11

The most widely used bedside rating scale to diagnose delirium for non-ICU patients is the “CAM” (Confusion Assessment Method, see Figure, page 54).12 For ICU patients it is the CAM-ICU worksheet (see Table, page 57).13 The most significant elements of the diagnosis are inattention and broad fluctuations of symptoms. These are not major components of any other psychiatric illness of late life. Unfortunately, they are the most difficult to identify and measure. Orientation is not considered an essential element of the diagnosis of delirium, and, as such, is insufficient.14

KEYS TO DIAGNOSIS

Inattention

Attention is a complex phenomenon that involves distractibility, vigilance, and concentration. It is our most basic means of interacting with the environment, the demands and contingencies of which it must adapt to in a continuous fashion. Inattention occurs when patients are unable to adequately process the complex demands of their internal and external environments. It often presents in very subtle ways and is easily mistaken for fatigue, uncooperativeness, dysphoria, or disinterest. These patients are best described as exhibiting quiet delirium. In contrast, patients who are grossly agitated and irrational are easiest to identify; they are described as having “hyperactive delirium.” Hyperactive delirium is defined as a state marked by agitation, physically aggressive and often violent activity, hyperattentiveness (the inability to suppress responses to the environment or to internal states), and emotional lability. In this condition, deficits in focused attention and fluctuations of presentation are easily identified. Quiet delirium is defined as a state marked by withdrawal, lack of involvement in or communication with the environment, bland or flat affect, and a depressed level of consciousness short of frank stupor. These symptoms reflect an inability to attend in sequential fashion to the demands of the environment. Mixed deliriums are considered to be a combination of the two. The prevalence of these subtypes is variably reported but averages 25% for quiet delirium, 45% for mixed delirium, and 30% for active delirium.

Fluctuations

Fluctuations, unless they are marked, are often missed, and are written off as the differing responses to a variety of health practitioners or variable responses to the hospital environment itself. Such indefinite characteristics go largely unrecognized, unless 24-hour staff notes are carefully examined for subtle evidence of these changes. In a number of studies, nurses have been found to be the first to recognize the early, presenting signs of delirium. Staff notes can greatly assist with diagnosis.15

Phenomenology

All subtypes of delirium can present with motor symptoms (dysarthria, difficulty swallowing, gait disturbances), affective symptoms (dysphoria, labil-
ity, anxiety), sensory difficulties that are associated with incorrect cerebral processing of sensory data (clinically expressed as difficulties with hearing, appreciation of pain, etc.), illusory phenomena, aphasia (partial or global), and impaired cognitive function (dysexecutive function, disorientation, nonsensical speech). Psychotic-like symptoms, such as disorganized speech and visual hallucinations, may also be associated, although the hallucinations in delirium are more illusory than hallucinatory. They commonly involve nonsensical images, such as “clowns on bicycles” or “tires in the ceiling.”

Symptoms of delirium present along a spectrum. Evidence for the existence of subsyndromal delirium has been presented by Levkoff, among others.16 Marcantonio found that patients with subsyndromal delirium had worse outcomes than patients with diagnosable mild delirium.17 Patients may deviate along a spectrum of subclinical delirium through clinically diagnosable delirium and then to a resolving pattern. During resolution, dysphoria and executive dysfunction may be prominent, along with difficulty with short-term memory. Patients, particularly those most vulnerable, may relapse at any time in the presence of new physiological stressors. The recognition of delirium remains one of the most prominent problems in the field. The term “recognition” includes not just diagnosis of the medical condition of delirium, but also the recognition that it is a severe illness that cannot be assumed to resolve on its own with few sequelae. Recent appreciation of this has led to skyrocketing numbers of publications in the field in the past 5 years.18

DIFFERENTIAL DIAGNOSIS

The differentiation of delirium from dementia and prominent psychotic illness, including schizophrenia, schizoaffective disorder, and bipolar disorder, can be problematic.

Dementia

Delirium presents more acutely than dementia, and the patient’s level of attention is not as severely affected in dementia. Clarifying the patient’s baseline level of cognitive function is critical to be able to distinguish delirium from dementia. Most often, delirium is a catastrophic and abrupt event. The evolution of dementia is significantly more gradual in most cases. Where there is doubt, it is safest to address the clinical situation as a delirium so that any precipitating causes are addressed as soon as possible. Patients with delirium superimposed on dementia had more than twice the risk of mortality at 12 months than patients with delirium alone, dementia alone, or patients with neither.19 Assisted-living facility residents classically have high rates of dementia, and the relative risk of discharge of such patients back to institutional settings after an episode of delirium was 9.1.20

Pre-existing Axis I Psychotic Diagnoses

Pre-existing Axis I psychotic diagnoses are a principal cause of the poor identification of delirium, as the patient’s symptoms are often attributed to a prior psychiatric history.21 Hyperactive delirium can easily be mistaken for mania, unless the patient’s underlying physiological risk is taken into account.

Depression

Quietly delirious patients are frequently misidentified as depressed. Farrell22 found that 42% of patients referred to psychiatry consultation services for depression were actually delirious. However, an additional finding was that 60% of patients with delirium have symptoms of dysphoria, and 52% have passive or active thoughts of suicide. Patients with a significant delirium risk burden in hospital who present with acute suicidal ideation should be considered delirious until proven otherwise. Suicide very rarely develops as an acute disorder.

THE ETIOLOGY OF DELIRIUM

A multitude of risk factors have been identified in treatment studies of delirium, often with little overlap. This has suggested a more laborious process than is necessary to identify causes and assist with management. A more streamlined and clinically adaptable system involves a construct initially proposed by Inouye in her landmark study published in 1996.23 This model, which involves the identification of a population at risk for delirium in whom she identified a limited number of precipitating factors, provides the best overall approach to the recognition of delirium. It more functionally enables the early identification of patients vulnerable to delirium or who are in the earliest stages of delirium and avoids the confusion associated with long lists of possible causes. Using these tools can assist clinicians to manage delirium as a relatively predictable complication in vulnerable patients.

Using this general approach, many workers in the field would agree that age, cognitive impairment, and medical burden, as well as the presence of a large number of medications and a prior history of alcohol abuse, define the population at highest risk. This so-called “baseline risk” also represents a continuum, such that a patient who is elderly, significantly cognitively impaired, and has a number of systemic illnesses is much more likely to develop delirium than a younger person with few medical problems and cognitive impairment.

It is easiest to conceptualize the etiology of delirium from the perspective of the syndrome of frailty. The term has many definitions, but it most commonly is used to identify deteriorated and significantly vulnerable patients as those who are aged, medically quite burdened, and cognitively impaired. These factors predispose to the downward spiral of health that leads to significant physiological instability. Specifically, the term “frailty” is meant to indicate a population that is not just disabled but in a disequilibrated state. Some investigators...
provide a broader view of the components of frailty by including socioeconomic stressors, psychiatric disorders, and limitations of the patient’s network of family and local support, as well as medical vulnerabilities. This references the network of resources that frail geriatric patients require to avoid catastrophic illness.24

The increased vulnerability of frail patients to the catastrophic decline associated with delirium is most often linked to their limited physiological reserve. With aging, there is diminution of redundant systems, such as neurons and neuronal circuits in the brain, liver, metabolic, and renal excretory capacity. Patients with the least reserve are more susceptible to any stressor, however small, than healthier individuals, in a striking resemblance to the behavior of chemical equations far from equilibrium. The implication that it is the patient’s underlying vulnerability and disequilibrated status, as opposed to any specific cause, is the most important predictor of delirium in an individual person. It is then easy to conclude that the precipitating causes of delirium are the most common complications, including iatrogenic ones, found in hospitalized patients. A short list includes hyponatremia, acute blood loss, pain, acute renal failure, fractures, acute infections (urinary tract or pulmonary), the use of longer term in-dwelling bladder catheters, dehydration, decubiti, malnutrition, and the use of psychotropic medications.23,25 Clearly, this significantly streamlines the approach to recognition and management.

**PATHOPHYSIOLOGY OF DELIRIUM**

Historically, multiple interacting theories have been proposed to explain the pathophysiology of delirium. Any given delirium may have an unknown number of associated clinical conditions, both at baseline and in the acute phase of development or continuation of delirium, and they do not necessarily occur in the same combinations. It is only when the patient has recovered that there is any clinical certainty that the basic physiological process(es) have been fully addressed. However, as noted in the introduction, many deliriums do not fully resolve. It is also not necessarily the case that two patients who appear to have equal degrees of baseline and precipitating stressors will both become delirious. We are left with broad-based geriatric clinical intervention methods that are helpful but quite nonspecific and that have not helped to clarify the molecular, cellular, and interactive processes in the brain that underlie the catastrophic changes that are reflected in the symptoms of delirium. Although there are many existing etiological theories of delirium, the principal ones, which involve increased neurotransmitter availability as well as the effect of inflammatory factors, will be discussed here.

In its most basic sense, the brain depends on the body to provide the glucose and oxygen it requires to function normally. Deficits in these substances have a dramatic impact on the production of adenosine triphosphate (ATP), itself the main “engine” of all cellular function, as well as the synthesis and metabolism of neurotransmission. Impairments in neuronal membrane function appear to underlie much of the clinical pathology observed in delirium.26 Specifically, the disruption of calcium channel networks in cell membranes, found in the presence of excess dopamine and magnified by hypoxia, leads to mitochondrial dysfunction. Significant evidence indicates that excess dopamine and/or insufficient acetylcholine in the brain can best explain the cascade of metabolic and behavioral manifestations of delirium. Each of these neurotransmitters has complex and interactive impacts on cellular function within the CNS.27 In the presence of impaired mitochondrial function, hypoglycemia, and hypoxia result in increases in cellular influx of Ca++. The influx of calcium into cells appears to trigger a series of reactions that lead to mitochondrial injury and activation of catabolic enzymes that lead to cellular dysfunction and potentially death. Hypoxic conditions in brain stimulate the activity of tyrosine hydroxylase. This results in increased production of dopamine, with resulting disruption of ATP production. Decreases in the production of ATP and the increase in toxic metabolites of dopamine then inhibit catechol-o-methyl transferase, the main extracellular de-activator of dopamine. These processes collectively result in increased dopamine levels in brain, up to a factor of 500 times during episodes of striatal ischemia.26 During states of cerebral ischemia, further cellular risk is posed by efflux of dopamine. Dopaminergic neurons themselves are known to be highly susceptible to oxidative stress, which may result in massive releases of dopamine into the extracellular space.27 Agents that increase available dopamine in the brain, such as stimulants, have also been associated with delirium. D2 blockers, such as haloperidol, are widely known to improve behavior in patients with delirium, and possibly the syndrome itself.

Cellular mechanisms associated with deficits of acetylcholine in the brain as they contribute to delirium have been less well worked out, but acetylcholine itself is a vital neurotransmitter in areas of the brain that subserve attention and memory.

Inflammatory processes, such as urinary tract infection and pneumonia, have been highly associated with delirium. With aging, there is an elevated inflammatory profile.28 Activated glial cells transmit peripheral inflammatory cytokine responses to the CNS. This is associated with decreased synaptic plasticity, a necessity for the modula-
tion of behavior, learning, and cognition. Inflammatory states are associated with impaired mitochondrial function. Release of the inflammatory cytokines TNF alpha, and interleukin 1 generate adhesion molecules that disrupt cerebral vasculature, further impacting the integrity of the blood brain barrier.28

Neuroimaging has recently been incorporated to evaluate structural and functional processes in brain during delirium, although the findings are diverse enough that consistent trends have so far been elusive. Yokota and colleagues, using xenon enhanced computerized tomography (CT), found that patients with delirium experienced a 42% reduction in overall cerebral blood flow compared with baseline. After recovery from delirium, the findings normalized.29 Such findings may explain the multisystem deficits observed, which include cognitive, affective, sensory, and motor function. There is not enough evidence at this time to determine whether structural correlates precede or follow delirium.

FOCUSED INTERVENTION TRIALS

A number of intervention programs have been employed to identify the optimal methods to manage delirium. These programs have been studied in hospitalized patients; there are no published trials among outpatients. Most of these trials have not found that they are helpful to the primary outcome of a decrease in incidence.30,31 But they may demonstrate decreases in severity and duration of symptoms of delirium, which have been significantly associated with worsened outcomes. Unfortunately, the impact of these interventions on longer term outcomes has been mixed and overall disappointing.32 In light of this, the current focus within the field is on prevention of delirium. In a prominent study published in 1999,10 a complex intervention that involved a significant amount of volunteer work, as well as a very high level of nursing care, was shown to significantly reduce length of stay, episodes, and incidence of delirium.

A second highly organized and comprehensive program demonstrated a decreased incidence of delirium in post-hip fracture patients.33 These interventions highlight an effective level of geriatric care that could be attained in any institution.

TREATMENT APPROACHES TO DELIRIUM

Overall Approach

Priorities of treatment include addressing acute medical contributors to the syndrome, providing a structured and predictable environment, and stabilizing patient behavior sufficient to enable the treatment of underlying medical causes. The primary task of clinicians is to identify and address modifiable conditions. In this way, the impact of relatively unmodifiable conditions, such as stroke, myocardial infarction, and end-stage renal function, can be minimized. Our effectiveness at treating delirium depends on the extent to which we can provide skilled and sophisticated nursing care that allows time for the multiple interventions required by elderly patients. Avoidance of iatrogenesis that can prolong the delirium is an essential part of this effort. Factors contributing to increased risks of delirium in hospital include the increased pace of healthcare settings, rapid and increasingly unstable discharges, and remarkably limited amounts of time spent with patients. Housestaff may have as little as 3.5 minutes per day per patient.1 Skilled nursing care alone could provide many of the most effective strategies for minimizing delirium, including mobilization, improvement of cognitive stimulation, and minimization of the need for restraints. Up to 50% of cases of delirium can be prevented.1

Treating the Causes

The basic principle of treatment of delirium is to aggressively seek and treat the reversible causes of delirium and to mitigate other nonreversible causes. Emphasis should be placed on illnesses that result in poor cerebral oxygen availability or have significant metabolic impairments. Elimination of infectious or inflammatory sources, addressing orthopedic injuries, and removal of offending medications are also essential. Correcting the most common metabolic and infectious factors, such as serum BUN, creatinine, sodium, and calcium and treatment of urinary tract infections and pneumonia are remarkably effective in the most vulnerable patients. Contrary to prior practices, lumbar puncture and head imaging are not helpful in the absence of focal findings.34

Removing Offending Medications

Evaluating the clinical need for opiates, anticholinergics, benzodiazepines, and dopaminergics is critical to identify whether any CNS active medications can be discontinued. Medications that alter the dopamine/acetylcholine balance within the CNS are a primary focus. Optimally, benzodiazepine use will be completely avoided. A significant number of deliriogenic medications are found in commonly used medications, including digoxin, penicillin, amantadine, quinolone antibiotics, furosemide, nifedipine, and warfarin. Further treatment of this topic is beyond the scope of this article.2

Pharmacologic Approaches

It is not yet clear that pharmacological approaches can impact the clinical course of delirium, although in one study the use of haloperidol at 1.5 mg per day for 2 to 3 days before hip fracture repair and 3 days post repair, was able to minimize severity and duration.34 However, behavioral stabilization is quite achievable, and a number of recent studies support this conclusion. The use of benzodiazepines, although standard for obligate alcohol detoxification regimens and for sedation in ICUs, have, nonetheless, been found in most studies to carry a high risk of inducing or significantly worsening delirium. There are few double-blinded, placebo-controlled,
Confusion Assessment Method in the ICU

**RASS is above -4 (-3 through +4)**

- Proceed to next Step

**If RASS is -4 or -5**

- Stop
  - Reassess patient at later time

**Delirium Assessment (CAM-ICU):**

1. **Acute Onset or Fluctuating Course**
   - An acute change from mental status baseline? Or Patient's mental status fluctuating during the past 24hrs
   - NO
     - Stop
     - No delirium
   - YES
     - ≥3 Errors
       - ≥3 Errors
         - Stop
         - No delirium
     - <3 Errors
       - < 3 Errors
         - ≥3 Errors
           - Patient is Delirious
         - < 2 Errors
           - Stop
           - No delirium
     - ≥2 Errors
       - ≥2 Errors
         - Patient is Delirious
     - < 2 Errors
       - < 2 Errors
         - Stop
         - No delirium

2. **Inattention**
   - Please read the following ten letters: SAVE A H A R T
   - Scoring: Error: when patient fails to squeeze on the letter “A”
   - Error: when the patient squeezes on any letter other than “A.”
   - ≥3 Errors
     - ≥3 Errors
       - Stop
       - No delirium
     - < 3 Errors
       - < 3 Errors
         - ≥3 Errors
           - Stop
           - No delirium
       - ≥2 Errors
         - Patient is Delirious
     - < 2 Errors
       - < 2 Errors
         - Stop
         - No delirium

3. **Altered Level of Consciousness**
   - (“actual” RASS)
   - If RASS is zero, Proceed to next step
   - 0 RASS
     - 0 RASS
       - Stop
       - No delirium
   - ≥3 Errors
     - ≥3 Errors
       - Stop
       - No delirium
   - < 3 Errors
     - < 3 Errors
       - ≥3 Errors
         - Stop
         - No delirium
     - ≥2 Errors
       - Patient is Delirious
     - < 2 Errors
       - < 2 Errors
         - Stop
         - No delirium

4. **Disorganized Thinking**
   - 1. Will a stone float on water?
      - (Or: Will a leaf float on water?)
   - 2. Are there fish in the sea?
      - (Or: Are there elephants in the sea?)
   - 3. Does one pound weigh more than two pounds?
      - (Or: Do two pounds weigh more than one?)
   - 4. Can you use a hammer to pound a nail?
      - (Or: Can you use a hammer to cut wood?)
   - 5. **Command:**
     - Say to patient: “Hold up this many fingers”
     - (Examiner holds two fingers in front of patient)
     - “Now do the same thing with the other hand”
     - (Not repeating the number of fingers).
     - If patient is unable to move both arms for the second part, ask patient “add one more finger”

randomized trials of neuroleptics for behavioral management in delirium. Of all these agents, haloperidol remains the most studied. Haloperidol can be effective in low dosages, starting with 0.25 mg orally or injections bid, and increasing to the 2 to 3 mg per day range only in severe cases. Risperidone in doses ranging from 0.5 to 4.0 mg daily and olanzapine in doses of 2.5 to 11.6 mg daily have been efficacious for the behavioral treatment of delirium with fewer extrapyramidal effects than haloperidol. In addition, case reports or case series regarding quetiapine, aripiprazole, and ziprasidone have been published, with no randomized, double-blinded, placebo-controlled studies yet reported. There are few such medication trials in the field.

The use of cholinesterase inhibitors has been trialed in delirium. In a randomized, placebo-controlled, double-blinded study by Liptzin et al, the use was not found to have a significant impact on the incidence of delirium among a non-demented, young-elderly population undergoing elective hip fracture. In a small retrospective study, a population of dementia patients chronically maintained on rivastigmine had significantly less incidence of delirium than a comparable, non-chronically medicated group.

Environmental Management

Patients with delirium are unable to manage a complex environment, make appropriate decisions, care for their basic needs, or problem solve. The environment must adapt to them. Most studies agree that limiting rotation of personnel caring for the patient, minimizing room changes, providing rest and quiet at night time, actively feeding the patient, providing a calming “interpreter,” such as a “sitter” or family member to reorient and guide the patient, limiting distractions (loud conversation, beepers, overhead speakers, televisions, crowded areas), providing hearing aids and glasses, and providing clear-cut and step-by-step instructions are critical. Mobilizing the patient will minimize recovery time and may assist in rehabilitation of muscle after critical illness. The patient must be kept safe by limiting dangerous objects in the patient’s reach, avoiding falls, and using assistive devices to limit the possibility of their pulling at lines or touching wounds. A brief period of restraint use, especially if it helps to minimize the needs for excessive CNS active medication, may be justified, even though restraints overall contribute to delirium. ICUs with windows have half the rate of delirium as those without. Complex protocols exist for the design of treating environments that are beyond the scope of this article.

CASE EXAMPLES

Case 1: What is the Cause of Delirium?

A 59-year-old man functional man with a lifetime history of bipolar disorder and no other medical comorbidities was initially treated 3 months PTA with lithium, valproate, and risperidone in slowly escalating doses. He had a 1-month history of steadily declining mental status and had become completely dependent in ADLs. He appeared cognitively very slowed on admission, struggling with attention questions and nearly inert. Li+ level is 2.15. What do you do now?

First, evaluate risk at baseline. On a scale of low, medium, and high, this patient is relatively young, has no medical comorbidities, and is functional at baseline. Thus, he must be considered to be at low risk for delirium.

Second, evaluate and address all other potential causes for delirium, starting with basic metabolic and infectious etiologies, and medications. Investigate the patient’s medical history to see if any other conditions may have contributed to the delirium. Given the severe deterioration observed in the patient, you should stop the patient’s lithium, and minimize valproic acid and risperidone, especially given the severe deterioration observed in the patient.

Despite this care, the patient did not, in fact, improve quickly. His workup did not discover any reason other than the toxic level of lithium for his delirium. What would be your next step?

First, analyze the situation in light of the patient’s low baseline risk for delirium. Given that his renal function is intact, his lithium level should have decreased rapidly and his cognitive status should therefore have improved very quickly.

Second, recognize that all the most common risk factors (metabolic, infectious, medication-related), as well as any inherent ones in the patient’s history, have been addressed. At this point, conclude that a more intensive examination must be performed because this patient’s low baseline risk for delirium does not predict his prolonged cognitive impairment.

Third, the more intensive workup should include an examination of the CNS. The first element of this was performed, a head CT. The patient’s brain demonstrated global atrophy, which was then identified as the primary reason for his prolonged delirium. Although he had originally appeared cognitively intact, this was based on a limited lifestyle in his own home. The use of the risk-based delirium assessment model quickly enabled the identification of the primary cause of his delirium.

Case 2: Is the Patient Delirious or Depressed?

A 79-year-old man with dementia, acute renal failure, diabetes, coronary artery disease, and chronic obstructive pulmonary disease but no psychiatric history was admitted for pneumonia. After a 3-week course complicated by a urinary tract infection, delirium, and hyponatremia, he became less agitated, more oriented, and more cooperative, in association with decreased white blood cell count and diminished oxygen requirements. You are consulted for acute suicidal ideation. What should you do?

First, identify baseline risk. As this patient is quite elderly, has a large burden of
<table>
<thead>
<tr>
<th>Feature 1: Acute Onset or Fluctuating Course</th>
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<tbody>
<tr>
<td>(positive if you answer “yes” to either 1A or 1B)</td>
</tr>
<tr>
<td>Positive</td>
</tr>
<tr>
<td>1A: Is the pt different than his/her baseline mental status? Or 1B: Has the patient had any fluctuation in mental status in the past 24 hours as evidenced by fluctuation on a sedation scale (e.g., RASS), GCS, or previous delirium assessment?</td>
</tr>
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**Feature 2: Inattention**

Positive if either score for 2A or 2B is less than 8. Attempt the ASE letters first. If pt is able to perform this test and the score is clear, record this score and move to Feature 3. If pt is unable to perform this test or the score is unclear, then perform the ASE Pictures. If you perform both tests, use the ASE Pictures’ results to score the Feature.

| Positive | Negative |
| 2A: ASE Letters: record score (enter NT for not tested). | Score (out of 10): ______ |
| Directions: Say to the patient, “I am going to read you a series of 10 letters. Whenever you hear the letter ‘A,’ indicate by squeezing my hand.” Read letters from the following letter list in a normal tone. S A V E A H A A R T. Scoring: Errors are counted when patient fails to squeeze on the letter “A” and when the patient squeezes on any letter other than “A.” |
| 2B: ASE Pictures: record score (enter NT for not tested). Directions are included on the picture packets. | Score (out of 10): ______ |

**Feature 3: Disorganized Thinking (Positive if the combined score is less than 4.)**

| Positive | Negative |
| 3A: Yes/No Questions (Use either Set A or Set B, alternate on consecutive days if necessary): |
| Combined Score (3A+3B) _____ (out of 5) |
| Score ___ (Patient earns 1 point if able to successfully complete the entire command) |

**Feature 4: Altered Level of Consciousness**

(positive if the Actual RASS score is anything other than “0” (zero))

| Positive | Negative |
| Overall CAM-ICU (Features 1 and 2 and either Feature 3 or 4) |
medical problems, and has a history of cognitive impairment, he must be placed, on a scale from low to medium to high risk, in a high risk category. Thus, we have a very low threshold for a diagnosis of delirium, which implies that common clinical problems are most likely to be responsible for his delirium. Delirium must be ruled out first here; it causes more morbidity than depression in this patient.

Second, evaluate the clinical symptoms. Suicidal ideation is common in delirium, but the key word is “acute.” Suicidality rarely occurs acutely in the context of depression.

Given the acute risks of delirium compared with the longer term risks of depression, it is wisest to consider this a case of recurrent delirium. Adding an antidepressant may worsen the picture — better to wait 2 to 3 days to rule out delirium because that delay would not greatly impact treatment to 3 days to rule out delirium because that delay would not greatly impact treatment of depression. Mislabelling the patient as depressed may result in failing to search for the cause of the delirium. In this patient, the cause would most likely be a recurrent pneumonia or urinary tract infection.

CONCLUSION

Delirium is a critical illness that occurs primarily in elderly and frail individuals. Modulation of this very significant problem, however, is increasingly within our reach.

REFERENCES