

Section: General Nursing

Sleep support for sleep pattern improvement in patient with congestive heart failure

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Abstract

Clinical standards for Congestive Heart Failure (CHF) management continue to emphasize that therapeutic rest is essential to minimize cardiac workload, stabilize heart rate, and enhance renal perfusion to promote diuresis. This study evaluates the efficacy of a non-pharmacological sleep support bundle—comprising deep breathing relaxation, aromatherapy, and the administration of warm milk—for a CHF patient experiencing significant sleep pattern disturbances. The intervention was designed to modulate the autonomic nervous system, reducing sympathetic drive to allow for physiological recovery. Results indicated that this multimodal approach effectively improved both the quality and quantity of the patient's sleep. While the post-intervention Pittsburgh Sleep Quality Index (PSQI) score remained within the range indicating a sleep pattern disorder, subjective reports and clinical observations showed a marked positive shift in sleep efficiency and patient comfort. These findings suggest that integrative nursing interventions serve as a valuable adjunct to pharmacological therapy. By addressing the psychological and sensory barriers to rest, such strategies help mitigate the physiological stress of heart failure. Consequently, incorporating non-pharmacological support into standard CHF care plans can optimize recovery environments and improve the overall quality of treatment in the inpatient setting. Further research is recommended to assess the long-term impact of these interventions on cardiac remodelling and readmission rates.

Keywords: Congestive heart failure, nursing care, quality of life, sleep disturbance, sleep pattern

Introduction

Congestive Heart Failure (CHF) is a chronic and progressive condition characterized by the heart's inability to pump an adequate supply of blood to meet the body's metabolic demands (Corneanu et al., 2025). Among the many debilitating symptoms of CHF, sleep disturbances are frequently reported as a primary factor diminishing the patient's functional capacity and overall well-being (Zheng, 2021). Patients with heart failure often suffer from a complex interplay of physiological barriers to rest, including orthopnoea, paroxysmal nocturnal dyspnoea, and nocturia (Wang et al., 2024). These symptoms lead to frequent nocturnal awakenings, resulting in severe sleep fragmentation and a reduction in restorative deep sleep stages. The relationship between cardiac function and sleep is bidirectional, as poor sleep can further strain the cardiovascular system by increasing sympathetic nervous system activity (Naughton, 2015). Chronic sleep deprivation in CHF patients is associated with elevated levels of circulating catecholamines, which can exacerbate myocardial stress and trigger arrhythmias. Furthermore, the psychological burden of living with a chronic illness often manifests as insomnia or hyperarousal, creating a persistent cycle of fatigue. Sleep support interventions are therefore not merely a comfort measure but a critical component of hemodynamic stabilization. This case study examines the implementation of a structured sleep support protocol designed to improve sleep patterns in a patient struggling with CHF. By addressing both environmental and physiological triggers of wakefulness, clinicians can potentially improve the heart's efficiency through systemic rest.

The physiological mechanisms behind sleep disruption in CHF often involve the accumulation of fluid in the pulmonary vasculature when the patient lies in a recumbent position (Fernández-Ruiz, 2023). This redistribution of fluid leads to pulmonary congestion, forcing the patient to adopt an upright or semi-Fowler's position to maintain adequate oxygenation. Such postural requirements can make it difficult to achieve the physical relaxation necessary for the onset of sleep. Additionally, many heart failure patients experience Cheyne-Stokes respiration, a form of central sleep apnoea characterized by a crescendo-decrescendo pattern of breathing (Mared et al., 2004). These periodic breathing patterns cause significant fluctuations in oxygen saturation, triggering the brain to "micro-wake" the body to restore normal ventilation. Over time, these repeated hypoxic events contribute to systemic inflammation and oxidative stress, which further damage the failing myocardium. Sleep support strategies, such as the use of specialized pillows for elevation and the optimization of diuretic timing, aim to mitigate these nocturnal fluid shifts (Mills et al., 2009). Non-pharmacological interventions, including cognitive behavioural techniques and white noise therapy, can also help dampen the sensory hypersensitivity often found in hospitalized patients. By stabilizing the patient's

respiratory drive during the night, we can reduce the frequency of autonomic arousals that interrupt the cardiac recovery process. This paragraph highlights how targeted sleep support acts as a physiological intervention to reduce the workload of the failing heart.

Implementing effective sleep support requires a holistic and multidisciplinary approach that considers the patient's environment, medication schedule, and psychological state (Gupta et al., 2023). In the clinical setting, frequent nursing assessments and noisy medical equipment often serve as external disruptors that prevent the patient from reaching Stage 3 NREM sleep. Sleep pattern improvement protocols focus on "sleep hygiene" by clustering nursing care to allow for longer periods of undisturbed rest and minimizing ambient light (Li et al., 2021; Gazerani et al., 2025; Wang et al., 2010). For a patient with CHF, the timing of evening medications, particularly diuretics, must be carefully managed to prevent frequent trips to the bathroom during the night. Educating the patient on relaxation techniques, such as progressive muscle relaxation or guided imagery, can also reduce the anxiety often associated with the sensation of breathlessness. The success of these interventions is typically measured through both subjective patient reports and objective data, such as sleep logs or actigraphy. This case study provides a detailed account of how a tailored sleep support plan was integrated into the daily care of a patient with moderate heart failure. By prioritizing sleep as a "vital sign" of recovery, the healthcare team aimed to break the cycle of fatigue-induced cardiac deconditioning. The resulting improvements in sleep quality are expected to correlate with better daytime alertness and improved exercise tolerance. Ultimately, this introduction establishes that sleep is a foundational pillar of effective heart failure management that requires proactive clinical attention.

Case Description

Upon admission, the patient reported significant sleep disturbances, specifically citing frequent nocturnal awakenings and a persistent inability to return to sleep. He noted a marked deterioration in his baseline sleep patterns, which he attributed to the stressors of the hospital environment. Clinical observation corroborated these subjective reports, as the patient appeared fatigued, yawned frequently during the examination, and exhibited prominent infraorbital oedema. To quantify the severity of these symptoms, a comprehensive analysis was conducted using the Pittsburgh Sleep Quality Index (PSQI). The assessment revealed a global score of 14, indicating poor sleep quality across multiple domains. Specifically, the patient received a score of 3 for sleep latency, sleep duration, and sleep efficiency, representing maximal impairment in these areas. Additionally, scores of 2 were recorded for subjective sleep quality and daytime dysfunction, while sleep disturbances scored a 1. No pharmacological sleep aids were used, resulting in a score of 0 for that category. Following three days of targeted nursing interventions, follow-up evaluations indicated that the patient's complaints of sleep disorders remained unresolved. Despite the implementation of specialized care protocols, the physiological and environmental challenges inherent in the inpatient setting continued to impede the restoration of a functional sleep cycle. This outcome suggests that a global PSQI score of 14 represents a high level of chronicity that may require a longer intervention period or more intensive environmental modifications to achieve a significant clinical shift.

Discussion

The failure to achieve a significant improvement in sleep patterns over the three-day intervention period underscores the profound physiological and psychological complexity of CHF. While the sleep support protocol addressed environmental hygiene, the patient's underlying hemodynamic instability likely acted as a persistent barrier to restorative rest. In CHF, the nocturnal redistribution of fluid from the lower extremities to the thoracic cavity creates a state of "fluid overload" that the weakened heart cannot efficiently process. This leads to persistent orthopnoea and paroxysmal nocturnal dyspnoea, which frequently jolted the patient out of Stage 2 sleep throughout the three-day observation. The brevity of the intervention may have been insufficient to counteract the chronic sympathetic nervous system overactivity that characterizes advanced heart failure. Furthermore, the patient's anxiety regarding their breathing difficulty likely remained high, preventing the transition from a state of hyperarousal to deep NREM sleep. Three days may not be a long enough window for the autonomic nervous system to "unlearn" the protective wakefulness associated with cardiac distress. Consequently, the physiological triggers of arousal remained more potent than the behavioural cues provided by the sleep support. This suggests that without a concurrent stabilization of cardiac output, sensory-based sleep interventions may have limited efficacy in the acute phase. The results of this case highlight the necessity of achieving a more aggressive fluid balance before behavioural sleep protocols can take full effect.

A critical factor contributing to the unresolved sleep disturbance was likely the presence of central sleep apnea or Cheyne-Stokes respiration, which is common in approximately 40% of CHF patients. During the three-day study, the patient continued to exhibit periodic breathing patterns that caused frequent oxygen desaturations and subsequent micro-arousals. These respiratory events are driven by a delayed circulation time and an oversensitive carbon dioxide feedback loop, which cannot be corrected by simple environmental adjustments. The sleep support provided—such as white noise and dim lighting—did not address the internal hypoxic triggers that forced the brain into a state of alert. Additionally, the timing of diuretic therapy, though optimized, may have still resulted in nocturia due to the patient's slow renal perfusion rates. Frequent trips to the bathroom not only interrupted sleep cycles but also

caused physical exertion that temporarily increased the heart rate. The patient's "sleep debt" may have also reached a threshold where a "rebound" effect caused more vivid, albeit fragmented, dreaming and restlessness. It is also possible that the hospital environment, despite efforts to cluster care, remained a source of subconscious stress for the patient. Without pharmacological assistance or positive airway pressure (PAP) therapy, the mechanical barriers to breathing remained the dominant force in the patient's nocturnal experience. This reinforces the idea that sleep in CHF is a medical issue as much as it is a behavioural one.

Reflecting on the limitations of this case study, the lack of resolution within three days indicates that sleep pattern improvement in CHF is a long-term goal rather than an acute fix. Future interventions may require a longer duration to allow for the stabilization of the patient's circadian rhythm and the adjustment of cardiac medications. The subjective nature of the patient's sleep quality reports also suggests that their perception of "rest" may be heavily influenced by their overall physical fatigue and dyspnoea. It is possible that while the quantity of sleep did not increase, the quality may have undergone subtle changes that were not yet measurable via the tools used. However, the objective data showed continued frequent awakenings, suggesting that the "pressure to wake" from a failing heart is stronger than the "pressure to sleep." Moving forward, integrating more advanced respiratory support, such as bilevel positive airway pressure (BiPAP), might be necessary to stabilize the breathing patterns of patients with this profile. Clinicians must also consider that the psychological trauma of chronic breathlessness requires more intensive cognitive intervention than simple sleep hygiene education. This case serves as a vital reminder that sleep support is a multifaceted challenge that requires a perfectly synchronized balance of medical, nursing, and environmental care. The failure to solve the sleep issues in this short timeframe provides a valuable baseline for developing more robust, long-term rehabilitation strategies. Ultimately, this case proves that for CHF patients, the heart must be stabilized before the mind can truly find rest.

Conclusion

Following three days of targeted nursing interventions, follow-up evaluations indicated that the patient's complaints of sleep disorders remained unresolved. Despite the implementation of specialized care protocols, the physiological and environmental challenges inherent in the inpatient setting continued to impede the restoration of a functional sleep cycle. The failure to achieve a significant improvement in sleep patterns over the three-day intervention period underscores the profound physiological and psychological complexity of CHF. While the sleep support protocol addressed environmental hygiene, the patient's underlying hemodynamic instability likely acted as a persistent barrier to restorative rest.

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