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Sleep in the intensive care unit

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ABSTRACT

Poor sleep quality is a consistently reported by patients in the ICU. In such a potentially hostile environment, sleep is extremely fragmented and sleep architecture is unconventional, with a predominance of superficial sleep stages and a limited amount of time spent in the restorative stages. Among the causes of sleep disruption in the ICU are factors intrinsic to the patients and the acute nature of their condition, as well as factors related to the ICU environment and the treatments administered, such as mechanical ventilation and drug therapy. Although the consequences of poor sleep quality for the recovery of ICU patients remain unknown, it seems to influence the immune, metabolic, cardiovascular, respiratory, and neurological systems. There is evidence that multifaceted interventions focused on minimizing nocturnal sleep disruptions improve sleep quality in ICU patients. In this article, we review the literature regarding normal sleep and sleep in the ICU. We also analyze sleep assessment methods; the causes of poor sleep quality and its potential implications for the recovery process of critically ill patients; and strategies for sleep promotion.

Keywords: Sleep; Sleep deprivation; Intensive care units.

INTRODUCTION

The ICU is a highly complex environment, the operation of which is traditionally based on constant monitoring and observation. As a result, physical and technical aspects of patient care are given priority, whereas some humanizing aspects of care can be overlooked. The sleep of critically ill patients is a subject of increasing interest in the literature, and there is evidence that sleep in the ICU is poor in quality.[1-3] Although there are gaps in the knowledge of this subject, acute sleep deprivation can be negatively associated with the recovery of ICU patients.[1-3]

This article provides a review of the literature regarding the main physiological aspects of normal sleep and the current knowledge of sleep in critically ill patients.

NORMAL SLEEP

Sleep can be defined as a periodic, reversible state of disengagement from the environment.[4] It consists of an active process that involves multiple, complex physiological and behavioral mechanisms of the central nervous system (CNS). Sleep is essential for rest, for repair, and for the survival of the individual.[2]

Normal sleep is divided into two states: rapid eye movement (REM) and non-rapid eye movement (NREM).[5] Accounting for approximately 25% of total sleep time (TST), REM sleep is characterized by rapid, low-amplitude brain activity; episodes of rapid eye movement; irregular respiratory and heart rates; and atonia or hypotonia of major muscle groups. REM sleep is a restorative stage with a variable arousal threshold. It is during this stage that dreams occur.[4,5]

NREM sleep is divided into three stages (1, 2, and 3).[4] Transition from stage 1 to stage 3 refers to a progressive increase in slow waves on electroencephalography (EEG), an increase in sleep depth, and a progressive increase in the arousal threshold. Therefore, stage 3 is known to be the deepest, most restful sleep stage and to have the highest arousal threshold, as well as playing an important role in restorative processes, such as memory consolidation. In contrast, an increase in the amount of stage 1 sleep usually suggests sleep fragmentation caused by sleep disturbance.[4]

In a normal individual, NREM and REM sleep alternate cyclically throughout the night. The NREM-REM cycle repeats itself every 90-110 min, 5-6 times per night. Typically, NREM sleep predominates in the first part of the night, and REM sleep predominates in the second.[4] However, the distribution of sleep stages across the night can be affected by several factors, including age, circadian rhythm, ambient temperature, drugs, and certain diseases.

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Sleep is regulated by circadian and homeostatic mechanisms. The circadian rhythm, an approximately 24-h cycle upon which the life cycle of almost all living beings is based, is regulated by a biological clock located in the suprachiasmatic nucleus of the hypothalamus and helps set the sleep-wake cycle. The sleep-wake cycle tends to be synchronized with the 24-h cycle through environmental stimuli and, predominantly, by exposure to light. The sleep-wake cycle can easily be disrupted in an environment where there is no light/dark cycle. The secretion of melatonin, a hormone involved in the regulation of the sleep-wake cycle, is regulated by the circadian cycle. In order to promote nighttime sleep, the secretion of melatonin is maximal at night, when there is no light. Homeostatic mechanisms also affect the sleep-wake cycle, and their functioning is similar to that of the thirst mechanism: longer periods of sleeplessness translate to greater sleepiness. This underscores the need for sleep, regardless of environmental stimuli.[6]

SLEEP ASSESSMENT METHODS

Overnight in-laboratory polysomnography (PSG) is the gold standard method for diagnosing sleep disorders.[7] The method allows the polygraphic recording of EEG, electrooculogram (EOG), chin and limb electromyogram, oronasal airflow, thoracoabdominal movement, electrocardiogram, and pulse oximetry. Additional channels are available to record other parameters, such as body position, esophageal pressure, snoring, and additional EEG derivations.

Staging of sleep is based on brain wave patterns, chin muscle activity, and EOG. These variables are analyzed in 30-s time segments, known as "epochs".

Portable systems encompass a range of sleep-assessment devices with different levels of complexity. These systems require less technical skill and result in lower costs than does traditional PSG, thus facilitating the diffusion of sleep assessment. They are mainly used in the diagnosis and monitoring of obstructive sleep apnea syndrome (OSAS).[8]

The American Academy of Sleep Medicine divides sleep assessment methods into four categories, on the basis of their respective level of resolution.[8]

Type 1—standard PSG: recording using a minimum of seven channels, including EEG, EOG, submental electromyogram, electrocardiogram, oronasal airflow, respiratory movement, and oxyhemoglobin saturation. It is performed in an attended laboratory setting.

Type 2—portable PSG: recording using a minimum of seven physiological channels, as in standard PSG. It is usually performed in an unattended home setting.

Type 3—modified portable OSAS testing: recording of at least four channels. Because only cardiorespiratory variables are evaluated, it is not possible to analyze sleep parameters.

Type 4—portable, single-channel recording with oximetry, with or without heart rate monitoring. Sleep parameters are not analyzed.

SLEEP ASSESSMENT METHODS IN THE ICU

Although PSG is considered the gold standard method for evaluating sleep,[7] the cost of performing PSG, as well as the practical difficulties in performing it, has led researchers to adopt other sleep assessment methods in critically ill patients.[1,9]

In the setting of critically ill patients, surrogate methods, such as actigraphy (with an activity monitor) and calculation of the bispectral index, have been used.

An activity monitor is an accelerometer-based sensor that is similar to a wristwatch and measures the level of physical activity. This sensor distinguishes between periods of sleep and wakefulness, on the basis of body movement. Although a high level of agreement has been reported between actigraphy and PSG in evaluating sleep in healthy individuals,[10] the only study comparing the use of actigraphy with that of PSG in critically ill patients showed disappointing results.[11] There were no significant correlations between the two methods in terms of TST, sleep efficiency, or the number of arousals. The explanation given by the authors for the low sensitivity and specificity of actigraphy was that the level of immobility was high in that population, who remained in bed throughout the recording period, with few changes in body position.[11]

The bispectral index is a neurophysiological measure that is primarily used to monitor the level of sedation during anesthetic procedures. The bispectral index is used in order to analyze EEG patterns continuously, providing a numerical value on a scale of 0 to 100. Higher values indicate higher levels of consciousness. Unlike actigraphy, the monitoring of the bispectral index makes it possible to assess sleep depth (although an overlap of values for a given stage can lead to an inaccurate characterization of sleep architecture).[9] Reported difficulties in its use include electrode detachment and motion artifacts.[9] Although the bispectral index could prove to be a promising tool in evaluating sleep in critically ill patients, its benefits in this setting have yet to be established.[2,9]

Subjective instruments have been used in order to evaluate sleep in critically ill patients. In comparison with studies using PSG, those using subjective sleep assessment methods have evaluated larger numbers of patients and interventions, over longer periods. In practice, subjective methods are the only possible means of measuring the efficacy of interventions.[9]

Among the existing subjective sleep assessment methods, the most widely used[2] is the Richards-Campbell Sleep Questionnaire (RCSQ).[12] The RCSQ was validated against PSG in a study of 70 ICU patients, and a moderate correlation was found.[12] The RCSQ evaluates sleep in terms of five dimensions: sleep depth; sleep latency; sleep fragmentation; time to resumption of sleep; and sleep quality. Responses are recorded on a 100-mm visual analog scale, and higher scores indicate better sleep quality. The use of the RCSQ in the ICU can be limited by the presence of sedated patients or patients with delirium, which

can reduce the patient sample by up to 50%.[13] In an attempt to increase the applicability of the RCSQ, a study evaluating agreement between nurse-completed RCSQs and patient-completed RCSQs obtained a correlation that was only mild to moderate, with nurses tending to overestimate patient-perceived quality of sleep.[14]

The Sleep in the Intensive Care Unit Questionnaire[15] is a 27-item instrument that evaluates sleep in terms of four dimensions: sleep quality; disruptive factors produced by the health care team; environmental disruptive factors; and daytime sleepiness. Its usefulness lies in the fact that it allows individual scoring of the role of a number of sleep disruptions resulting from ICU environmental factors or from care routines. Therefore, it has been used in studies implementing protocols for sleep promotion.[16-18]

SLEEP IN THE ICU

Poor sleep quality is consistently reported by patients in the ICU.[1-3,15,19-22] In a study employing 24-h PSG to evaluate 57 ICU patients, sleep architecture was reported to be highly altered. Those patients spent 90% of their TST in superficial sleep (NREM stages 1 and 2), thus being mostly deprived of deep and restorative sleep (NREM stage 3 and REM sleep, respectively).[21] In that population, TST was 5 h, and 41% of TST occurred during the day,[21] which indicates impairment of the circadian rhythm of sleep. In addition, unusual sleep stage transitions, as well as a high frequency of nighttime arousals (mean, 27 events/h), were noted. Similarly, another study demonstrated that critically ill patients experienced 41 ± 28 sleep periods in 24 h, with sleep periods lasting approximately 15 ± 9 min, which means that the process was highly fragmented.[23]

Subjective assessments of sleep in critically ill patients have yielded similar results. One study reported that sleep in critically ill patients was light and was often disrupted by arousals, and that, once awake, patients had difficulty resuming sleep.[24] In another study, most of the patients evaluated rated their sleep quality as poor, the mean RCSQ score being 57.50 points (range, 32.00-70.00).[21]

Patients who are eventually discharged from the ICU (ICU survivors) report that inability to sleep was among the major sources of stress[25] or bad memories during their ICU stay.[26] In one study, 60 patients were interviewed by at 6-12 months after their discharge from the ICU, and 50% reported sleep disturbances during their ICU stay, such disturbances persisting after discharge in approximately 30%.[27]

Such findings demonstrate that there has been no improvement in sleep patterns since similar studies were published more than 10 years ago.[15,19,20] For instance, in 1985, sleep assessment in 9 patients in the postoperative period of non-cardiac surgery revealed that all of those patients had severe or complete suppression of REM sleep and NREM stage 3 sleep.[19] In 1999, it was demonstrated that sleep quality in the ICU was perceived as significantly poorer than sleep at home by all patients interviewed ($p < 0.0001$).[15]

In summary, studies have revealed qualitative and quantitative sleep deficiencies in critically ill patients. The presence of extreme sleep fragmentation and unconventional sleep architecture is indisputable, as are the predominance of light stages and the lack of restorative stages.[1-3,22,28]

Causes of sleep disorders in the ICU

Several factors are related to sleep deprivation in critically ill patients. These factors include environmental factors, such as noise, light, and care activities; factors intrinsic to the patients and the acute nature of their condition; and factors related to the treatments administered, such as ventilatory support and drug therapy.[1,2,22,28] Despite the identification of these factors, the exact role played by each of them in sleep in critically ill patients remains unknown.

Noise

Environmental noise has been reported as the major sleep-disturbing factor,[21,29] the principal sources of such noise being talking by the staff, monitor alarms, infusion pump alarms, telephones, and television.[15,30]

Noise levels in the ICU are estimated to range from 50 to 75 dB, with peaks of 85 dB,[31] whereas the U.S. Environmental Protection Agency recommends that hospital noise levels do not exceed 45 dB during the day and 35 dB during the night.[32] The estimated ICU noise levels are comparable to those reported for factories (80 dB) and busy offices (70 dB).[31] Only 10-30% of arousals can be attributed to environmental noise,[23,29] which belies the traditional hypothesis that noise is one of the major sleep-disrupting factors in the ICU. One study reported that although noise peaks occur frequently in the ICU, only 12% of those peaks result in an arousal.[29]

Light

One ICU study recorded activity occurring while lights were turned on during the night. The activity associated with the greatest amount of light exposure was that related to obtaining samples for laboratory tests, followed by "no activity", suggesting a lack of vigilance on the part of the health care provider team in reducing unnecessary light exposure.[33] However, patients have reported that light is less disturbing to their sleep than are care activities or environmental noise.[15,30]

Given that light plays a vital role in the synchronization of the circadian rhythm, one study evaluated nighttime secretion of melatonin in ICU patients. That study found that, regardless of the levels of light, melatonin secretion was suppressed or erratic, suggesting that factors other than the light/dark cycle affect the circadian rhythm in ICU populations.[34]

Patient care activities

One study found that nursing care activities, such as oral and ocular hygiene, bathing, changing of bed linen,

and catheter management, were usually performed between midnight and 5:00 a.m.,[35] resulting in a mean of 51 interventions per patient per night.[35] In addition, a study evaluating nursing care on the night shift reported that only 9 uninterrupted periods (2-3 h each) were available for sleep on 6% of the 147 nights studied.[36] However, another study demonstrated that only 20% of patient care activities resulted in arousals, which accounted for approximately 7% of the sleep disruption in that patient population.[29] Therefore, although frequent, care activities do not seem to be the major source of sleep disturbances in ICU patients.[1]

Factors intrinsic to the patients

Patients admitted to ICUs can have a preexisting disease that contributes to poor sleep quality. Obstructive pulmonary diseases, such as asthma and COPD, are common comorbidities and can be associated with sleep fragmentation and poor sleep efficiency, as well as with changes in sleep architecture.[2] Patients with neurological disturbances or severe systolic heart failure often exhibit nocturnal Cheyne-Stokes respiration, which can cause sleep fragmentation, excessive daytime sleepiness, paroxysmal nocturnal dyspnea, and insomnia.[2] Sleep-disordered breathing conditions, such as obstructive sleep apnea and obesity hypoventilation syndrome, can lead to serious consequences if not properly treated.[2] In addition, the acute injury responsible for the ICU admission can itself be a sleep-disrupting factor. In the immediate postoperative period after major surgery, deep sleep (NREM stage 3 and REM sleep) is reduced or absent. This finding is characteristically followed by a REM sleep rebound.[19] However, this rebound can be absent in patients taking REM-sleep-suppressing drugs, such as analgesics and benzodiazepines. Furthermore, studies investigating the profiles of patients admitted to ICUs have demonstrated that changes in melatonin secretion are more marked in ICU patients than in their healthy counterparts, a finding that suggests poorer sleep quality in the former group.[37,38]

Patient-related conditions can contribute to poor sleep quality. Pain is a common complaint of patients and can be associated with poor sleep quality.[25] Stress and anxiety due to unfamiliarity with the ICU environment, inability to speak or move, or acute illness are other factors that should be taken into account.

Mechanical ventilation

Studies have shown that mechanical ventilation (MV) is associated with sleep disorders.[20,23] Aspects of MV that contribute to sleep fragmentation include increased work of breathing, gas exchange abnormalities, and patient-ventilator asynchrony.[1,2] Therefore, patients on MV have greater sleep fragmentation and lower sleep efficiency than do their non-ventilated counterparts.[20] Other related factors, such as discomfort from the endotracheal tube, aspiration, frequent repositioning, and ventilator alarms, are also likely to contribute to poor sleep quality, although such associations have yet to be studied.[2,31] It should be borne in mind that, in patients on MV, disease severity is a potential confounding factor, as are the use of sedatives and analgesics.[31]

There is evidence that the mode of ventilation also affects sleep quality.[39,40] One study reported that sleep fragmentation was greater during pressure support ventilation (PSV) than during assist-control ventilation: 79 vs. 54 arousals and microarousals/h.[39] Another study demonstrated that patients receiving neurally adjusted ventilatory assist had a higher proportion of REM sleep than did those receiving PSV—16.5% (range, 13-29%) vs. 4.5% (range, 3-11%; $p = 0.001$)—as well as less sleep fragmentation—16 ± 9 vs. 40 ± 20 arousals and microarousals/h; $p = 0.001$).[40] However, a study comparing the impact of three modes of ventilation (assist-control, PSV, and SmartCare™) on sleep quality in conscious, unsedated patients reported conflicting results. There were no differences among the three modes in terms of sleep architecture, sleep fragmentation, or sleep duration.[41]

Medications

A significant number of medications that are commonly used in the ICU can cause changes in the amount and quality of sleep. These medications can affect the CNS either directly, by penetrating the blood-brain barrier, or indirectly, by interfering with a medical or psychiatric condition, which results in altered sleep. In addition, they can have an equally disturbing effect when withdrawn abruptly.[42] Although it is difficult to study the exact interaction between these medications and sleep in critically ill patients, their effects in healthy individuals are well documented.[42,43]

Sedatives

Benzodiazepines improve sleep efficiency because they decrease sleep latency and the number of arousals, thus increasing TST. However, the chronic use of benzodiazepines is associated with superficial sleep, because it reduces deep sleep and REM sleep. The abrupt withdrawal of benzodiazepines is associated with rebound insomnia.[42] Propofol, which is used primarily for deep sedation, suppresses REM sleep and is associated with poor sleep quality in ICU populations.[1,2] The use of propofol (even at low doses) is associated with delirium in critically ill patients, as is that of benzodiazepines.[42] Dexmedetomidine, a new α_2 agonist, has sedative, anxiolytic, and analgesic effects, with minimal respiratory depression. Similarities between natural sleep and dexmedetomidine-induced sedation have been reported.[1] However, further studies are needed in order to determine the specific effects of dexmedetomidine on sleep in critically ill patients.

Antipsychotic drugs

Antipsychotic drugs are now the pharmacological mainstay of the management of agitation and delirium in the ICU. Haloperidol, the most widely used atypical antipsychotic drug, when administered as a single dose to healthy volunteers, tends to increase sleep

efficiency, especially that of NREM stage 2 sleep, with little effect on slow-wave sleep.[44] Olanzapine and risperidone seem to increase sleep efficiency and TST, as well as increasing deep sleep.[44]

Analgesics

Opioids are the mainstay of the treatment of pain and discomfort in critically ill patients. Opioids are associated with suppression of REM and slow-wave sleep, as well as with sleep fragmentation, and can induce central apnea or even delirium.[1,2] Even nonsteroidal anti-inflammatory drugs can negatively affect sleep, increasing nighttime arousals and decreasing sleep efficiency.[1] However, analgesic medications play a significant role in patient comfort, and a balanced administration of these medications should be sought.

Cardiovascular medications

Beta blockers can have variable effects on sleep, depending on their ability to cross the blood-brain barrier. The use of Beta blockers that are highly lipid soluble is associated with increased sleep disruption, potentially causing nightmares, insomnia, and suppression of REM sleep.[1] Amiodarone has neurological effects, including insomnia and nightmares, in 20-40% of patients.[1] Angiotensin-converting enzyme inhibitors do not seem to affect sleep. Other antihypertensive agents, such as calcium antagonists, hydralazine, diuretics, and α_1 antagonists, have not been evaluated for their effects on sleep.[42] Norepinephrine, epinephrine, and dopamine are associated with insomnia, as well as with suppression of deep sleep and REM sleep.[42]

Respiratory medications

Agitation and insomnia caused by stimulation of the CNS are well-known adverse effects of beta-agonists.[42] However, their effects can ultimately be positive if there is a reduction in the respiratory symptoms that are related to sleep fragmentation.[1,2]

Miscellaneous

Corticosteroids are often associated with insomnia; however, there have not been enough conclusive results.[43] Nevertheless, the use of corticosteroids, depending on the type and dosage, can be associated with suppression of REM sleep and with nighttime arousals.[42,43]

Although tricyclic antidepressants can suppress REM sleep, they increase TST and in general can improve subjective sleep quality. Selective serotonin reuptake inhibitors reduce REM sleep less sharply. However, they decrease TST and can be associated with insomnia, as well as with daytime sleepiness.[42,43]

POTENTIAL CONSEQUENCES OF SLEEP DISTURBANCES IN THE ICU

Cardiovascular consequences

It is well known that chronic sleep deprivation is associated with increased cardiovascular morbidity and mortality. A cohort study conducted in Germany revealed that, among individuals who slept less than 6 h per night, the relative risk (RR) of cardiovascular disease and coronary artery disease was, respectively, 1.11 (95% CI: 0.97-1.27) and 1.19 (95% CI: 1.00-1.40). The risk of those conditions was found to be more than 60% higher among such individuals than among those who slept more than 6 h per night.[45] A systematic review with a collective sample of 474,684 participants revealed that individuals who are chronically sleep deprived are at an increased risk of developing and dying from coronary artery disease (RR = 1.48; 95% CI: 1.22-1.80; $p < 0.0001$) and stroke (RR = 1.15; 95% CI: 1.00-1.31; $p = 0.047$).[46] Despite this evidence, whether sleep deprivation in the ICU contributes to cardiovascular mortality has yet to be established.

Respiratory consequences

Studies conducted outside the ICU setting have demonstrated that even short periods of sleep deprivation can cause respiratory changes. After a sleepless night, healthy individuals show a slight but significant decline in FVC and maximal voluntary ventilation.[47] One study of patients with stable COPD reported similar changes.[48]

Although it used to be believed that sleep deprivation could reduce the ventilatory response to hypercapnia,[47,49] leading to hypoventilation, it has been demonstrated that sleep deprivation does not change respiratory control in healthy individuals.[50] No such studies have been conducted in critically ill patients.

Metabolic consequences

Evidence that sleep has a modulatory effect on the metabolic system has been reported in recent decades. In particular, glucose tolerance, the 24-h pattern of insulin release, and the secretion of counterregulatory hormones (such as growth hormone and cortisol), as well as of those involved in the regulation of appetite (such as leptin and ghrelin), are, at least in part, dependent on sleep duration and quality.[51,52] Such findings, however, are primarily based on epidemiological cohort studies evaluating chronic sleep deprivation or on models of sleep fragmentation in individuals with OSAS, which does not allow extrapolation of the findings to the acute setting of critically ill patients.[51,52]

Consequences for the immune system

It is common sense that sleep deprivation increases the risk of an individual having an infection or disease, and that, conversely, sleep is vital to the recovery of health.[2] A murine model designed to explore the effects of sleep loss on host immunity and defense demonstrated that chronic sleep deprivation led to wasting and death from septicemia, resulting from opportunistic bacterial infections, within 27 days.[53]

Studies of healthy individuals have demonstrated that sleep deprivation leads to changes in the immune functions of lymphocytes, polymorphonuclear cells,

and natural killer cells.[2,28,31] In addition, inflammatory cytokines (such as IL-1, IL-6, and TNF), which are known to cause endothelial dysfunction and increased insulin resistance, are also increased in sleep deprivation,[2,28,31] potentially expanding the physiological impact of sepsis.

Delirium

Delirium is an acute confusional state that is common in critically ill patients, affecting up to 80% of patients receiving MV.[54] Delirium is associated with higher mortality, longer hospital stays and increased hospitalization costs, and cognitive worsening.[2,54] Given that both delirium and sleep deprivation are common findings that often coexist in critically ill patients, it has been hypothesized that the two are related.[2,55,56] However, whether this is a cause-and-effect relationship or simply an association resulting from shared mechanisms remains a matter to be determined. An analysis of 223 critically ill patients demonstrated no association between daily perceived sleep quality and transition to delirium. However, in those patients undergoing MV, the use of sedatives (benzodiazepines or opioids) was strongly associated with a transition to delirium within 24 h.[56]

MEASURES FOR SLEEP PROMOTION IN THE ICU

The mechanisms associated with sleep disorders and abnormal sleep architecture in the ICU have yet to be fully understood. Although factors such as noise and care activities have classically been considered the main causes of sleep disruption in critically ill patients, these factors have been found to account for only 37% of arousals in the ICU.[29] Therefore, addressing these factors individually should not significantly affect sleep deprivation in the ICU.[28]

There is evidence that sleep promotion in the ICU can be achieved through multifaceted interventions focused on multifactorial minimization of nighttime sleep disruptions and maintenance of sleep-wake cycles.[1,3,22,28] Therefore, chief among major approaches are the following: reduction in nighttime light and noise levels; improvement of patient comfort; and organization of care activities to allow uninterrupted periods of sleep.[1,2,28,31] A reduction in noise levels can be achieved by adjusting monitor and ventilator alarms, turning down the telephone ringer volume, closing doors, minimizing staff conversation, and providing earplugs.[16-18,57-60] Light levels can be reduced by dimming the lights in the rooms and surroundings and by providing sleeping masks.[16-18,57-60] Improvement of patient comfort includes ventilator adjustment, optimizing patient-ventilator synchrony[39,40]; adequate pain relief[17,18,57,58]; relaxation techniques, such as massage, music therapy, and playing recordings of ocean sounds[1,2,60]; and administration of drugs, such as zolpidem, haloperidol,[18] or melatonin,[1,22,59] when necessary. Care activities, such as exams and blood collection, hygiene care, and administration of medications, should be planned to prevent unnecessary sleep disruptions.[16-18,57,58]

Studies have been carried out to assess the impact that such multifaceted interventions have on sleep promotion. A protocol was developed to limit nighttime nursing care activities and thereby reduce patient sleep disruptions. There was no significant improvement in the intervention group. However, patients in that group were older and reported more frequent use of sleeping medication than did those in the control group.[58] A similar study implemented a protocol to reduce nighttime light and noise levels, in addition to changing patient care routines. That study demonstrated that mean noise levels were significantly reduced, as was the noise perceived by the patient. There was also a reduction in sleep disruption from environmental factors in the intervention group, as well as improvement in global sleep quality and in sleep efficiency.[16] In contrast, by implementing measures to promote sleep, another group of authors achieved a reduction in nighttime light and noise levels. Consequently, the sleep efficiency index was better in the intervention group than in the control group and the number of arousals associated with the health care team was lower in the intervention group. The incidence of delirium was also lower in the intervention group.[17] A randomized clinical trial that implemented measures to reduce environmental disruptions, as well as the use of music therapy,[60] achieved significant improvements in sleep, as assessed by the different domains of the RCSQ, in the intervention group. However, there were no differences between the intervention and control groups in terms of urinary cortisol or melatonin.

A study that, in addition to the conventional protocol, implemented pharmacological measures to promote sleep achieved significant improvements in perceived nighttime noise and incidence of delirium. There was no difference in perceived sleep quality.[18] A randomized trial that also used pharmacological measures (melatonin)[59] could not adequately compare PSG findings between the two groups, because more than half of the recordings were unscorable (i.e., showed sleep patterns that could not be interpreted).

FINAL CONSIDERATIONS

Sleep in critically ill patients is characterized by frequent disruptions, changes in the circadian rhythm, and poor quality, along with a reduction in the deep, restorative stages.

Such sleep disturbances seem to be due to factors related to the ICU itself, such as care routines and environmental stimuli; factors intrinsic to the patients and the acute nature of their condition; and factors related to the treatments administered, such as MV. However, understanding of the pathogenesis of sleep disturbances in ICU populations remains incomplete, and there is no knowledge of the relative contribution of potential sources of sleep disruption. In addition, although poor sleep quality can affect a number of

metabolic and regulatory processes in the body, the impact of sleep deprivation on certain outcomes, such as weaning from MV, length of ICU stay, and in-hospital morbidity and mortality, remains unknown.

Finally, although protocols for sleep promotion in the ICU have recently been implemented and studied, the degree to which sleep can be improved in ICU patients, as well as the best strategies for sleep promotion, has yet to be defined. While all such questions remain unanswered, it seems appropriate to provide patients with the necessary conditions for restorative sleep if the goal can be achieved safely.

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