REVIEW ARTICLE

Best Practices in Intensive Care Unit Sedation Across Diverse Patient Populations: An Evidence-Informed Review of US, Canadian and European Guidance

Mohamed Abdellatif[1]

Received: 10 November 2026 / Accepted: 11 November 2026
Corresponding Author: Mohamed Abdellatif
Affiliation: [1] Department of Pharmacy, Burjeel Medical City, Abu Dhabi, United Arab Emirates
© The Author(s) 2026 | Published in IJCPM

Citation: *Mohamed Abdellatif, et al. (2026). Best Practices in Intensive Care Unit Sedation Across Diverse Patient Populations: An Evidence-Informed Review of US, Canadian and European Guidance. International Journal of Clinical Pharmacy and Medicine, 1(4), 5. https://doi.org/10.68760/s2026145*

Abstract

Background: Sedation can either support or obstruct organ protection in critical illness. Standard light-sedation principles do not always apply to severe brain injury, acute respiratory distress syndrome (ARDS) requiring neuromuscular blockade, or refractory status epilepticus.
Objective: To translate recent US, Canadian-linked and European evidence into a practical, patient-population-specific approach to analgesia, sedation selection, monitoring and de-escalation in adult intensive care.
Approach: An evidence-informed narrative synthesis prioritizing multidisciplinary guidelines, trials and reviews published in 2022-2026, including 2025 SCCM PADIS, 2026 SCCM neuromuscular-blockade, 2026 Surviving Sepsis Campaign, European ARDS and neurocritical care guidance.
Key findings: Analgesia-first care, documented sedation goals, delirium screening and daily reassessment remain the default. Severe TBI may require deeper, physiology-directed sedation; severe ARDS may require short-term deep sedation during paralysis; refractory seizures demand EEG-guided anesthetic therapy; and septic shock requires particular attention to hemodynamic and organ-function changes.
Conclusion: The safest practice is a dynamic sedation prescription linked to the principal disease, a measurable target, a harm-monitoring plan and an explicit transition back to lighter sedation when feasible.

Keywords: analgosedation; critical care; traumatic brain injury; ARDS; status epilepticus; septic shock; delirium; neurocritical care

1. Introduction

Analgesia and sedation are not interchangeable interventions: pain relief treats nociceptive distress, whereas sedation controls arousal, anxiety and tolerance of essential treatment. In most mechanically ventilated adults, a light, individualized approach is preferred because prolonged deep sedation may delay awakening, assessment, mobility and liberation from ventilation. The 2025 US Society of Critical Care Medicine (SCCM) focused PADIS update conditionally favours dexmedetomidine over propofol when light sedation or delirium reduction is the dominant priority. It does not establish dexmedetomidine as universally superior in every critical illness. [1]

The 2026 Dutch multidisciplinary PADIS guideline similarly prioritizes light or no sedation when feasible, while explicitly recognizing exceptions for intracranial hypertension, neuromuscular blockade, proning and major trauma. A recent pharmacology review coauthored by Toronto-based critical care researchers emphasizes that sedative selection must reflect altered drug disposition, the primary organ injury and changing treatment goals. [2,3]

This review addresses adult intensive care patients with traumatic brain injury (TBI), acute respiratory distress syndrome (ARDS), refractory seizures, sepsis or septic shock, and post-cardiac arrest brain injury, followed by organ dysfunction and complex sedation scenarios. The central proposition is that sedation depth should be prescribed as a time-limited physiological intervention, not simply as a drug infusion.

2. Scope and evidence approach

This evidence-informed narrative review synthesizes English-language society guidance, randomized studies and clinically relevant reviews published primarily between

2022 and November 2026. US SCCM and American Thoracic Society recommendations, European ESICM, Dutch and UK guidance, and recent Canadian-linked critical care scholarship were prioritized. Documents from different jurisdictions were treated as sources of evidence rather than as interchangeable national mandates. This is not a systematic review, and no pooled treatment effect or formal risk-of-bias estimate is claimed. [4]

For clinical implementation, each recommendation is grouped into one of three practical categories: routine practice, condition-dependent adaptation, or specialist rescue therapy. Published reviews of ICU sedation protocols show why a single infusion target cannot safely cover a heterogeneous ICU: the protocol must specify pain assessment, sedation depth, triggers for reassessment and when routine awakening is unsafe. [5]

3. A practical sedation pathway for most mechanically ventilated adults

3.1. Establish a measurable target. Within the first ICU shift, record the reason sedation is needed, an expected level of responsiveness and a plan for reassessment. When deep sedation is not required, a Richmond Agitation-Sedation Scale (RASS) target in the light range (commonly -1 to +1, or a local equivalent) is reasonable; the 2026 Dutch guideline permits a broader light range from -2 to +1. Deep targets such as RASS -4 or -5 should have a documented indication, duration and exit criteria. RASS is not a valid pain scale. [2]

3.2. Treat pain before escalating hypnotics. Use patient self-report whenever possible; otherwise employ a validated behavioural tool such as the Critical-Care Pain Observation Tool (CPOT) or Behavioural Pain Scale, acknowledging their limitations during paralysis. Repositioning, procedures, fractures, invasive devices and anxiety can produce distress that is misread as insufficient sedation. Titrate opioid or non-opioid analgesia to the likely pain generator and reassess after interventions. A continuous opioid infusion must not become a substitute for a documented sedation plan. [1] [3]

3.3. Choose the least disruptive hypnotic. Propofol is rapidly adjustable and allows relatively prompt neurologic reassessment but can reduce arterial pressure and adds lipid calories. Dexmedetomidine often permits interactive sedation and facilitates communication or extubation, but bradycardia and hypotension may limit it; it is not reliable as the sole sedative for profound anesthetic-level suppression. Midazolam is useful for specific indications, including seizure-related anesthesia or clinically important withdrawal, but prolonged infusions may accumulate and postpone awakening. Selection should follow the clinical phenotype and local formulation, not a fixed hierarchy. [4] [1]

3.4. Link sedation to the ventilator and delirium bundle. During each nursing shift, compare achieved RASS with prescribed RASS, review ventilator synchrony, screen for delirium when assessable (CAM-ICU or ICDSC), and check readiness for breathing trials and mobilization. A spontaneous awakening trial is appropriate only when neurologic and respiratory contraindications have been excluded; it should never be automatic in uncontrolled intracranial hypertension, active status epilepticus or patients requiring profound sedation for lung protection. Family orientation, sleep protection, hearing and vision aids and early rehabilitation are foundational. [2,5]

3.5. Avoid treating every agitation episode as delirium. Assess pain, urinary retention, hypoxemia, withdrawal, metabolic disturbance, drug toxicity, ventilator dyssynchrony and intracranial pathology before increasing sedatives or antipsychotics. The AID-ICU randomized trial found no statistically significant improvement in days alive and out of hospital at 90 days with routine haloperidol compared with placebo; antipsychotic use should therefore be reserved for selected circumstances such as dangerous agitation after reversible causes are addressed. [6]

4. Traumatic brain injury and intracranial hypertension

4.1. Sedation as an element of brain protection. Severe TBI makes the routine light-sedation strategy conditional. Nociception, coughing, agitation, hypoxemia and excessive ventilatory effort can increase metabolic demand or intracranial pressure (ICP); conversely, excessive sedative-related hypotension may compromise cerebral perfusion pressure (CPP). The American College of Surgeons 2024 best-practice guidance supports staged management, frequent neurologic assessment and escalation directed by evolving brain physiology. A sedative is appropriate only if it protects the brain without producing preventable systemic harm. [7]

4.2. Preferred practical approach. Following airway stabilization and analgesia, propofol with a titratable short-acting opioid is frequently practical when rapid adjustment or ICP control is necessary. Midazolam may be an alternative or adjunct when propofol-induced circulatory depression, prolonged high-dose requirements or another contraindication limits its use. Dexmedetomidine may help during recovery or extubation when ICP is stable, but generally cannot replace deeper therapy in an unstable brain-injured patient. Ketamine is not an absolute contraindication solely because of TBI; its role depends on hemodynamic state, co-sedation, local practice and specialist review. [8,9]

4.3. Match titration to cerebral rather than behavioural outcomes. Integrate ICP trend, CPP, pupils, head position, imaging, EEG when indicated, oxygenation, $PaCO_2$, temperature and blood pressure. An isolated RASS score cannot determine cerebral safety. Contemporary discussions of ICP emphasize the value and limitations of multimodal monitoring and warn against applying a universal physiologic number without context. Acute increases in ICP prompt assessment of mechanical and

physiological causes, followed by staged analgesia, sedation and neurosurgical interventions as appropriate. [10]

4.4. Avoid reflexive sedation interruptions. A daily awakening test is not automatically safe when ICP is uncontrolled, cerebral perfusion is unstable or seizures are active. Instead, document when an examination can safely be attempted and coordinate reductions in sedation with the neurocritical care team. International observational data show substantial variation in acute brain-injury sedation practices; these findings describe associations, not superiority of a particular agent. [11]

4.5. Differentiate seizure prevention from treatment. Moderate-to-severe TBI may warrant short prophylactic antiseizure therapy in selected cases, but a 2024 Neurocritical Care Society guideline judged comparative evidence uncertain and suggested a course of no more than seven days when prophylaxis is chosen. Prophylaxis is not a reason to maintain anesthetic-level sedation or to defer a neurologic examination. [12]

5. Acute respiratory distress syndrome and neuromuscular blockade

5.1. Sedate for lung protection, not merely to quiet the ventilator. The European Society of Intensive Care Medicine (ESICM) 2023 ARDS guideline and the 2024 American Thoracic Society update prioritize protective ventilation and interventions selected according to disease severity. Sedative requirements increase when severe dyssynchrony threatens tidal-volume, plateau-pressure or prone-position goals; however, routine deep sedation is not itself a treatment for ARDS. Optimize analgesia, ventilator settings and reversible distress before escalating to deep sedation. [13,14]

5.2. Neuromuscular blockade creates a special safety obligation. The 2026 SCCM guidance conditionally supports neuromuscular blockers in adults with ARDS and PaO2/FiO2 below 150 when hypoxemia persists or lung-protective targets cannot be achieved despite appropriate sedation. Paralytic agents provide no analgesia, consciousness suppression or amnesia. Administer adequate analgesia and hypnosis before and throughout paralysis; use clinical assessment and, where appropriate, processed EEG as adjuncts when behavioural scoring is impossible. Sedation depth must not be inferred from immobility. [15]

5.3. Reassess the indication for paralysis each day. If satisfactory oxygenation and protective ventilation can be maintained without continuous paralysis, lighten the regimen toward assessment-friendly sedation. The choice between bolus and infusion neuromuscular blockade, monitoring intensity and duration depends on patient trajectory and the evidence base; blanket paralytic infusions are not justified solely by prone positioning. During proning, check enteral access, pressure points, airway security and hemodynamics while ensuring that sedation and analgesia are uninterrupted. [13,15]

5.4. Weaning is a distinct phase. When lung mechanics and oxygenation improve, reduce unnecessary sedative accumulation, assess spontaneous breathing and restore an interactive target. Consider dexmedetomidine when ongoing mild sedation is needed near extubation, provided blood pressure and heart rate tolerate it. Patients with concomitant TBI or refractory seizures may not be suitable for routine ARDS awakening practices. [1]

6. Convulsive and refractory status epilepticus

6.1. The target is electrographic seizure control. Sustained convulsive seizure activity requires immediate resuscitation, prompt benzodiazepine treatment and timely administration of an appropriate maintenance antiseizure medication. Refractory status epilepticus generally follows failure of a benzodiazepine and a non-benzodiazepine antiseizure drug; specialist-led anesthetic infusion and continuous EEG then become central. The 2025 UK multidisciplinary ICU guidance explicitly places refractory and super-refractory cases within a specialist pathway. [16]

6.2. Deep sedation is therapeutic in refractory status. Midazolam, propofol and other continuous anesthetic strategies may be used under EEG guidance, with ketamine or barbiturates reserved for selected refractory situations. The precise EEG endpoint, duration of seizure suppression and taper schedule should be agreed with neurocritical care or epilepsy specialists. Clinical stillness does not prove seizure cessation, especially after paralysis; suspected non-convulsive seizures require EEG confirmation. [16]

6.3. Prevent predictable complications. Secure the airway when needed; closely monitor perfusion, lactate, acid-base status, triglycerides, muscle injury and cumulative sedative exposure. Prolonged high-dose propofol warrants vigilance for propofol-infusion syndrome. Treat infection, metabolic triggers, toxic exposures and structural pathology concurrently. Do not rapidly withdraw anesthetic therapy without an agreed EEG-guided plan, maintenance antiseizure treatment and a rescue strategy. [16]

6.4. De-escalation differs from routine ICU sedation. Whereas most ventilated patients benefit from daily efforts to awaken, seizure recurrence during anesthetic reduction may be harmful. For refractory status epilepticus, medication reduction is timed to sustained EEG seizure control and underlying-cause management, rather than the calendar day. [16]

7. Sepsis and septic shock

7.1. Perfusion and sedation are coupled. The 2026 Surviving Sepsis Campaign (SSC) guideline updates hemodynamic, antimicrobial and respiratory management. In septic shock, rapid changes in vasopressor needs, distribution volume and organ perfusion make sedative sensitivity unpredictable. Sedation should not mask worsening encephalopathy, hypoperfusion or respiratory deterioration. Treat the source of shock while choosing doses that minimize avoidable cardiovascular depression. [17]

7.2. An analgesia-first, light-target approach is reasonable after stabilization. Propofol and dexmedetomidine are commonly preferred over prolonged benzodiazepine-based sedation when no condition demands deep anesthesia. Propofol can precipitate hypotension; dexmedetomidine can cause bradycardia and hypotension, particularly around loading doses or unstable resuscitation. Avoiding an initial loading bolus, starting cautiously and titrating against RASS and pressure response are practical choices, but no single drug has demonstrated universal superiority in all septic shock phenotypes. [1,18]

7.3. Shock with ARDS requires two plans. When sepsis is accompanied by moderate-severe ARDS, lung-protective ventilation and proning determine whether temporary deep sedation is necessary. The 2026 SSC guidance favours intermittent neuromuscular-blocker boluses over continuous infusion in sepsis-associated moderate-severe ARDS, while separate 2026 SCCM guidance allows blockade for persistent hypoxemia or failure to achieve ventilator targets. The practical reconciliation is to reserve continuous paralysis for selected unresolved problems and reassess frequently, not to presume every proned patient needs it. [15,17]

7.4. Review renal and hepatic dysfunction daily. Circulatory failure can evolve into acute kidney injury or impaired hepatic metabolism, causing accumulation of active metabolites or extended sedation after drug cessation. A previously stable infusion rate may become excessive; compare arousal trends with changes in organ function and cumulative doses. [3]

8. Post-cardiac arrest and other acute brain injuries

Post-cardiac arrest care often requires sedation to tolerate mechanical ventilation, temperature management and treatment of seizures or shivering. However, sedative accumulation obscures serial examinations and can delay appropriate prognostication. The 2025 European Resuscitation Council/ESICM post-resuscitation guideline supports multimodal assessment and avoiding premature conclusions while sedative confounding remains. [19]

Prefer titratable regimens when feasible, reassess the need for analgesics and hypnotics with each transition in temperature and hemodynamic management, and use EEG when seizures are possible. Rewarming, hypothermia, evolving organ failure and prolonged infusions can change the time required for reliable awakening. Sedative discontinuation does not mean an immediate examination is interpretable. [19]

9. Special populations and pharmacologic safety

9.1. Acute kidney injury or renal replacement therapy. Review opioid metabolites, benzodiazepine accumulation, concomitant antiseizure agents and potential drug loss or adsorption in extracorporeal circuits. Fentanyl and short-acting propofol may be easier to titrate than morphine or prolonged midazolam in selected patients; remifentanil can be useful when frequent neurological checks are required, subject to local availability and expertise. Sedative dose must be titrated to clinical effect rather than estimated solely from creatinine clearance. [2,3]

9.2. Cirrhosis or severe hepatic dysfunction. Minimize cumulative benzodiazepines and other hepatically cleared agents when feasible, consider altered protein binding and encephalopathy, and avoid interpreting delayed awakening as proof of neurological injury. Sedatives remain necessary when safety or mechanical ventilation requires them, but doses may need substantial adjustment. A multidisciplinary medication review can identify drug-drug interactions and additional contributors to impaired consciousness. [3]

9.3. Older or frail adults. Reduce unnecessary sedative exposure, reassess pain frequently, and avoid defaulting to a prolonged deeply unresponsive state. Frailty, baseline cognitive impairment, sensory deprivation and polypharmacy increase vulnerability to delayed mobilization and confusion. Use explicit functional and respiratory recovery goals; individualize rather than equate age with a fixed dose reduction. [1,2]

9.4. ECMO and major obesity. Circuit sequestration, altered pharmacokinetics and prolonged ICU courses may create variable sedative requirements. In obesity, avoid extrapolating all continuous-infusion doses directly from total body weight without reviewing the drug-specific dosing scalar and toxicity risk. Drug concentrations and bedside response may diverge, particularly early after circuit initiation or changes. Specialist pharmacy review is appropriate when escalating beyond usual titration ranges. [3]

9.5. Withdrawal syndromes. Alcohol or benzodiazepine withdrawal and selected epileptic emergencies are exceptions to indiscriminate benzodiazepine avoidance. Treat the withdrawal mechanism directly, use objective assessment when possible, and maintain additional monitoring for respiratory depression and accumulation. Dexmedetomidine may attenuate sympathetic manifestations but does not replace GABAergic treatment or seizure prevention. [4]

10. Practical agent selection and monitoring

For all drug classes, prescribe a clinical target and a reassessment interval before selecting an infusion. Propofol is useful for rapid titration and deeper hypnosis but can cause hypotension, hypertriglyceridemia and, with prolonged high exposure, propofol-infusion syndrome. Dexmedetomidine supports an arousable state but requires close heart-rate and blood-pressure monitoring. Opioids provide analgesia but can accumulate and delay extubation; consider non-opioid adjuncts and bowel regimens. Midazolam offers anticonvulsant and anxiolytic effects but is a poor default for prolonged routine sedation when rapid wake-up is desirable. [2,4]

Ketamine may offer analgesic and anesthetic benefits and can be considered in selected shock, neurocritical care or refractory seizure scenarios, but it should not be promoted as universally vasopressor-sparing or free of psychotropic effects. Deep sedation strategies require explicit monitoring for hemodynamic intolerance, metabolic abnormalities, withdrawal and prolonged ventilation. When a drug is being used for a condition-dependent indication, record the trigger to stop it. [3] [9]

11. A reproducible daily bedside workflow

At the start of each shift, the nurse, respiratory therapist, intensivist and pharmacist should be able to answer six questions: (1) why is sedation indicated today; (2) what pain and RASS targets are ordered; (3) are ICP, oxygenation, EEG, vasopressors or paralysis changing the target; (4) are treatment-related adverse effects emerging; (5) can sedation be lightened safely; and (6) what are the next reassessment and liberation milestones? A written exception to routine awakening is as important as a written routine awakening plan. [5]

The pharmacy review should examine weight and dosing scalar, infusion duration, cumulative opioid or benzodiazepine exposure, propofol caloric contribution, triglycerides and lactate when appropriate, concomitant QT-prolonging or serotonergic agents, organ dysfunction, compatibility, line access and infusion interruptions during transfer or proning. Sedation audits should track time spent within target RASS, documented pain scores, avoidable days of paralysis or deep sedation, delirium assessment, unplanned extubations, hypotension, and time to awakening rather than sedative consumption alone. [3]

12. Differences in the US, Canadian and European evidence context

In the United States, the 2025 SCCM PADIS focused update provides an agent-specific conditional preference for dexmedetomidine when interactive sedation or delirium reduction is paramount. Its 2026 neuromuscular-blockade guideline places additional emphasis on assuring adequate sedation and analgesia during paralysis. Canadian critical care teams can apply these data alongside local health-authority protocols, ventilator-liberation resources, drug formularies and neurocritical care pathways; a single distinct, nationally binding Canadian adult ICU sedation guideline was not identified for this narrative synthesis. Recent Canada-linked scholarship stresses pharmacologic individualization rather than a single agent rule. [1,3,15]

European guidance complements this framework. The 2026 Dutch guideline makes light sedation the default with clearly named exceptions; 2023 ESICM ARDS guidance and 2025 UK status-epilepticus guidance define population-specific respiratory and electrographic priorities; and the 2025 ERC/ESICM post-cardiac-arrest guideline addresses sedation-related confounding during neurologic recovery. These recommendations should be adopted through multidisciplinary local protocols rather than transposed without considering resources and patient mix. [2,13,16,19]

13. Limitations and future directions

This review is deliberately selective and is not a formal systematic review. Some decisions, particularly drug combinations for intracranial hypertension, optimal anesthetic depth in refractory status epilepticus and sedation monitoring during neuromuscular blockade, rest on low-certainty evidence or expert consensus. Randomized evidence is frequently extrapolated from mixed ICU populations to patients with unstable brain injury, profound shock or extracorporeal support. Differences in geography and the organization of ICU care further limit direct generalization.

Future studies should compare physiology-driven sedation protocols with generic RASS-based approaches, clarify patient-reported outcomes and delirium after deep sedation, identify better measures of awareness during paralysis, and evaluate pharmacist-led medication safety. An effective pathway should preserve an option for deep sedation while specifying when that option is no longer required.

14. Conclusion

The best ICU sedation strategy is neither uniformly shallow nor uniformly deep. Most ventilated adults benefit from pain-first, protocolized, light sedation with frequent readiness checks; severe TBI may require brain-protective control of stimulation and ICP; severe ARDS may require short-term deeper sedation with selected neuromuscular blockade; refractory status epilepticus requires EEG-guided anesthetic therapy; and septic shock requires cautious hemodynamic titration. Current US, Canadian-linked and European evidence supports a documented, condition-specific sedation goal that is reviewed at least daily and adapted to recovery.

Table 1. Practical disease-adapted sedation targets and safety priorities

Population	Primary sedation objective	Practical approach / important safety check
Routine ventilated adult	Comfort, interaction, early liberation	Treat pain first; aim for light RASS; propofol or dexmedetomidine as appropriate; screen delirium and reassess daily.
Severe TBI / rising ICP	Avoid secondary brain injury	Short-acting opioid plus titratable hypnotic; follow ICP/CPP/neurologic trend; defer awakening if unsafe.
Moderate-severe ARDS	Protect ventilation and oxygenation	Correct dyssynchrony; deep sedation only if needed; during paralysis assure both analgesia and unconsciousness.
Refractory status epilepticus	Electrographic seizure suppression	Continuous EEG, specialist anesthetic infusion, treat underlying cause; taper to EEG plan, not a routine SAT.
Septic shock	Comfort without worsening perfusion	Analgesia-first; cautious sedative initiation and frequent MAP/pressor checks; avoid unneeded loading boluses.
Post-cardiac arrest	Support ventilation while preserving valid prognosis	Use titratable agents; reassess after temperature control; avoid premature prognosis while sedative effects persist.
Renal / hepatic dysfunction	Prevent accumulation and delayed awakening	Review pharmacokinetics, metabolites, organ function and all concomitant drugs; adjust to achieved effect.

Abbreviations: ARDS, acute respiratory distress syndrome; CPP, cerebral perfusion pressure; EEG, electroencephalography; ICP, intracranial pressure; RASS, Richmond Agitation-Sedation Scale; SAT, spontaneous awakening trial.

Declarations

Author contributions: All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by all authors. All authors read and approved the final manuscript.

Ethics Approval: Not applicable

Consent to Participate: This study did not involve human participants, animal experimentation, or confidential patient clinical data.

Consent for Publication: Not applicable.

Funding Statement: The authors declare that no funds, grants, or other financial support were received during the preparation of this manuscript.

Data Availability Statement: Data sharing is not applicable to this article as no new datasets were generated or analyzed during the current study.

References

1. Lewis K, Balas MC, Stollings JL, et al. A focused update to the clinical practice guidelines for the prevention and management of pain, anxiety, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. Crit Care Med. 2025;53(3):e711-e727. https://doi.org/10.1097/CCM.0000000000006574

2. van den Boogaard M, Ham F, van Dusseldorp I, et al. The Dutch Clinical Practice Guideline: Pain, analgesia/sedation, delirium, immobility and sleep disturbances; PADIS. Intensive Crit Care Nurs. 2026;95:104438. https://doi.org/10.1016/j.iccn.2026.104438

3. Morales Castro D, Carini FC, Fan E. Sedation, analgesia, and neuromuscular blockade. Clin Chest Med. 2026;47(3):463-481. https://doi.org/10.1016/j.ccm.2026.04.003

4. Northam KA, Phillips KM. Sedation in the ICU. NEJM Evid. 2024;3(11):EVIDra2300347. https://doi.org/10.1056/EVIDra2300347

5. Remmington C, McKenzie CA, Camporota L, et al. Protocolised analgosedation in mechanically ventilated adult patients: a scoping review. J Crit Care. 2026;97:155766. https://doi.org/10.1016/j.jcrc.2026.155766

6. Andersen-Ranberg NC, Poulsen LM, Perner A, et al. Haloperidol for the treatment of delirium in ICU patients. N Engl J Med. 2022;387(26):2425-2435. https://doi.org/10.1056/NEJMoa2211868

7. American College of Surgeons Trauma Quality Programs. Best Practices Guidelines in the Management of Traumatic Brain Injury. Chicago, IL: American College of Surgeons; 2024. https://www.facs.org/quality-programs/trauma/quality/best-practices-guidelines/

8. Slot RER, Helbok R, van der Jagt M. Update on traumatic brain injury in the ICU. Curr Opin Anaesthesiol. 2025;38(2):93-99. https://doi.org/10.1097/ACO.0000000000001468

9. Merhavy ZI, Raeburn T, Torres-Ayala GM, McCulloch MA, Varkey TC. Sedation and analgesia strategies in the neuro intensive care unit. World J Crit Care Med.

2025;14(4):111787. https://doi.org/10.5492/wjccm.v14.i4.111787

10. Taccone FS, Arabi Y, Baggiani M, et al. Intracranial pressure physiology, monitoring and individualized management in the acute brain injured patient. Intensive Care Med. 2026;52(10):2108-2128. https://doi.org/10.1007/s00134-026-08558-4

11. Feng SN, Laws LH, Diaz-Cruz C, et al. Sedation practices in mechanically ventilated neurocritical care patients from 19 countries: an international cohort study. Neurocrit Care. 2025;43(1):232-242. https://doi.org/10.1007/s12028-024-02200-1

12. Frontera JA, Gilmore EJ, Johnson EL, et al. Guidelines for seizure prophylaxis in adults hospitalized with moderate-severe traumatic brain injury: a clinical practice guideline from the Neurocritical Care Society. Neurocrit Care. 2024;40(3):819-844. https://doi.org/10.1007/s12028-023-01907-x

13. Grasselli G, Calfee CS, Camporota L, et al. ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies. Intensive Care Med. 2023;49(7):727-759. https://doi.org/10.1007/s00134-023-07050-7

14. Qadir N, Sahetya S, Munshi L, et al. An update on management of adult patients with acute respiratory distress syndrome: an official American Thoracic Society clinical practice guideline. Am J Respir Crit Care Med. 2024;209(1):24-36. https://doi.org/10.1164/rccm.202311-2011ST

15. Erstad BL, Cobas MA, Qadir N, et al. Society of Critical Care Medicine guidelines for the administration of neuromuscular blockade in adults with acute respiratory distress syndrome. Crit Care Med. 2026;54(3):634-643. https://doi.org/10.1097/CCM.0000000000007002

16. Mullhi R, Hayton T, Midgley-Hunt A, et al. Guidance for: the acute management of status epilepticus in adult patients. J Intensive Care Soc. 2025;26(2):249-262. https://doi.org/10.1177/17511437251321338

17. Prescott HC, Antonelli M, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2026. Crit Care Med. 2026;54(4):725-812. https://doi.org/10.1097/CCM.0000000000007075

18. Marra A, Smith RJ, Pandharipande PP, Hayhurst CJ. Optimal sedation in ventilated patients with sepsis and septic shock. Crit Care Clin. 2025;41(4):775-790. https://doi.org/10.1016/j.ccc.2025.05.004

19. Nolan JP, Sandroni C, Cariou A, et al. European Resuscitation Council and European Society of Intensive Care Medicine Guidelines 2025 Post-Resuscitation Care. Resuscitation. 2025;215(Suppl 1):110809. https://doi.org/10.1016/j.resuscitation.2025.110809