

Improving Outcomes by Optimizing Sleep Opportunities in the Critically Ill Neuroscience Patient

Marie Wilson, MSN, RN, CRNP, CCRN, CNRN

Thomas Jefferson University Hospital

Purpose

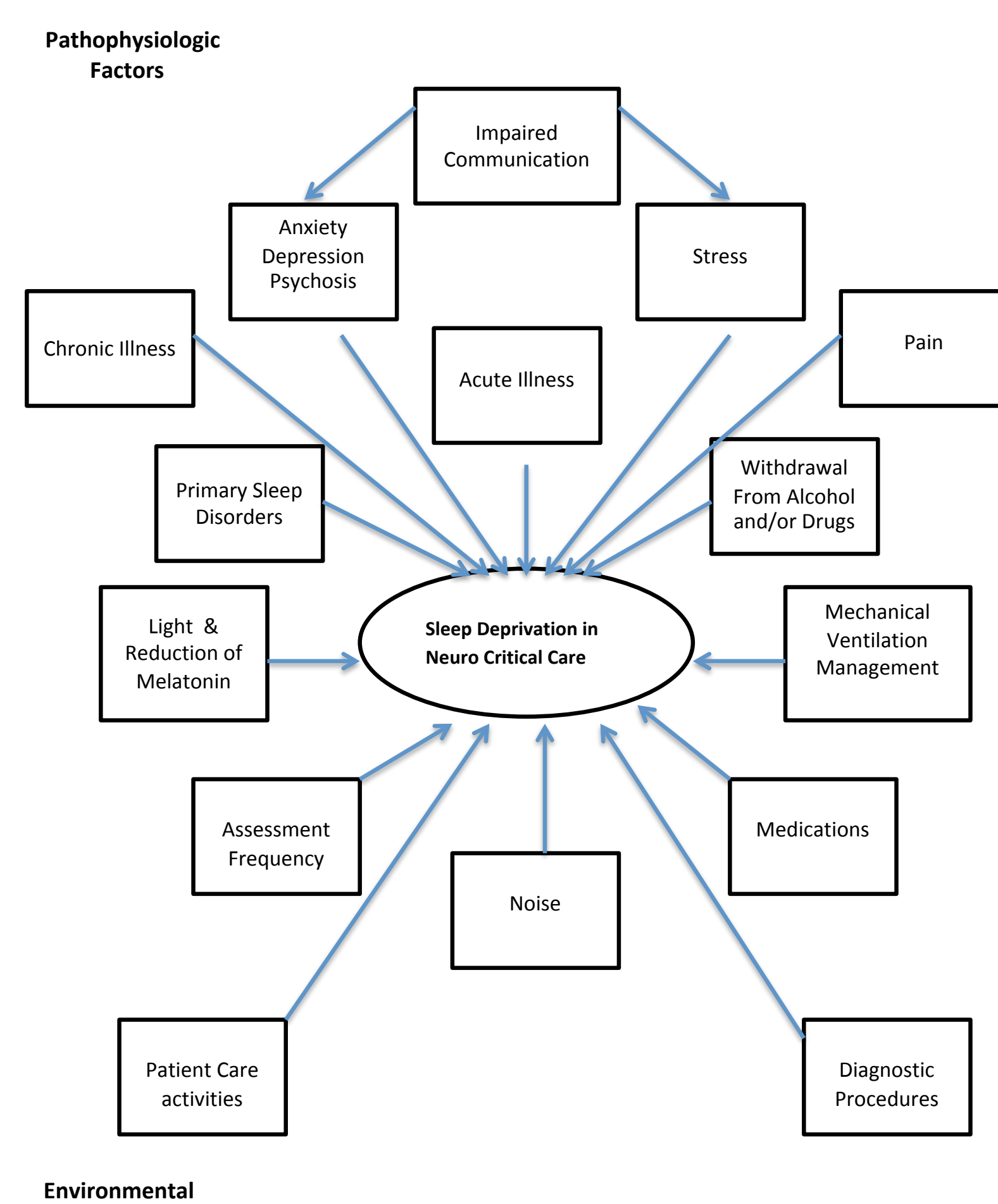
The purpose of the Neuroscience Intensive Care Unit is to provide specialized quality care to a high acuity and vulnerable critically ill population with the desired outcome of health improvement. It would appear that the goal for both Neuroscience Intensive Care Units and “sleep” would be one of healing; however it is also well documented that routine sleep disturbances due to either deprivation or disruption in neuroscience critical care units negatively affects patient outcomes.

Further exploration of sleep architecture, physiologic responses that occur during sleep and factors contributing to a lack of qualitative and quantitative sleep in critical care units needs to take place. With an enhanced understanding of these concepts, strategies to guide practice in order to improve quality of sleep in patients recovering in critical care environments can be formulated.

Search Methods

CINAHL, PubMed and OVID databases were used to search the literature. Key words sleep, sleep deprivation, sleep restriction, sleep architecture, sleep assessment tools were used. Articles were published between 2000 and 2015 in professional, peer reviewed journals and available in English to review.

Causes of Sleep Deprivation in Neuro Critical Care



Background

- Normal sleep architecture
 - Nonrapid eye movement (NREM) phases contribute to neural plasticity:
 - NREM- ↑ glucose uptake by basal forebrain, ventral striatum, hypothalamus, hippocampus and pontine reticular formation
 - N1 & N2- light sleep, body restoration and memory consolidation
 - N3- ↓ metabolic activity, ↓ oxygen consumption, ↓ cortisol, ↑ growth hormone
 - Rapid eye movement (REM) phase: ↑ cerebral blood flow, emotional recovery, brain rebuilding and growth
 - NREM+REM= a sleep cycle; Four to six sleep cycles of 90 -100 minutes each/night
 - 24 hour sleep/wake cycle
 - Regulation of circadian rhythm by norepinephrine, acetylcholine, dopamine, histamine, γ -aminobutyric acid (GABA), melatonin & serotonin
 - Optic sensors detecting ↓ light, neurons respond by stimulating pineal gland to secrete melatonin
 - Auditory pathways sense ↓ stimulation--connect with thalamus, brainstem (reticulating activating system) lead to permissive sleep
- Lack of Quantitative & Qualitative Sleep
 - Impaired higher cognitive and executive functions due to effects on prefrontal cortex
 - Psychomotor deficiencies and limited muscle strength
 - Depressed immune system: cortisol stimulation → decreased lymphocytes, monocytes and cytokines
 - Decreased tissue repair, ↓ pain tolerance and profound fatigue

Practice Implications for Bedside Clinicians and Advanced Practice Nurses

- **Advanced Physiology & Pathophysiology**
 - Working knowledge of dynamic process of sleep & impact on bodily functions
 - Providing education- critical illness & sleep disruption → consequences in air exchange, hemodynamics, immunity
 - Effects evident in 24-48 hours; short term recovery & long term recuperation effects
 - Heightening awareness that sleep interruption can occur as a product of health care delivery
 - Light & noise → stimulation of sympathetic nervous system → catecholamine production
 - Mechanical ventilation → dyssynchrony, alarms/suctioning → discomfort
- **Advanced Pharmacology**
 - Involvement in medication reconciliation- identification of prehospital sleep offending agents
 - Identifying/prescribing alternative pharmacologic agents to avoid sleep impairment & delirium
- **Assessment Directed Toward Health Promotion**
 - Assessment of chronic illnesses, chronic sleep disorders & substance use/withdrawal
 - Knowledge that sedation ≠ sleep
 - Identifying difficulties with age-related sleep + critical illness = effects on rehab & long term quality of life
- **Models of Care Delivery**
 - Current healthcare models: Neuro ICU environment centered on productivity & efficiency NOT on sleep
 - Provision of education on sleep; acting as a role model/mentor in promotion of rest
 - Facilitate change in culture: more patient centered care → less interrupted sleep
 - Promotion of creating/maintaining a healing environment (designated “Quiet Time”)
 - Reorganization of needed nocturnal interventions; ear plugs & light blocking masks
- **Decision Making**
 - Balance between sleep needs and necessary interventions (neurological assessment, pulmonary care, etc.)
- **Interprofessional Practice**
 - Creation of collaborative atmosphere among APN’s, bedside clinicians, physicians, pharmacists, respiratory therapists, pain management specialists, physical & occupational therapists, nutritionists, complementary practitioners and patient/family to address areas of sleep facilitation improvement

Common Neuroscience ICU Medications and Effects on Sleep Architecture

Drug Class or Individual Drug	Examples	Effects on Sleep Architecture
α_1 Receptor Blocker	Doxazosin, prazosin, terazosin	↑ TST
α_2 Receptor	Clonidine, dexmedetomidine	↑ Stage 1, SWS, nightmares ↓ REM
AED's	Carbamazepine, phenytoin, phenobarbital	Very sedating ↑ TST, SWS, sleep fragmentation ↓ sleep latency, REM
Alpha/beta agonists	Norepinephrine, epinephrine	Insomnia ↓ REM, SWS
Amiodarone	Amiodarone	Nightmares
Antibiotics	Beta-lactam, quinolones	Insomnia
Antihistamines	Diphenhydramine, promethazine	Sedating
Anxiolytic/benzodiazepines	Alprazolam, diazepam, lorazepam	Very sedating ↑ TST, stage 2 sleep ↓ REM, SWS, sleep latency
Corticosteroids	Dexamethasone, prednisone	Insomnia, nightmares ↓ REM, SWS
Dopaminergic	Dopamine	Insomnia ↓ REM, SWS
H2 receptor agonists/PPI	Ranitidine, omeprazole	Insomnia
Lipophilic β -blocker	Propranolol, metoprolol	Insomnia, nightmares ↓ REM; ↑ awakenings
NSAID	Ibuprofen, indomethacin, celecoxib	↓ TST, SE
Opioid	Codaine, morphine, hydrocodone	Sedating ↓ REM, SWS
SSRI	Paroxetine, fluoxetine, sertraline, citalopram	Sedating, but -TCA and MAOI's ↑ TST; ↓ REM, SWS, SE
TCA	Amitriptyline, nortriptyline	Very sedating ↑ TST, stage 2 sleep; ↓ REM

Abbreviations: REM- rapid eye movements; SWS- slow wave sleep; TST- total sleep time; SE- sleep efficiency

Conclusions

Although still not completely understood, more insight has been acquired regarding viewing “sleep” as an active biological recuperative method essential for physical and psychological well-being. Yet, many barriers to this restorative process continue to plague the critically ill neuroscience patient causing sleep deficit and taking a significant toll on patient recovery and outcomes.

Some of the contributions which alter sleep efficiency are nonmodifiable in this population, as they are primary sleep disorders or chronic illnesses which are present prior to the current acute illness; however there are many alterable situations and opportunities for the nurses to assist in augmenting the sleep process. Solutions to sleep issues can be derived from a collaborative, integrated, patient centered team approach assessing sleep disturbances, environmental influences, appropriate pharmacological management, optimizing behavioral interventions, providing education and engaging in future research on utilization of non-invasive positive pressure ventilation, exogenous melatonin and various complementary therapies.

The neuroscience nurse continues to be positioned to participate in and lead both quality improvement projects measured with validated tools, as well as cutting edge research, to optimize outcomes as sleep is facilitated.