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Journal of Anesthesia & Critical Care: Open Access

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Sedation and analgesia in critical care

Volume 7 Issue 3 - 2017

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Received: February 16, 2014 | **Published:** June 02, 2017

Introduction

Sedation and analgesia go hand in hand in achieving success in management of critically ill patients. It is of great importance in all types of intensive care units: surgical, medical, neurosurgical, oncology and cardiac. A patient-based approach is incorporated, taking into consideration the predisposing and causative factors of pain, agitation and delirium as well as patient-safety. A successful outcome can be achieved by using patient-appropriate analgesia and sedation scales; proper medication selection; monitoring; and protocol-based weaning strategies from sedative-analgesic medications. This narrative review chapter is intended to elucidate the reasons and techniques to achieve adequate sedation and analgesia. Focus is also given on the various scales used for its accurate assessment.

Background

Sedation and analgesia outside the operation theatre is a dynamic and systematic procedure intended to achieve the goals of optimum critical care management. In this regard, nurse anesthetists and intensive care nursing personnel are indispensable. Their dedicated efforts guided by the intensivists are undoubtedly responsible for improved patient outcomes. This article is primarily intended for them and other budding critical care enthusiasts as a mark of recognition to their tremendous contribution.

Discussion and brief review of literature

Pain, agitation and delirium are termed the "ICU triad" and appropriate sedation-analgesic techniques must be adopted to mitigate the ill-effects of this triad. Several randomized trials advocate the principle of "sedation holiday" with daily sedation interruptions to reduce drug side-effects or a protocolized reduction of sedation to minimum possible dose for facilitating weaning. Recent guidelines prefer the use of nonbenzodiazepine sedatives (either propofol or dexmedetomidine) over benzodiazepines for improving clinical outcomes in mechanically ventilated adult ICU patients. Despite several comparative studies, no single agent has been shown to be superior to other agents for ICU sedation. Deep sedation is required only in patients having severe respiratory failure, intra-cranial hypertension, status epilepticus, tetanus and concurrent use of muscle relaxants. In particular, some studies highlight that accurate assessment and measurement of pain in critical care patients is difficult. With advancements in drug discoveries, newer drugs can satisfy our unending quest for an ideal analgo-sedation agent.

Definitions

Analgesia[1] is defined as pain control in the form of diminution or elimination of pain. American Society of Anesthesiologists[2] and the American Dental Association have defined sedation and analgesia to comprise a continuum of states ranging from mild sedation (anxiolysis) to general anesthesia. Minimal sedation is defined as a minimally depressed level of consciousness, which retains the patient's ability to independently and continuously maintain an airway and to respond normally to tactile stimulation and verbal command. Moderate sedation (conscious sedation) is a drug induced depression of consciousness during which patients respond purposefully to verbal commands or light tactile stimulation, with patent airway and spontaneous ventilation. Deep sedation/analgesia is a drug induced depression of consciousness during which patients cannot be easily aroused, but respond purposefully following repeated or painful stimuli. Patients may require assistance in maintaining a patent airway and the cardiovascular function is usually maintained.

Why sedation and analgesia?

Sedation is important in the ICU to facilitate amnesia during critical illness, to prevent delirious patients from causing harm to self and others, to facilitate invasive management, to promote ventilator-patient synchrony, to circumvent post-traumatic stress disorder[3] and to relieve dyspnea. Inability to meet goals of proper sedation and analgesia has deleterious consequences in the form of increase in adverse events, poor overall outcomes, longer ICU stays and economic effects. Analgesia is as important a component of ICU patients as is sedation. An "analgesia-first" approach can prove beneficial in agitated or delirious patients. Following are the potential consequences of poor pain management:

a. Occurrence of physiological stress
b. Metabolic and endocrine responses
c. Difficulty in sedation
d. Development of chronic pain

Children[4] of all age groups and neonates (including premature) experience pain, just like adults. Lack of proper guidelines, reluctance to incorporate pain relief as a part of overall management and inadequate training of intensive care nurses are responsible for improper sedation or analgesia practices. Multi-modality should be the dictum for pain management in both pediatric and adult population.

Sedation scales

One of the most important and technically challenging tasks in this entire topic is, how to quantify a patients' current and desired level of sedation. Critically ill patients, especially intubated ones may not be able to express their requirements correctly. Hence, several scores or scales were developed for measuring degree of sedation at any

given point and to guide therapy. Two commonly used sedation scales in ICU include: Ramsay sedation.[5] scale and Richmond Agitation Sedation Scale (RAAS).[6] In Ramsay sedation scale, scales 1 to 3 constitute awake states and scales 4 to 6 constitute asleep levels.

Sedative agents

There is a wide choice of sedative medications which can be used to achieve the desired level of sedation:

a. Benzodiazepines: Diazepam, Midazolam, Lorazepam

b. Opioids: Morphine, Fentanyl, Remifentanyl, Sufentanil

c. Anesthetics: Propofol, Fospropofol, Ketamine

d. Alpha-2 agonists: Dexmeditomidine

e. Anti-delirium drugs: Haloperidol

Benzodiazepines[7]: They are metabolized in the liver by hepatic microsomal oxidation or glucoronidation. Their metabolism may be impaired in elderly and in patients with liver disease. Metabolites of diazepam (dose 2-10mg) can accumulate and prolong its sedative effects. Hence it is not used as infusion. Midazolam (Dose 1-5mg) has a fast onset and short duration of action. It is highly lipophilic, with an elimination half-life of 1-4 hours. It is the most commonly used sedative agent which can be used for continuous infusions. Alpha-hydroxy midazolam is the active metabolite which accumulates with prolonged infusions. Lorazepam (dose 1-5mg) is the slowest onset, longest acting benzodiazepine, whose metabolism is not affected by liver disease. It can lead to ethylene glycol toxicity on prolonged infusions.

Drug	Dose	Remarks
Midazolam	1-2 mg bolus I.V 2-3mg per hour infusion	Metabolite active, accumulates in liver disease
Lorazepam	1-2mg bolus I.V 1-5mg per hour infusion	Metabolism not affected by liver disease
Diazepam	5 or 10mg bolus I.V	Not suitable for infusions, Metabolites active & accumulate
Propofol	Induction dose: 1.5 – 2.5mg/kg I.V Maintenance: For sedation 25-75mcg/kg/min For hypnosis 100-200mcg/kg/min	Rapid recovery; Causes pain on injection, apnea, cardiovascular depression, anti-emetic properties, hyperlipidemia, anti-oxidant activity, neuro-protective effects.

Propofol[8]: It is one of the most commonly used intravenous sedative in all ICUs. It can be given in a bolus dose of 2mg/kg followed by a maintenance infusion of 5 – 50 microgram/kg/min or 4-5 microgram/kg/hour. Onset of action is 1-2 min and elimination half life is 1-4hours. It has no active metabolite, but it can accumulate in fatty tissues (being lipophilic) and cause delayed effects. It is rapidly cleared from central circulation by hepatic metabolism. Hypotension is a common side effect. Prolonged infusions can lead to propofol infusion syndrome, characterized by bradycardia, cardiac failure, metabolic acidosis, rhabdomyolysis and hyperkalemia.

Ketamine: It is a unique agent as it provides both sedation and analgesia. It has a quick onset of action (30 seconds) and is suitable for procedural sedation in the ICU, especially in patients with reactive airway disease and those with depressed cardiac function. Its side effects include increase in intra-cranial pressures, oral/airway secretions and hallucinations. It can be administered in a continuous infusion dose of 10-50 mcg/Kg/hour.

Newer agents

Fospropofol[9]: is a prodrug which is converted to propofol inside the body. It is water soluble as opposed to propofol (lipophilic) and has a much smaller volume of distribution. Drug contamination (which is a problem with propofol) is less of a concern here. Its onset of action is slightly longer than propofol and it is safe to use in renal insufficiency.

Dexmeditomidine[10]: It is a specific alpha-2 agonist that acts centrally to inhibit nor-epinephrine release. Its combined sedative and analgesic effect make it an ideal drug for ICU sedation. It does not cause respiratory depression and allows for a more awake and interactive patient, with lesser incidence of delirium. Its primary side effects are bradycardia, hypotension and sore throat. Discontinuation of a prolonged infusion can cause a withdrawal syndrome characterized by agitation, tachycardia and hypotension. Dose: 0.2 – 0.6 microgram/Kg/Hour.

AnaConDa system[11]: It is an inhalational anesthesia system designed in Sweden for use with ventilators in the ICU. It is attached to the mechanical ventilators to recycle the anesthetic agents. Volatile anesthetics have a better pharmacokinetic profile than many intravenous sedatives, resulting in quicker and reliable awakening and extubation, which can be a boon for the critically ill patient.

Role of muscle relaxants: The neuromuscular blocking agents[12] have been used since time-immemorial for critically ill patients on ventilatory support. They have gone into disrepute as they are associated with prolonged mechanical ventilation, delayed awakening, residual muscular weakness and its economic implications. They are also known to cause critical illness myopathy and neuropathy. Monitoring of the neuromuscular block is recommended if their use is prolonged or longer acting agents are employed. Nevertheless, they are important to facilitate intubation, promote patient- ventilator synchrony and to decrease work of breathing. It is better to use a shorter-acting agent which does not accumulate on prolonged use and which is not dependent on body metabolism for elimination.

The following are the muscle relaxants used:

1. Pancuronium: It's a long acting agent with vagolytic properties. It is usually used in cardiac surgeries and where a tachycardiac response is required. It is generally administered only in post-cardiothoracic surgery ICU patients.

2. Vecuronium: It does not cause tachycardia. Caution needs to be exercised in patients with kidney disease as its metabolites can accumulate in renal failure.

3. Atracurium: It is an intermediate acting neuromuscular blocking agent which does not depend on either liver or kidney for elimination. It is degraded by Hoffmann elimination and hence its toxicity is minimal. It causes histamine release and can lead to hypotension.

4. Cis-atracurium: It is the most preferred muscle relaxant for prolonged infusions. But its availability is limited to larger centres.

5. Rocuronium: As it has a rapid onset of action (1-2 minutes), Rocuronium Bromide is recommended for rapid sequence intubation in a bolus dose of 1-2 mg/Kg.

Precautions

The titration of the sedative dose to a defined endpoint is recommended with systematic tapering of the dose or daily interruption

with re-titration to minimize prolonged sedative effects. Patients who were woken up on a regular basis during their ICU stay had lesser days on ventilator. It is important to use titrated doses and infusions to prevent the occurrence of side effects. Over-sedation in a patient with unprotected airway can be disastrous, leading to aspiration, unplanned intubations, raised intracranial pressure and even death. Inadequate sedation can be very distressing for the patient leading to unwanted alteration in physiological parameters, increased work of breathing, exhaustion and increased myocardial oxygen demand. Institutions must device their own sedation guidelines, depending upon the resources available, so that patient safety and comfort is not compromised. Antagonist or reversal agents 13 must be readily available for treating drug overdoses, viz. Flumazenil (0.2-1mg I.V) for Benzodiazepines and Naloxone Hydrochloride (0.4-2mg) for Opioids. Muscle relaxants must be used sparingly, only to tide over the crisis and they must not be substituted for sedatives. Adequate pain control is the pre-requisite to achieving desirable sedation.

Special concern in current day Critical Care: Delirium

Delirium.[14] is characterized by an acute onset of disturbance in cognitive abilities with a fluctuating course over time. It is a multifactorial form of brain dysfunction leading to increased mortality, ventilator days and ICU length of stay. The Society for Critical Care Medicine recommends daily noting of delirium in mechanically ventilated patients. There are 2 well validated tools for delirium assessment: *CAM-ICU* (Confusion Assessment Method for the Intensive Care Unit, consisting of noting the presence or absence of Acute onset/fluctuating course, Inattention, Disorganized thinking and Altered level of consciousness) and *ICDSC* (Intensive Care Delirium Screening Checklist). Use of simple measures such as reorientation to time/place/person, enhancement of sleep environment, early mobilization and minimization of medications associated with delirium can help decrease its incidence. Critical nurses play a significant role both in the quantification and management of delirium.

Haloperidol, an anti-psychotic, is the drug most commonly used to treat delirium. It acts by blocking dopamine receptors in the brain, which causes tranquility, decreased initiative and drowsiness. It may be administered in the doses of 1-2 mg intravenously and titrated to the effect with a doubling of the dose every 30 minutes as necessary. It may be repeated every 8-12 hourly. It can cause extra-pyramidal side effects such as dystonia, akathisia, pseudo-parkinsonism, heart rhythm disturbances and neuroleptic malignant syndrome. Newer agents, such as *Rivastigmine* (a cholinesterase inhibitor), are being tried for treatment of ICU delirium. Needless to emphasize here, prevention is better than cure.

Pain and analgesia

Pain not only causes human suffering, but also leads to relationship or job disruption and has a tremendous economic impact on society. Pain is an universal aftermath of injury. Despite the tremendous scientific and technological advances, pain still remains inappropriately or inadequately treated. The word pain is derived from the Latin *poena,* meaning punishment.Pain is defined by IASP.[15] (International Association for Study of Pain) as: an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." It has 3 components: sensory-discriminative, motivational-affective and cognitive-evaluative. Acute pain[16] is defined as a physiologic response to adverse chemical, thermal or mechanical stimulus, associated with surgery, trauma and acute illness. Unmyelinated C or thinly myelinated A delta afferent fibres convey pain sensation. Pain mediators include bradykinin, phospholipase C and nitric oxide, leading to the final common pathway of increase in intracellular calcium and protein kinase levels. Allodynia implies pain by a stimulus, which does not normally provoke pain. Incident pain is generated by mechanical factors characteristic of movement and position. Hyperalgesia is an increased response to a stimulus that is normally painful, due to abnormal processing of nociceptor input. Paresthesia is any abnormal sensation, whether spontaneous or evoked. Neuropathic pain is that initiated or caused by a primary lesion or dysfunction in the nervous system.

Chronic pain[17] is defined as pain persisting for longer than the expected time for an injury to heal and temporally, pain lasting beyond 3 months is deemed chronic. Complex regional pain syndrome *(CRPS)* is divided into 2 disorders: type 1, which occurs following a soft tissue injury (similar to reflex sympathetic dystrophy) and type 2, occurring after well-defined nerve injury (similar to causalgia). Analgesia means absence of pain in response to stimulation, which would normally be painful. It can be produced peripherally or centrally.

Pain scoring scales

Assessment of the severity of pain is a very important part of pain management. It is of special importance in the critically ill, as pain not only causes deleterious physiologic effects but also the patient may not be able to express pain properly. There are various scales or pain scores available to help the caregiver. Assessment can be difficult in the pediatric population, where more pictorial representations and expertise are required to elicit pain levels.

The following is a brief description of the pain assessment scales[18]:

1. Wong-Baker FACES pain rating scale: Faces are drawn on a straight line to pictorially describe the severity of pain from no hurt (Face 0) to worst hurt (Face5). This is recommended for children aged 3 years and above. They have to be asked to choose the cartoon face that best describes how he/she is feeling.
2. Numeric pain rating scale: It's a simple, linear pain scale from 0 to 10, where 0 stands for no pain, 5 for moderate pain and 10 for worst possible pain.
3. VAS (Visual Analogue Scale): It is the most commonly used scale in pain practice. It uses a 10cm long line with one end marked as no pain and the opposite end marked as worst pain.
4. McGill pain questionnaire: Frequently known as McGill pain index, it is an elaborate, self- report questionnaire allowing patients to describe their intensity and quality of pain.
5. Pain Quality Assessment Scale: PQAS measures the various aspects and types of pain that a patient experiences. There are 20 questions regarding the intensity, sharpness, tenderness, hot/cold, dullness, shooting, tingling and radiation of pain.
6. CRIES scale: It stands for Crying, Requirement of oxygen, Increased vital signs, Expression of face and Sleep. Each parameter is scored from 0 to 2, based on changes from baseline. It is very helpful for postoperative pain in neonates.
7. FLACC scale: Stands for Face, Legs, Activity, Crying and Consolability scale. It is designed for infants and uses a 0 to 10 scoring.
8. CHEOPS scale: Stands for Children's Hospital for Eastern Ontario Scale. It is intended for children aged 1-7 years, with a score of > or =4 signifying pain.

Pain scoring scales in mechanically ventilated patients

Mechanically ventilated cannot effectively communicate symptoms of pain. The following are the tools available to assess pain objectively[19]:

i. NRS (Numeric Rating Scale) – It uses a 0 to 10 scale, anchored by the descriptors 'no pain' and 'pain as bad as it could be', where even delirious patients communicate by pointing.

ii. BPS (Behavior Pain Scale): It can be tested in adults, especially for procedural pain. 3 aspects are considered: Facial expression, upper limbs movement and compliance with ventilation. Each is scored from 1 to 4, according to patient response.

iii. Critical-Care Pain Observation Tool: CPOT was developed in French and translated into various languages. It includes 4 behavioral categories: facial expression, body movements, muscle tension and compliance with the ventilator for ventilated patients. Items in each category are scored from 0 to 2, with a possible total score ranging from 0 to 8.

iv. Physiological parameters: Heart rate, blood pressure and respiratory rate; change in these indicators following pain therapy can guide us regarding the adequacy of analgesia.

v. Non-verbal Pain Scale: It is a relatively accurate score for ventilated patients. It uses 5 parameters: Facial expression, Activity/Movement, Guarding, Physiological signs 1(vitals: systolic B.P, Heart rate, Respiratory rate) and Physiological signs 2 (Temperature, Pupils, Perspiration, Pallor). Each parameter is graded from 0 to 2 categories, according to the response of the patient.

Importance of pain relief

Relief of pain is one of the great goals of medicine. Prevention is still the key to avoiding the consequences of chronic pain. Acute pain causes autonomic hyperactivity in the form of hypertension, tachycardia, sweating and vasoconstriction, which are deleterious for the critically ill. Adequate pain management often reduces the need for sedation. Multimodal sedation and analgesia (both systemic and regional) must be utilized for ICU patients with adequate monitoring and scoring for optimum results.

Critically ill patients may not be able to communicate pain effectively. Hence it has to be assessed both subjectively and objectively. Pain can occur in ICU in the following situations[20]: intubation, mechanical ventilation, tube suctioning, peripheral neuropathies, cannulation, invasive monitoring, positioning, tracheostomy, sampling, wound dressing, and use of restraints. All these factors need to be addressed in every patient to improve overall outcome.

Methods of analgesia

The first pain clinics were established in the 1940sand□the1960sand□the1960s, multidisciplinary approach to pain management was born. In India, the holy Rig-Veda (written in 4000B.C), describes hundreds of methods deriving from mineral, plant and animal sources, many still in use. The current day pain management is complex and multi-modal. It can be divided into: pharmacological and non-pharmacological methods.

Non-pharmacological methods[20]

Hypnosis, Massage therapy, Music therapy, TENS (trans-cutaneous electrical nerve stimulation), Auriculotherapy and Acupuncture are some of the non-pharmacological methods which are in vogue. But their efficacy is limited and unpredictable. They are usually devoid of any side-effects and repeatable. The ambience of the ICU, its lighting, noise levels, color of the walls/screens and behavior of the staff are equally important in giving a better experience for the critically ill patient.

Pain relief medications

Analgesic agents can be administered via several routes: Enteral/oral, Parenteral (intra-muscular or intravenous), Transdermal/trans-mucosal, Neuraxial (Intrathecal/Epidural), Intra-arterial and Regional techniques. Opioids are the mainstay of analgesia in most ICUs. Pure as well as partial opioid agonists are useful, depending upon the situation and patient profile. Other non-opioid analgesics which are commonly used include: Paracetamol, NSAIDs (non-steroidal anti-inflammatory drug), Ketamine, Clonidine and Dexmeditomidine.

Following are the opioid agents[21] used in ICU and their dosages:

a. Fentanyl Citrate: 25-50 microgram I.V every 30mins to 1 hour; 25-50 microgram/hr

b. Morphine: 2- 4 mg I.V every 1 to 2 hours; 2-4 mg/hour infusion. Its metabolites can accumulate on prolonged infusion and in kidney disease, leading to delayed respiratory depression and weaning failure.

c. Hydromorphone: 0.2 to 0.6 mg I.V every 1-2hours; 0.4 - 0.8 mg/hour infusion.

d. Methadone: 10-40 mg orally every 6-12 hours.

e. Oxymorphone: Intermittent dosing of 0.2 – 0.5mg I.V over 2-5 mins, every 4 hrs.

f. Tramadol: 50- 100mg I.V 8 hourly in adults. It can cause nausea and vomiting.

g. Alfentanil: 5-7.5 microgram/Kg I.V over 3-5min; 0.1- 0.2 microgram/Kg/min infusion.

h. Sufentanil: 0.05 microgram/Kg/Hour I.V infusion

i. Remifentanil: It is unique as it does not accumulate in renal or hepatic failure and has a short half life of 3-10 mins. Its sudden stoppage can lead to hyperalgesia. Loading dose of 0.5 microgram/Kg followed by I.V. infusion of 0.5-1 microgram/kg/hour.

j. Butorphanol: It is an opioid agonist-antagonist, which is much more potent than morphine. It can be given intramuscularly (2mg I.M), intravenously or intra-nasally. It is contraindicated in patients with coronary insufficiency or ventricular dysfunction. Their main advantage the ceiling effect on respiratory depression.

Role of regional anesthesia and blocks

Acute as well as chronic pain is amenable treatment by regional techniques. Trauma and postoperative patients will be in severe pain due to multiple injuries or tissue trauma. Blockade of the various peripheral nerves and plexuses with local anesthetics (lignocaine and bupivacaine hydrochloride) is being practiced widely in surgical and trauma ICUs. Caution needs to be exercised on the dosage of local anesthetic agents used to avoid toxicities. Use of (USG) ultrasound[22] (real time ultrasonography) in accurately locating the nerves and plexuses have improved the success rates and decreased the

complication rates. Continuous brachial or lumbar plexus catheters can be inserted for extremity pain using nerve stimulators and USG-guidance, to facilitate reduction in systemic analgesics. Opioids can also be used for regional blocks and epidural infusions.

Patient-controlled analgesia (PCA)

PCA pumps[23] have revolutionized the field of pain management. It is an electronic programmable device which can be tailored with respect to patient's requirements. Initially nurse-controlled pumps were in vogue, especially for pediatric patients. In PCA, the patient decides when he wants to take a bolus of the analgesic agent. A preset baseline infusion of the analgesic is given and supplemented with top-ups at the press of a button, at pre-set intervals. It gives a greater sense of control to the patient and improves their general well-being. Both parenteral (intravenous PCA pumps) and neuraxial (epidural PCA pumps) are available for use. This of special importance in analgesia for post-operative and post-traumatic critical care patients. Opioids and local anesthetic agents are the most commonly used agents in PCA Tables 1 & 2.

Table 1 Ramsay sedation scale

Sedation Level	Description Level
1	Anxious, Agitated
2	Cooperative, oriented, tranquil
3	Responds only to verbal commands
4	Asleep with brisk response to light stimulation or loud auditory stimulus
5	Asleep without response to light stimulation (glabellar tap)
6	Non-responsive

Table 2 Richmond Agitation Sedation Scale (RAAS)

Target RAAS	RAAS description
4	Combative, violent, danger to staff
3	Pulls or removes tubes and catheters, aggressive
2	Frequent non-purposeful movements, fights ventilator
1	Anxious, apprehensive, but not aggressive
0	Alert & calm
1	Awakens to voice (eye opening/contact>10s
2	Light sedation, brief awakening to voice, eye opening/contact < 10s
3	Moderate sedation, movement or eye opening. No eye contact
4	Deep sedation, no response to voice, but movement or eye opening to physical stimulation
5	Un-arousable, no response to voice or physical stimulation

Interventional pain management

Interventional pain management[24] is a relatively recent feather in the cap of critical care pain management. It includes procedures which are done for intractable, resistant pain, when conventional techniques have failed. Epiduroscopy, Radiofrequency neuro-ablation, Intra-thecal pumps and Vertebroplasty have been used in various chronic pain states (like CRPS), failed back syndromes and spasticity. Spinal cord stimulators can be used in some neuropathic pain states. Most of these require the use of radiographic image for insertion and are usually done under sterile conditions and sedation in the operation theatre. Cost, equipment availability, expertise and long-term follow-up are the chief considerations in this field.

Pitfalls

Patient safety is more important than the choice of sedative-analgesic agent in any ICU. But inadequate therapy can be deleterious in more ways than one. Critically ill patients may have dysfunction of one or more organ systems. Liver or kidney function derangement can cause the primary drug or its metabolite to accumulate in the body. This can lead to delayed drug toxicity or prolonged effects, which can significantly delay weaning. Any drug which produces active metabolites in the body can lead to such complications. Proper assessment of the level of sedation required and achieved is important in deciding about the duration of ICU stay of patients. Similarly, the severity, site and nature of pain may not be accurately measured in pediatric, elderly, neurosurgical and psychiatric patients. This can lead to faulty therapy and increase in morbidity.

Conclusion

Sedation is incomplete without analgesia and vice-versa. Not only the correct agent needs to be chosen, but also its dose, route, side-effect profile and duration of dosing needs to be understood for all patients. Any error at any stage of assessment or treatment can have far-reaching implications. A combined, multi-modal sedative-analgesic regime may be preferable. The Society for Critical Care Medicine (SCCM) frequently reviews and updates its recommendations on ICU sedation and analgesia, which can be followed for better results. Nurse anesthetists and paramedical staff play a pivotal role in helping the intensivist attain a balanced sedation-analgesic regime in critically-ill patients.

Clinical pearls

All ICU staff must remember the pneumonic "ABCDE".[25], as vital steps of critical care. 'A' stands for Awakening, 'B' for Breathing, 'C' for Choice of sedative and analgesic, 'D' for Delirium monitoring and 'E' for Early mobilization. It is an evidence based bundle shown to decrease morbidity and mortality in critical care units.

Conflicts of interest

There is no conflict of interest.

Acknowledgements

None.

Funding

None.

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