Sedation and delirium in the intensive care unit

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Abstract

Sedation is a widely used technique in critical care, and delirium is a common and serious complication of hospital stay. An understanding of drug choice and administration for safe use of sedation is important. Equally, an understanding of the mechanisms of delirium, along with preventative factors and treatments is an essential part of caring for many patients admitted to hospital. This article will cover these aspects of the subject.

Keywords CAM-ICU; delirium; RASS; sedation

Introduction

It is the responsibility of all healthcare professionals to understand and appreciate the principles underlying therapeutic sedation and the problems caused by delirium in ICU. With 45% of ICU patients in the UK under the care of surgeons, the importance of keeping up to date in this area is evident.

This article will outline current practices in sedation and delirium management on the intensive care unit.

Sedation

Sedation in critically ill patients on intensive care differs from that in other hospital environments due to the underlying physiology of the ICU patient making it more hazardous, thereby requiring additional resources.

Definitions

It is important to understand the difference between sedation and general anaesthesia.

Sedation is defined as:

> A technique in which a drug or drugs produce depression of the CNS, enabling treatment to be carried out without physical or psychological stress, but during which verbal contact with the patient is maintained.[1]

This is in contrast to general anaesthesia which is defined as:

> A drug induced loss of consciousness during which the patient is not rousable even by painful stimulation.[1]

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Reasons for sedation in ICU

Critically ill patients are often sedated on the intensive care unit to:

- allow tight control of physiological parameters such as in the patient with an acute head injury
- facilitate mechanical ventilation
- minimize physical discomfort and anxiety
- facilitate safe completion of procedures.

Drugs used for sedation

Drugs used to sedate patients can be divided into sedatives and analgesics. A sedative produces depression of the CNS whereas an analgesic provides pain relief. These two effects should be considered separately when considering which drugs to use for individual patients.

First line drugs: In the UK, sedation in intensive care is most commonly administered by intravenous infusion.[2] This provides a smooth pharmacological profile, avoiding peaks and troughs in drug concentration, and therefore optimize sedation levels. The common drugs used for sedation and analgesia by continuous infusion are shown in Table 1.[1,3] The half-life of the drug is important when considering how long it will take the patient to wake-up after cessation of the infusion. It is important to consider the pharmacokinetics of a drug used in ICU as the patients often have renal or hepatic failure, which can lead to accumulation of sedative and analgesic drugs.

Second line drugs:

Barbiturates

- **Thiopentone** infusions may be used to treat patients with raised intracranial pressure as it reduces cerebral metabolic rate and controls seizure activity. The main disadvantage is that it has a long-half life and can take several days to wear off once the infusion has been stopped.

α_2-agonists

- **Clonidine** is a centrally acting drug with antihypertensive, analgesic and sedative properties. It is useful when weaning patients from opiate or benzodiazepine infusions but accumulates in renal failure. Rapid withdrawal may result in rebound hypertension and tachycardia.
- **Dexmedetomidine** is another centrally acting drug. It produces sedation and analgesia. Dexmedetomidine is shorter acting than clonidine. The major side effect seen is bradycardia. The main advantages of dexmedetomidine the lack of respiratory depression and the blunting of the stress response. Although this drug has been used in the USA for many years it is yet to become widely available in the UK.

Sedation holds

Traditionally patients were sedated for the duration of their mechanical ventilation and only 'woken up' once it was deemed they no longer required a high level of physiological support. More recent evidence in the literature has shown daily interruption of sedation (sedation hold) reduces the duration of mechanical ventilation without increasing the risk of adverse events.

Benefits

- Optimize the dose of sedative drug being used.
- Reduce the requirement for cardiovascular support.

Drugs commonly used for sedation in ICU as intravenous infusions

Drug	Half-life	Advantages	Disadvantages
γ-aminobutyric acid (GABA) agonists			
Propofol *Anaesthetic induction agent*	30—90 minutes	Speed of onset of action and elimination Does not accumulate	Hypotension Depression of laryngeal reflexes Hyperlipidaemia Propofol infusion syndrome (severe metabolic acidosis and muscle necrosis)
Midazolam *Benzodiazepine*	3—11 hours	Speed of wake-up following cessation of infusion compared to other benzodiazepines	Respiratory depression Hypotension Dependence Accumulation due to active metabolite Risk factor for delirium
Opiod receptor agonists			
Morphine *Opiod*	3—7 hours	Inexpensive Good safety profile	Respiratory depression Gastro-intestinal ileus Histamine release Pruritus Accumulation due to active metabolite Risk factor for delirium
Fentanyl *Opiate*	1.5—6 hours	Less accumulation in renal failure compared to morphine	Respiratory depression Bradycardia Muscle rigidity
Remifentanil *Opiate*	3—4 minutes	No accumulation in renal or hepatic failure Wake-up time is constant irrespective of the duration of infusion	Respiratory depression Bradycardia Hypotension

Table 1

- Assess neurological function.
- Facilitate communication with the patient.
- Reduce the incidence of post-traumatic stress disorder.

Concerns

- Adverse events such as accidental extubation, line removal.
- Patient suffers an increased level of anxiety and pain.

The 'wake up and breathe' trial demonstrated that a paired interruption of sedatives protocol and spontaneous breathing trial leads to an improved 1-year survival and for every seven patients treated with the intervention, one life was saved.[4] The length of ICU stay was significantly reduced and patients were more likely to be discharged home.[5]

Given the strength of this evidence, many hospitals now have sedation guidelines to aid nursing and medical staff, and all clinicians with any ICU involvement should have a working knowledge of these.

Measurement of sedation

Titrating sedatives to achieve the required level of sedation with the minimum amount of drug reduces the duration of mechanical ventilation and decreases the duration of ICU stay. In order to titrate drug doses, the depth of sedation must be assessed.

This is now a routine part of ICU monitoring and is documented by the nursing staff along with other physiological parameters. A variety of assessment tools are available.

Qualitative measurement: The most common method of assessing sedation is to use a clinical scoring system. The Richmond Agitation-Sedation Scale (RASS) is a simple bedside scoring system that can be used by the nursing staff to monitor patient sedation levels and adjust infusions accordingly (Table 2). A RASS score of between −2 and +1 is optimal for the patient on ICU.[6]

Quantitative measurement:

- *Electroencephalogram* (*EEG*) — the EEG measures the electrical activity of the brain and the waveforms seen are interpreted by a neurophysiologist. Due to the specialist personnel and equipment it is rarely used in ICU to monitor sedation levels.
- *Bispectral index* (*BIS*) *monitor* — the BIS monitor is a crude form of the EEG. It measures the electrical activity in the brain and displays this activity as a number between 1 and 100. One is EEG silence and 100 is fully awake. A BIS score of 60—80 indicates a lightly sedated patient. BIS may be used in ICU, but limiting factors include uncertainties regarding the clinical correlation between raw BIS score and level of sedation, along with equipment availability and staff training.

Measurement of pain

Unrelieved pain causes a stress response comprising tachycardia, increased myocardial oxygen demand and persistent