

Sleep quality in intensive care unit: Are we doing our best for our patients?

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All critically ill patients admitted to intensive care units (ICU) sleep poorly. This conclusion is evident after analyzing a number of surveys in surviving patients after discharge from ICU. In frequency, sleep deprivation as a symptom is the second grievance, after pain.

Intensivists overestimate the amount of sleep in our patients. Nevertheless, we must remember that sleep characteristics are quite peculiar: Fragmented, inefficient, superficial (difficulty in reaching rapid eye movement [REM] sleep), with long sleep onset and increased arousals. In most severe cases, it seems that sleep disruption is even greater, and this will lead to serious consequences.

Despite this, after decades of study, the consequences of sleep deprivation have not been detailed and even worse, universal clinical protocols or guidelines to prevent it have not been established. This being unacceptable, we should act.

Causes of Sleep Deprivation

Most of the causes of sleep deprivation in critically ill patients are environmental and potentially modifiable.^[1] The most common cause is noise. The World Health Organization states that noise levels at the hospital should not exceed 40 dB. There have been several studies analyzing the noise levels of the ICU, with no clear result.^[2]

A patient admitted to an ICU box is usually surrounded by machines, with very noisy alarms, and everything becomes more pronounced at night. However, this is not the most common cause of complaint of patients,

but the voices of healthcare personnel. These voices are obviously louder when the admission of a patient in ICU is performed, and can make patients already admitted not to sleep that night. In addition to noise is the problem of lighting.

In patients not analgosedated, other major causes of sleep deprivation tests conducted during the night (blood tests, X-rays, ultrasounds...), taking vital signs and certain treatments (benzodiazepines, antipsychotics, and opioids decrease REM sleep).

Patients with mechanical ventilation and weaning process are subjected to multiple interventions that cause stress and anxiety: Discomfort with endotracheal or tracheostomy tubes, waking up unable to speak or move in bed, several awakenings, inability to sleep and depending on the mode of ventilation used, more or less dyssynchrony.

Consequences

Sleep deprivation subjectively affects the quality of life of critically ill patients during their admission to the ICU, hospitalization, and for a variable time after discharge. There have only been a few studies published on this subject, describing deleterious effects on most organ systems, causing increased morbidity, but not to increased mortality.

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Sedation of critically ill patients with midazolam or propofol differs from natural sleep in maintaining noradrenaline release by the locus coeruleus, and this predisposes to increased stress and catabolic state. Dexmedetomidine is an adrenergic alpha-2 agonist that acts as a sedative and anxiolytic drug and has shown that it doesn't maintain noradrenaline secretion. Actually, this drug is only indicated in patients with mechanical ventilation, but its mechanism of action ("conscious sedation"), is promising.^[3]

Other consequences of induced sleep are altered electroencephalographic cyclical progression, altered circadian rhythm and a different reversibility (in natural sleep is immediately and completely reversible to an external stimulus).^[4]

Possible Solutions

Obviously, quality and quantity of sleep are not the same. There have been several studies in critically ill patients where investigators could improve sleep quantity, but not quality. Gabor *et al.*^[4] performed a polysomnographic comparison of patients admitted to the ICU with their cabin doors open or closed. Patients with closed doors had more quantity of sleep, but sleep quality was the same in both groups.

We should make all protocols in ICUs include environmental, technical, and pharmacological interventions.

Environmental

Control of noise and light exposure, use of earplugs, music therapy, keep doors closed, and preferably cabins with light schedules similar to the circadian rhythm (which would work even in reducing delirium).

Technical

Taking blood samples at night only if strictly necessary. Spacing noninvasive measures of blood pressure and diuresis appropriately. Employing mechanical ventilation modalities that are suitable to the waking state of the patient. Standardizing the weaning process would be nice, starting early in the morning and in case

of weaning failure mildly sedate at night to respect their circadian rhythm and to avoid undesirable awakenings.

Pharmacological

We have to treat the underlying medical cause of lack of sleep onset with analgesic, anxiolytic and antipsychotic drugs at optimal doses. For patients with spontaneous ventilation, preferably employ short half-life benzodiazepines and only if strictly necessary. In these cases, there are no studies comparing the effectiveness of oral benzodiazepines with intravenous infusions of propofol, midazolam, haloperidol or dexmedetomidine. For the mechanisms of action they have, it seems logical that short-life benzodiazepines provide optimal sleep onset, but not its maintenance; something that could be obtained with other sedative infusions at optimal doses.

Conclusions

Sleep is an important factor during the process of recovery of critically ill patients, as a part of an integrated homeostatic mechanism. It remains to show that sleep deprivation is related to decreased survival of critically ill patients. However, something that is fully demonstrated is the high morbidity generated. We need active investigation in this area. If we have the tools to prevent it, why not use them?

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