Chapter 1

Altered Mental Status: Delirium and Coma

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Key words: coma, delirium, spontaneous awakening trial, spontaneous breathing trial, Confusion Assessment Method for the ICU, Intensive Care Delirium Screening Checklist, Richmond Agitation-Sedation Scale

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Whether from acute disease processes or iatrogenic therapies, many critically ill patients have altered mental status, ranging from coma to hyperactive delirium. A comatose patient is unresponsive to physical or verbal stimuli. Delirium is an acute and fluctuating disorder of consciousness characterized by inattention, disorganized thinking, and perceptual disturbances with hypoactive and hyperactive subtypes (Figure 1 on page 2). Altered mental status has traditionally been considered an expected consequence of critical illness. Delirium has been underdiagnosed in the ICU, and increasing research and investigations have shown that delirium is a form of acute brain dysfunction that is associated with worse clinical outcomes. The true magnitude of the effects of coma and delirium has just recently begun to be understood, partly because of the many different terms (e.g., confusional state, ICU psychosis, acute brain dysfunction, and encephalopathy) that have been used to describe altered mental status. With increased acceptance of the Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition) definition of delirium, validation of screening tools for delirium in ICU patients, and recognition of the burden to patients and society associated with this acute brain dysfunction, increasing emphasis is being placed on investigating the causes and consequences of brain organ dysfunction in the critically ill.
Figure 1.
Delineation between delirium and coma, highlighting the cardinal symptoms of delirium. *Optional symptoms of delirium (may be present but are not required for the diagnosis of delirium).*

Brain Organ Dysfunction

- Coma
  - Unaroused to voice
  - Arousable to voice

- Acute mental status change
- Fluctuating mental status
- Inattention
- Disorganized thinking
- Altered consciousness
- Hallucinations, delusions

DIAGNOSIS OF ACUTE BRAIN DYSFUNCTION

Traditionally, many scales have been used to assess the level of sedation and agitation in ICU patients, including the Ramsay scale, Riker Sedation-Agitation Scale, motor activity assessment scale, and Richmond Agitation-Sedation Scale (RASS). Their reliability and validity in the ICU patient population have allowed for their use in goal-directed medical therapy. However, only the RASS (Figure 2 on page 3) has been shown to detect variations in the level of consciousness over time or in response to changes in sedative and analgesic drug use. The Glasgow Coma Scale is the most widely used tool to characterize coma in the trauma and critical care patient populations. This scoring system evaluates eye opening, verbal response, and motor response on a 15-point scale (Table 1 on page 3).

OUTCOMES ASSOCIATED WITH BRAIN DYSFUNCTION

Acute brain dysfunction in critically ill patients has been demonstrated to be independently associated with worse clinical outcomes. Patients experiencing delirium have been shown to take longer time to wean from mechanical ventilation. They have increased ICU and hospital length of stay and are more likely to be readmitted to the hospital after discharge. Consequently, the presence of delirium is associated with significantly higher ICU and hospital costs. Furthermore, patients with delirium have higher mortality, and each additional day of delirium increases the risk of dying. Although delirium and coma represent acute brain dysfunction, many critically ill patients also have subsequent long-term cognitive impairment (chronic brain dysfunction), which may persist for months to years after their hospitalization, significantly degrading their quality of life. Among patients who survive their critical illness, up to 75% experience long-term cognitive impairment, and longer periods of acute brain dysfunction in the hospital are associated with greater degrees of cognitive decline 1 year after hospital discharge. With increasing recognition of this tremendous burden, society and the medical profession have now placed increasing attention and emphasis on the prevention and treatment of acute brain dysfunction.
The Richmond Agitation-Sedation Scale is a 10-point scale with discrete criteria to distinguish levels of agitation and sedation. Reproduced with permission from Dr. E. Wesley Ely (www.icudelirium.org).

### Richmond Agitation-Sedation Scale

<table>
<thead>
<tr>
<th>Score</th>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>+4</td>
<td>Combative</td>
<td>Overtly combative, violent, immediate danger to staff</td>
</tr>
<tr>
<td>+3</td>
<td>Very Agitated</td>
<td>Pulls or removes tubes or catheters; aggressive</td>
</tr>
<tr>
<td>+2</td>
<td>Agitated</td>
<td>Frequent nonpurposeful movement, fights ventilator</td>
</tr>
<tr>
<td>+1</td>
<td>Restless</td>
<td>Anxious but movements not aggressive or vigorous</td>
</tr>
<tr>
<td>0</td>
<td>Alert and calm</td>
<td></td>
</tr>
<tr>
<td>-1</td>
<td>Drowsy</td>
<td>Not fully alert but has sustained awakening (eye-opening/eye contact to voice (≥10 seconds)</td>
</tr>
<tr>
<td>-2</td>
<td>Light sedation</td>
<td>Briefly awakens with eye contact to voice (&lt;10 seconds)</td>
</tr>
<tr>
<td>-3</td>
<td>Moderate sedation</td>
<td>Movement or eye-opening to voice (but no eye contact)</td>
</tr>
<tr>
<td>-4</td>
<td>Deep sedation</td>
<td>No response to voice but movement or eye opening to physical stimulation</td>
</tr>
<tr>
<td>-5</td>
<td>Unarousable</td>
<td>No response to voice or physical stimulation</td>
</tr>
</tbody>
</table>

Acute change or fluctuation in mental status (feature 1), inattention (feature 2), disorganized thinking (feature 3), and an altered level of consciousness (feature 4). The diagnosis of delirium using the combination of the RASS scale and the CAM-ICU requires the following 4 items:

1. RASS score of −3 or higher
2. Feature 1 of CAM-ICU (acute change or fluctuation in mental status)
3. Feature 2 of CAM-ICU (inattention)
4. One of the following: Feature 3 (disorganized thinking) or Feature 4 (altered level of consciousness of CAM-ICU)

The ICDSC uses 8 diagnostic features to evaluate brain function. A diagnosis of delirium requires 4 or more features from the checklist to be present during the evaluation period. Additionally, patients manifesting some features from the ICDSC, but who do not meet all the requisite criteria for delirium diagnosis, are considered

### Table 1.

#### Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Eye opening</th>
<th>Spontaneously</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>To speech</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Verbal response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Inappropriate</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Incomprehensible</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Motor response</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>Localizes to pain</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Withdraws from pain</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Flexion to pain</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Extension to pain</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

**Maximal score 15**
Figure 3.
Confusion Assessment Method for the Intensive Care Unit (CAM-ICU). Patients are considered to have delirium if they have Richmond Agitation-Sedation Scale scores of -3 and above (see previous figure) and are CAM-ICU positive by having features 1 and 2 present and either feature 3 or feature 4 positive. Reproduced with permission from Dr. E. Wesley Ely (www.icudelirium.org).

**Feature 1**
Acute onset of mental status changes or fluctuating course

**Feature 2**
Inattention

**Feature 3**
Disorganized thinking

**Feature 4**
Altered level of consciousness

AND

OR

to have subsyndromal delirium. This segment of the spectrum of acute brain dysfunction lies between normal and full-blown delirium and is associated with worse outcomes than normal cognition but better outcomes than delirium. A complete description of delirium monitoring tools and training materials (including clinical vignettes and translations of the CAM-ICU) can be found at www.icudelirium.org.

**INCIDENCE AND RISK FACTORS FOR BRAIN DYSFUNCTION**

The incidence of acute brain dysfunction is common in the ICU, with between 50% and 80% of patients with critical illness developing delirium depending on the severity of illness and the need for mechanical ventilation. Despite increasing research in the field, the multifactorial pathophysiological processes of delirium and coma remain poorly understood. Numerous hypotheses exist and include neurotransmitter imbalance (e.g., dopamine, \( \gamma \)-aminobutyric acid, and acetylcholine), inflammatory perturbations (e.g., tumor necrosis factor-\( \alpha \), interleukin-1, and other cytokines and chemokines), impaired oxidative metabolism, cholinergic deficiency, and changes in various amino acid precursors. Contributing sources can be summarized as chronic patient-related factors (e.g., age, previous dementia, diabetes, heart failure) and acute illness-related risk factors (e.g., psychoactive medications, hypoxemia, shock, hypothermia) (Table 3 on page 7). Importantly, sedative regimens and medications are risk factors readily modifiable by clinicians that have been
<table>
<thead>
<tr>
<th>Patient Evaluation</th>
<th>Characteristics</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Altered level of consciousness</strong></td>
<td>A: No response (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>B: Response to intense and repeated stimulation (loud voice and pain) (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C: Response to mild or moderate stimulation (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>D: Normal wakefulness (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>E: Exaggerated response to normal stimulation (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Inattention</strong></td>
<td>Difficulty in following a conversation or instructions</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Easily distracted by external stimuli</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Difficulty in shifting focuses</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Disorientation</strong></td>
<td>Any obvious mistake in time, place, or person</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Hallucinations, delusion, psychosis</strong></td>
<td>Unequivocal hallucination or behavior likely due to hallucination or delusion</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Gross impairment in reality testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Psychomotor agitation or retardation</strong></td>
<td>Hyperactivity requiring additional sedative drugs or restraints</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Hypoactivity or clinically noticeable psychomotor slowing</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Inappropriate speech or mood</strong></td>
<td>Inappropriate, disorganized or incoherent speech</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Inappropriate display of emotion related to events or situation</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sleep–wake cycle disturbance</strong></td>
<td>Sleeping &lt;4 h or waking frequently at night (not initiated by medical staff or loud environment)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Sleeping during most of the day</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Symptom fluctuation</strong></td>
<td>Fluctuation of the manifestation of any item or symptom over 24 h</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total score (0-8)</strong></td>
<td>A score ≥4 indicates delirium</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Implicated in the development of delirium in the critical care patient population.

SEDATIVE AND ANALGESIC MEDICATIONS AND BRAIN DYSFUNCTION

Psychoactive medications, depth of sedation, and certain sedation paradigms have been associated with worse cognitive outcomes.
Psychoactive Medications

The temporal association of psychoactive medications with delirium in critically ill patients has been examined in different ICU cohorts. In a cohort of mechanically ventilated medical ICU patients, lorazepam administration was found to be an independent risk factor for the daily development of delirium after adjustment for important covariates such as age, severity of illness, and presence of sepsis. Propofol, morphine, and fentanyl were associated with higher but not statistically significant odds ratios for delirium development in the same cohort. In surgical, trauma, and burn ICU patients, midazolam has been associated with increased delirium incidence.

The effects of analgesic medications, and specifically opioids, on acute brain dysfunction are not as consistently demonstrated as the effects of benzodiazepines. In fact, insufficient pain relief has been shown to be a risk factor for delirium and can contribute to sleep disturbances, disorientation, anxiety, and long-term effects such as posttraumatic stress disorder (PTSD). In a prospective cohort study that enrolled patients with hip fractures, none of whom had preoperative delirium, patients who received less than 10 mg of parenteral morphine equivalents per day were more likely to develop delirium than patients who received more analgesics. Additional studies have reported beneficial effects of morphine and methadone on the incidence of delirium. However, providing adequate analgesia needs to be balanced against the risk of predisposing patients to delirium due to excessive opiate administration, for meperidine and morphine have been associated with increased risk for delirium. Thus, opiates may be protective of acute brain dysfunction in patients at high risk for pain but may be detrimental if used excessively to achieve sedation.

Depth of Sedation

To optimize patient care, safety, and comfort while minimizing the negative outcomes associated with psychoactive medications, bedside caregivers must achieve the right balance of sedative and analgesic drug administration. Undersedation and inadequately treated pain can lead to a hyperactive stress response that includes tachycardia, increased oxygen consumption, hypereagulability, immunosuppression, hypermetabolism, and increased endogenous catecholamine activity. Agitation can lead to the removal of medical devices (e.g., endotracheal tubes, intravascular lines), creating life-threatening situations. Unrelieved agitation and anxiety may become a significant source of physical and psychological stress for patients both during an acute event and subsequently, when PTSD may ensue. Consequently, oversedation of patients occurs commonly in the ICU. Excessively deep levels of sedation can result in worse clinical outcomes, including longer duration of mechanical ventilation and ICU length of stay, increased use of radiological evaluations of mental status, and higher probability of developing delirium. Furthermore, the presence of burst suppression on electroencephalograms, associated with deep sedation, has been shown to be an independent predictor of mortality in critically ill patients.

As a result of increasing evidence of the harm of deep sedation, multiple strategies have been evaluated to decrease patients’ psychoactive drug exposure. Using target-based sedation, protocolizing sedative drug delivery, and instituting daily interruptions of sedation have all been shown to decrease sedative administration and improve patient outcomes. By combining daily spontaneous awakening and breathing trials, the Awakening and Breathing Controlled Trial showed a 50% reduction in sedative use, a reduction in coma and ventilator days during the ICU stay, and, most notably, a reduction in mortality at 12 months. Although these studies support the contention that deep sedation is not required in the majority of ICU patients, it is also important to note that interrupted sedation methods have not been associated with an increase in long-term neuropsychological outcomes, including PTSD. In fact, sedating agents have been associated with increased PTSD symptoms, and an increased number of days of sedation in the ICU is associated with a greater likelihood of depression and PTSD. Although unpleasant memories may contribute to PTSD symptoms in survivors of critical illness, delusional memories of the ICU stay are more likely to
result in PTSD than factual, even if painful, memories.\textsuperscript{5,56} Further supporting the concept that sedation and amnesia may have prolonged neuropsychological and cognitive effects, patients with recall of their ICU stay have been shown to manifest less cognitive dysfunction than patients who have complete amnesia for their ICU experience.\textsuperscript{57}

**Sedation Paradigms**

The choice of sedation regimen has implications in acute brain dysfunction beyond the effects of target-based and goal-directed sedation with daily interruption of sedatives. In the majority of studies comparing propofol to benzodiazepines, patients randomized to the propofol group have experienced better outcomes.\textsuperscript{58,59} Similarly, sedation techniques based on analgesia with the use of morphine or remifentanil have shown shorter times on mechanical ventilation.\textsuperscript{4-60} With regard to acute brain dysfunction specifically, the MENDS study (a randomized controlled trial of dexmedetomidine vs lorazepam) provided evidence that sedation with dexmedetomidine can decrease the duration of brain organ dysfunction, with a lower likelihood of delirium development on subsequent days.\textsuperscript{61,62} Comparing dexmedetomidine with midazolam, the SEDCOM study demonstrated a reduction in the incidence of delirium with dexmedetomidine and a shorter time on mechanical ventilation.\textsuperscript{63} In another randomized controlled trial, the DEXCOM study showed that dexmedetomidine reduced the duration but not the incidence of delirium after cardiac surgery compared with morphine-based therapy.\textsuperscript{64} These studies support the contention that reducing benzodiazepine exposure by using alternative sedation paradigms can improve ICU patient outcomes, including acute brain dysfunction.

**ACUTE BRAIN DYSFUNCTION PREVENTION AND MANAGEMENT**

To prevent delirium from occurring and to manage its consequences, the clinician must recognize and proactively prevent or treat reversible causes of delirium. The extensive list of contributing factors in the ICU includes, but is not limited to, pain, anxiety, sleep disturbances, hypoxia, hypercarbia, hypoglycemia, metabolic derangements, shock, and medication effects (Table 3 on page 7). Beyond that, just as the causes of delirium are multifactorial, the approach to prevention and management must be multifaceted.

**Delirium Prevention**

A landmark study of non-ICU medical patients demonstrated a 40% reduction in the incidence of delirium by focusing on several key interventions, including regular provision of stimulating activities, a nonpharmacological sleep protocol, early mobilization activities, appropriate and early removal of catheters and restraints, optimization of sensory input, and attention to hydration.\textsuperscript{65} Some similar studies have shown a decrease in the duration and severity of delirium without decreasing overall incidence;\textsuperscript{66,67} whereas others have shown benefit only in specific subgroups\textsuperscript{68} or have not shown any patient benefit.\textsuperscript{69}

### Table 3.

**Risk Factors for Delirium**

<table>
<thead>
<tr>
<th>Host Factors</th>
<th>Acute Illness</th>
<th>Iatrogenic and Environmental Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Sepsis</td>
<td>Anticholinergic medications</td>
</tr>
<tr>
<td>Baseline comorbidity</td>
<td>Hypoxemia</td>
<td>Sedative medications</td>
</tr>
<tr>
<td>Baseline cognitive impairment</td>
<td>Global severity of illness</td>
<td>Analgesic medications</td>
</tr>
<tr>
<td>Genetic predisposition (?)</td>
<td>Metabolic disturbances</td>
<td>Sleep disturbances</td>
</tr>
</tbody>
</table>
efficacy of these sorts of strategies in ICU patients has not been studied and hence is unknown.

Specific to the ICU population, however, early initiation of physical therapy has been associated with improved outcomes, including decreased length of stay both in the ICU and hospital. Recently, a randomized controlled study evaluated the combination of daily interruption of sedation with physical and occupational therapy on cognitive and functional outcomes. The investigators demonstrated that patients who underwent early mobilization had an approximately 50% decrease in the duration of delirium in the ICU and hospital and had significant improvement in functional status at hospital discharge. A liberation and animation strategy focusing on the ABCDEs (Awakening and Breathing trials, Choice of sedation, Delirium monitoring and management, and early Exercise) during critical illness has been shown to improve patient outcomes and likely reduce the incidence and duration of acute and long-term brain dysfunction in critically ill patients.

Delirium Management

Only after minimizing contributing factors and optimizing treatment of the underlying physiological abnormalities should pharmacological therapy to manage delirium be undertaken. Although numerous studies have examined the effects of antipsychotic medications on delirium, we are still lacking large randomized controlled trials in the ICU patient population comparing the efficacy of typical and atypical antipsychotics to placebo. As a result, small studies and case reports constitute the best data available from which to formulate management recommendations for the antipsychotic medications most suitable for the treatment of delirium. To assist in developing a delirium management algorithm, an empirical protocol (Figure 4 on page 9) is proposed here that is largely based on the current clinical practice guidelines. Although the nonpharmacological interventions recommended in this protocol have shown beneficial results in non-ICU patients, the extrapolation to ICU populations is speculative.

In one of the first studies specifically evaluating pharmacological management of delirium in critically ill patients, olanzapine and haloperidol were shown to be equally efficacious in reducing the severity of delirium symptoms. Quetiapine was shown to be superior to placebo in time to resolution of the first episode of delirium in a small study of patients with delirium and orders to receive as-needed haloperidol. Another randomized controlled trial found that a single dose of risperidone sublingually after cardiac surgery reduced the incidence of delirium compared with placebo. However, the MIND study compared an atypical antipsychotic (ziprasidone) with haloperidol and placebo and found no differences in brain dysfunction outcomes between groups. Rivastigmine was studied as an adjunct to haloperidol and was not found to decrease the duration of delirium and might have contributed to increased mortality. Dexmedetomidine was reported as an adjunct to assist with weaning patients from psychoactive medications and was also shown to reduce the time to extubation, decrease the incidence of tracheostomy, and decrease ICU length of stay compared with haloperidol in critically ill patients with agitated delirium. Despite the intended use of these agents to treat delirium and improve cognition, it is important to recognize that they all have psychoactive effects that may further cloud the sensorium and promote a longer overall duration of cognitive impairment. Thus, using the lowest effective dose for the shortest possible time may be the most important delirium management recommendation.

SUMMARY

Altered mental status (delirium and coma) is a prevalent and costly problem in the critical care patient population that is associated with significant mortality, morbidity, and cost. Physicians must strive to balance the necessity and use of sedation with the cost of acute and long-term cognitive dysfunction to patients and society. With the appropriate attention, diagnostic tools, and medical practice, however, clinicians have the ability to significantly decrease the burden of this acute organ dysfunction. A management strategy that uses an integrated approach
Figure 4.

An empirical delirium management protocol. CAM-ICU, Confusion Assessment Method for the Intensive Care Unit; RASS, Richmond Agitation-Sedation Scale. Reproduced with permission from Dr. E. Wesley Ely (www.icudelirium.org).

DELIRIUM PROTOCOL

Sedation Scale/Delirium Assessment

Nondelirious (CAM-ICU negative)

Reassess brain function every shift
Treat pain and anxiety

Delirious (CAM-ICU positive)

Consider differential diagnosis, eg, sepsis, congestive heart failure, metabolic disturbances

Remove delirigenic drugs¹
Nonpharmacological protocol²

Stupor or coma while on sedative
an analgesic drugs⁷
(RASS –4 to –5)

Does the patient require deep sedation?

Yes
Perform SAT³

No
Reassess target sedation goal
Every shift
If tolerates SAT, perform SBT⁴

1. Consider stopping or substituting for delirigenic medications such as benzodiazepines, anticholinergic medications (metoclopramide, H₂ blockers, promethazine, diphenhydramine), and steroids.
2. See nonpharmacological protocol at right.
3. Analgesia: Adequate pain control may decrease delirium. Consider intermittent narcotics if feasible. Assess with objective tool.
4. Typical or atypical antipsychotics: While tapering or discontinuing sedatives, consider haloperidol, 2-5 mg IV initially (0.5-2 mg in elderly patients) and then every 6 hours. Guideline for maximum haloperidol dose is 20 mg/d due to ~60% D₂ receptor saturation. Consider using any of the atypicals (eg, olanzapine, quetiapine, risperidone, ziprasidone, or aripiprazole). Discontinue if high fever, QTc prolongation, or drug-induced rigidity.
5. Spontaneous awakening trial (SAT): Stop sedation or decrease infusion (especially benzodiazepines) to awaken patient as tolerated.
6. Spontaneous breathing trial (SBT): Trial of continuous positive airway pressure if on ≤50% FiO₂ and ≤8 cm H₂O positive end-expiratory pressure and oxygen saturation is 90%.

7. Sedatives and analgesics may include benzodiazepines, propofol, dexmedetomidine, fentanyl, or morphine.

NONPHARMACOLOGICAL PROTOCOL²

Orientation
• Provide visual and hearing aids.
• Encourage communication and reorient patient repetitively.
• Have familiar objects from patient’s home in the room.
• Attempt consistency in nursing staff.
• Allow television during day with daily news.
• Allow nonverbal music.

Environment
• Maintain sleep hygiene: Keep lights off at night, on during the day. Consider sleep aids (zolpidem, mirtazapine).
• Control excess noise (staff, equipment, visitors) at night.
• Ambulate or mobilize patient early and often.

Clinical parameters
• Maintain systolic blood pressure >90 mm Hg.
• Maintain oxygen saturations >90%.
• Treat underlying metabolic derangements and infections.
that includes meticulous attention to medication regimens, deployment of preventive strategies, standardization of delirium monitoring, and judicious use of pharmacological therapy can reduce the incidence and impact of this important clinical challenge.

REFERENCES


