Intensive & Critical Care Nursing xxx (2018) xxx–xxx

Contents lists available at ScienceDirect

Intensive & Critical Care Nursing

journal homepage: www.elsevier.com/iccn

Research article

Music intervention to prevent delirium among older patients admitted to a trauma intensive care unit and a trauma orthopaedic unit

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ARTICLE INFO

Article history:
Accepted 27 March 2018
Available online xxxx

Keywords:
Delirium
Music listening
Non-pharmacologic approach
Pharmacologic approach
Roy Adaptation Model
Heart rate
Systolic blood pressure
Diastolic pressure
Neuroscience of music

ABSTRACT

Purpose: Evaluate music listening for delirium prevention among patients admitted to a Trauma Intensive Care and Trauma Orthopaedic Unit. The Roy Adaptation Model provided the theoretical framework focusing on modifying contextual stimuli.
Methods: Randomised controlled trial, 40 patients aged 55 and older.
Intervention: Participants randomly assigned to receive music listening or usual care for 60 minutes, twice a day, over three days. Pre-recorded self-selected music using an iPod and headsets, with slow tempo, low pitch and simple repetitive rhythms to alter physiologic responses.
Outcomes: Heart rate, respiratory rate, systolic and diastolic blood pressure, confusion assessment method.
Results: Repeated measures ANOVA, $F(4, 134) = 4.75$, $p = .001$, suggested statistically significant differences in heart rate pre/post music listening, and $F(1, 37) = 10.44$, $p = .003$ in systolic blood pressure pre/post music listening. Post-hoc analysis reported changes at three time periods of statistical significance; ($p = .010$), ($p = .005$) and ($p = .039$) and a change in systolic blood pressure pre/post music listening; ($p = .001$) of statistical significance. All participants screened negative for delirium.
Conclusion: Music addresses pathophysiologic mechanisms that contribute to delirium; neurotransmitter imbalance, inflammation and acute physiologic stressors. Music to prevent delirium is one of few that provide support in a critical care setting.

Implications for clinical practice

- Music listening for Delirium prevention has been shown to be both effective and safe.
- Music listening addresses pathophysiologic mechanisms that contribute to delirium; neurotransmitter imbalance, inflammation, and acute physiologic stressors.
- Music to prevent delirium is one of few that provide support in a critical care setting.

Introduction

Delirium is a neurobehavioural syndrome characterised by alterations in consciousness, attention, cognition and perception (Mattar et al., 2013; Kalaria and Mukaetova-Ladinska, 2012). Recent theories addressing the pathophysiology of delirium propose that different interacting biological factors disrupt neuronal networks in the brain resulting in cognitive dysfunction (Inouye et al., 2014). Leading mechanisms that contribute to delirium include neurotransmitter imbalance, inflammation and physiologic stressors (Inouye et al., 2014). Delirium prevention has recently been emphasised in national safety reports and as a health care quality indicator (Inouye et al., 2014; Field and Wall, 2013) and

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https://doi.org/10.1016/j.iccn.2018.03.007
0964-3397/

is clearly of significant importance when addressing the care of older adults.

Delirium in older adults

The highest rate of delirium occurs in hospitalised older adults (Nouwen et al, 2012; Inouye et al., 2014). Delirium increases intensive care unit (ICU) days, hospital length of stay (LOS), cognitive and functional decline and is most significant for family and caregivers, increased dependency, long term neuropsychological impairment and institutionalisation (Allen and Alexander, 2012; Inouye et al., 2014; Field and Wall, 2013). Delirium remains a direct indicator of morbidity and mortality in older adults following hospital discharge (Neufeld et al., 2011; Mistraletti et al., 2012; Devlin et al., 2012). Adults aged 65 years and older who are hospitalised for delirium cost United States hospitals $164 billion per year and greater than $182 billion per year in 18 European countries combined (Inouye et al., 2014).

Clinical presentation

Clinical manifestations of delirium include hyperactive, hypoactive and mixed delirium (Allen and Alexander, 2012). Patients with hyperactive delirium present with agitation, restlessness and attempting to remove devices (Allen and Alexander, 2012; Lundstrom et al., 2012). Mixed delirium is a combination of hyperactive and hypoactive delirium, is the second most prevalent in ICU settings, and is more difficult to manage. Hypoactive delirium is seen most often (60%) in ICU settings with 65% to 75% of cases not recognised and treated (Hipp and Ely 2012; Allen and Alexander 2012; Lundstrom et al., 2012). Characteristics of hypoactive delirium include flat effect, withdrawal, apathy, lethargy, decreased level of responsiveness and minimal psychomotor activity (Allen and Alexander, 2012).

Pathophysiology of delirium

Main mechanisms that contribute to delirium include neurotransmitter imbalance, inflammation and physiologic stressors (Inouye et al., 2014). Normal age related changes in the brain can alter neurotransmission. Neurotransmitters acetylcholine (ACh), serotonin (5HT), dopamine (DA), glutamate and Gamma Aminobutyric Acid (GABA), are involved in delirium (Lorenzo et al., 2013). The neurotransmitter GABA is a major inhibitor in the central nervous system (CNS) and a reduction can lead to delirium. A reduction in the neurotransmitters ACh can result in impaired behavioral responses related to delirium (Inouye et al., 2014; Brown and Purdon, 2013). Neurotransmitter 5HT is involved in mood, wakefulness and cognition; when elevated can cause impaired memory and learning with selective reuptake inhibitors associated with delirium (Hughes et al., 2012; Lorenzo et al., 2013). Glutamate is involved in learning and memory and with certain drugs can cause anxiety and psychosis which can lead to delirium (Brown and Purdon, 2013; Inouye et al., 2014). An imbalance of the neurotransmitter DA can result in adverse effects from DA excess, increasing delirium (Inouye et al., 2014; Mora et al., 2012).

With normal age related changes there is a low grade inflammation with chronic neurodegenerative changes in brain (Mora et al., 2012). Older adults who have an inflammatory state due to age related changes, have a more severe central nervouse system (CNS) response when an infection or a trauma which may require surgery adds to this present inflammatory state. An inflammatory cascade happens after surgery, peaks 24 hours later and increases risk for delirium (MacLullich et al., 2013).

With age related changes there is a lower physiologic reserve to maintain homeostasis in response to stress (Mora et al., 2012). Stress hormones are released due to a stress response from surgery, pain, trauma, & systemic inflammation, causing and prolonging delirium (Mora et al., 2012; MacLullich et al., 2013). Hospitalised older adults also experience acute stress from sensory impairment, medications, immobilisation, physical restraints, noise stimuli and sleep deprivation (Inouye et al., 2014).

Delirium prevention

Preventing delirium is considered standard of care and is the most effective course in decreasing frequency and adverse outcomes (Hipp and Ely 2012; Inouye et al., 2014; MacLullich et al., 2013; Khan et al., 2012). Current clinical approaches to prevention include pharmacologic and non-pharmacologic approaches (Hipp and Ely, 2012).

Pharmacologic approaches

Pharmacologic approaches in the treatment of delirium can include atypical and typical (Haloperidol) and atypical (Olanzapine, Risperidone, Ziprasidone) antipsychotics, Cholinesterase inhibitors (Rivastigmine, Donepezil), sleep aides (Melatonin and Ramelteon), and sedatives (dexmedetomidine and benzodiazepines) (Kluger et al., 2018). There is no present evidence to support the use of antipsychotics, cholinesterase inhibitors or psychostimulants to prevent delirium in acute care settings (Kluger et al., 2018). Delirium prevention can include avoiding use of sedatives, specifically benzodiazepines (Kluger et al., 2018).

Clinical trials have been conducted to examine the efficacy of pharmacologic management in treating delirium and have found no evidence to support use of antipsychotics in preventing and treating delirium in medical patients (Kluger et al., 2018). Current recommendations from the American Geriatric Society do not support use of antipsychotics for preventing delirium or as first line treatment of delirium (Kluger et al., 2018).

Inouye et al. (2014) reviewed 16 studies that used a pharmacologic approach for delirium prevention and treatment. There was no evidence reported to support use of pharmacologic approaches for delirium prevention (Inouye et al., 2014). Rates of delirium did not differ significantly in six trials. In eight trials, the treatment group reduced rates with no effect on clinical outcomes (intensive care unit (ICU) admission, length of stay (LOS), complications or mortality). Treatment groups in two studies had worse outcomes compared to the control group. Olanzapine decreased the incidence of delirium but increased duration and severity of delirium, and rivastigmine increased duration and mortality. Overall findings for pharmacologic approaches for delirium prevention and treatment was not recommended (Inouye et al., 2014).

Siddiqi et al. (2016) reviewed 39 clinical trials that recruited 16,082 participants, examining 22 different interventions and comparisons. Pharmacologic approaches included typical antipsychotics (haloperidol) and atypical antipsychotics (olanzapine). There was low-quality evidence to support use of haloperidol, (RR 1.05, 95% CI 0.69–1.60; two studies; 516 participants, low-quality evidence) and moderate-quality evidence to support use of olanzapine, (RR 0.36, 95% CI 0.24–0.52; one study; 400 participants).

Non-pharmacologic approaches

Non-pharmacologic approaches for delirium prevention focused on optimizing cognition, early mobilisation, and sleep promotion (Barr et al., 2013; Inouye et al., 2014; Khan et al., 2012).

Please cite this article in press as: Johnson, K., et al. Music intervention to prevent delirium among older patients admitted to a trauma intensive care unit and a trauma orthopaedic unit. Intensive & Critical Care Nursing (2018), https://doi.org/10.1016/j.iccn.2018.03.007