Nursing Care of the Patient with Aneurysmal Subarachnoid Hemorrhage

AANN Clinical Practice Guideline Series

AANN
American Association of Neuroscience Nurses

8735 W. Higgins Road, Suite 300
Chicago, IL 60631
888.557.2266
International phone: 847.375.4733
Fax: 847.375.6430
info@aann.org • www.AANN.org

Clinical Practice Guidelines Editorial Board Editor
Sarah Livesay, DNP RN ACNP-BC ACNS-BC SCRN

Clinical Practice Guidelines Editorial Board Liaison
Molly McNett, PhD RN CNRN FNCS

Lead Author
Joan Censullo, PhD MSN

Content Authors
Abed Al Wahab Al Firikh, MSN CNRN
Malissa Mulkey, MSN APRN CCNS CCRN CNRN
Molly McNett, PhD RN CNRN FNCS
Sarah Livesay, DNP RN ACNP-BC ACNS-BC SCRN
Renee Ovando, SCRN (author mentee)

Content Reviewers
Megan Brissie, DNP APRN ACNP-BC
Michelle Kearney, MSN APRN ACNP-BC
Jillian Polonchak, BSN RN ACLS PHN SCRN

AANN National Office
Joan Kram
Executive Director

Bryan O'Donnell
Managing Editor

Sonya Jones
Graphic Designer

Publisher's Note
The authors, editors, and publisher of this document neither represent nor guarantee that the practices described herein will, if followed, ensure safe and effective patient care. The authors, editors, and publisher further assume no liability or responsibility in connection with any information or Recommendations contained in this document. These Recommendations reflect the judgment from the American Association of Neuroscience Nurses regarding the state of general knowledge and practice in our field as of the date of publication and are subject to change based on the availability of new scientific information.

Copyright © 2018 by the American Association of Neuroscience Nurses. No part of this publication may be reproduced, photocopied, or republished in any form, print or electronic, in whole or in part, without written permission of the American Association of Neuroscience Nurses.

Preface

In 1997 the American Association of Neuroscience Nurses (AANN) created a series of patient care guidelines, the *AANN Reference Series for Clinical Practice,* to meet neuroscience nurses' needs for educational resources. To reflect the nature of the guidelines and the organization's commitment to developing each guideline based on current literature and evidence-based practice, the name of the series was changed in 2007 to the *AANN Clinical Practice Guideline Series (CPG).*

Volumes in the AANN CPG series are reviewed annually and updated when evidence emerges that renders the nursing recommendations invalid or when new literature supports existing recommendations.

The CPG development process evolved to ensure congruence with best practices, specifically alignment with Institute of Medicine recommendations for guideline development. Therefore, each new CPG now contains a general overview of the disease state along with targeted PICOT questions to guide a systematic review of the literature in order to identify cumulative evidence related to the specific topic. The Grading of Recommendations Assessment, Development, and Evaluation (GRADE) methodology is used to guide generation of these PICOT questions, subsequent literature searches, analysis of findings, and interpretation of the literature to generate practice recommendations.

Consistent with the GRADE methodology, recommendations are made as either strong or weak, with an accompanying level of evidence quality: low, moderate, or high.

Resources and recommendations in this document reflect best practices that can help nurses provide optimal care for adults after aneurysmal subarachnoid hemorrhage (aSAH). However, they are not a substitute for nursing judgment. Adherence to these guidelines is voluntary, and the ultimate determination about their application must be made by the nurse in light of each patient's circumstances. In addition, these guidelines may be superseded by institution-specific policy. This document is not intended to replace formal education but rather to augment the nurse's knowledge base and provide a readily accessible reference.

The nursing profession and AANN are indebted to the volunteers who have devoted their time and expertise to this valuable resource, which was created for those committed to excellence in caring for patients after aSAH.

Introduction

Medical and surgical management of patients with aneurysmal subarachnoid hemorrhage (aSAH) has been extensively studied, with numerous organizations conducting critical reviews of the scientific literature to formulate evidence-based recommendations. However, nursing management of this population has been examined less, and evidence-based recommendations are needed to guide nursing care when working with these high-risk patients. In response, the American Association of Neuroscience Nurses (AANN) appointed a writing group to conduct a critical review of the literature to determine best-practice recommendations for nursing care of patients with aSAH. Grading of Recommendations Assessment, Development, and Evaluation (GRADE) methodology was used to evaluate evidence and formulate practice recommendations. Problem/patient/population, intervention/indicator, and comparison and outcome (PICO) questions were formulated a priori, with systematic literature searches performed using specific terms to target key components of each question. A writing group conducted an exhaustive search of the literature using PubMed, CINAHL, MEDLINE, the Cochrane Library, and the Joanna Briggs Institute Evidence-Based Practice Database to find studies on aSAH published within 10 years of September 2017, with exceptions for limited available research and seminal manuscripts. Non-English, nonhuman, and unpublished works were excluded from final search results. Priority was given to meta-analyses, randomized clinical trials (RCTs), observational studies, and existing evidence-based practice guidelines that pertained to each PICO question. Evidence was critically reviewed for each question and summarized in this guideline's evidence tables. Each content expert provided initial recommendations involving evidence from the included studies and evidentiary tables that were reviewed by the writing group. Recommendations then were submitted for internal and external review in a stepwise progression before final approval by AANN and subsequent publication. Clinical questions that guided each search strategy are presented sequentially, followed by a summary of the literature and specific recommendations to guide nursing care of patients with aSAH. Any absence of quantitative recommendation parameters was attributed to an absence of definitive outcomes measures in the literature.

aSAH Background

aSAH involves bleeding into the subarachnoid brain space caused by the rupture of a cerebral aneurysm. Because the pathophysiology is cerebrovascular in nature, aSAH often is grouped with other cerebrovascular diseases such as ischemic stroke and intracerebral hemorrhage (ICH). Approximately 795,000 people in the United States experience a new or recurrent stroke each year, with 3% (approximately 24,000) of those affected experiencing an aSAH (Benjamin et al., 2017). The approach to aSAH has evolved over the decades to include treatment of aneurysms using open surgical and endovascular approaches and management of delayed cerebral ischemia (DCI) during the days and weeks following the initial bleed (Milinis, Thapar, O'Neill, & Davies, 2017). Patients with aSAH often require an extended intensive care unit (ICU) and hospital stay (Diebolt, Sims, Conners, & Lee, 2014), which necessitates coordinated and expert nursing care.

Morbidity and Mortality

Do aSAH scales help practitioners predict recovery and treatment efficacy during hospitalization and for the long term?

Several scoring systems that grade severity of injury among patients with aSAH have been described in the literature during the past 10 years. Because scores commonly are referred to during routine clinical care, nurses should be familiar with the various scoring systems and their implications. Common scoring systems that grade injury severity upon admission and during acute care management of aSAH include the Hunt and Hess (H & H), modified and original Fisher Scales, and World Federation of Neurologic Surgeons (WFNS) scale scores. Because scoring systems indicate some degree of aSAH severity after aneurysm detection, their ability to predict short- and long-term outcomes inclusive of mortality, clinical progression, disease course, and functional outcomes at discharge and for up to 3 years postinjury has been investigated. Morbidity after hemorrhage or intervention frequently is documented via the modified Rankin Scale (mRS; 0 denotes *asymptomatic* and 5 denotes *severe disability*), which quantifies level of disability.

Studies examining the predictive ability of the H & H scale have examined relationships between scores and mortality, DCI incidence, need for retreatment or shunt dependence, and functional outcome among patients with aSAH. H & H grades upon admission and prior to aneurysm treatment are predictive of mortality at 1 year, with higher scores associated with higher mortality (L. H. Bian et al., 2012). Higher H & H grades also have been associated with increased need for vasospasm retreatment (Chalouhi et al., 2014), incidences of cerebral infarction (Dengler, Sommerfeld, Diesing, Vajkoczy, & Wolf, 2018), and decreased functional outcome at 1 year as measured by an mRS score higher than 3 (Chalouhi et al., 2014; Dengler et al., 2018; Duan et al., 2016; Mocco et al., 2006; Salary, Quigley, & Wilberger, 2007). Scores in isolation and in combination with other factors are important components when attempting to evaluate shunt-dependent hydrocephalus and the need for ventriculoperitoneal shunting (VP; Yu, Zhan, Wen, Shen, & Fan, 2014). In multiple risk prediction models, inclusion of H & H grade can improve predictive ability for functional outcome, particularly when compared to the prognostic value of radiographic findings to predict the same outcomes (Dengler et al., 2018; Jaja et al., 2013). H & H grades also help predict in-hospital mortality, with Grade 2 denoting an associated risk of 3% morality and Grade 5 suggesting a mortality rate as high as 71% (Lantigua et al., 2015).

Both the modified Fisher Scale (0 = *no SAH* to 4 = *diffuse SAH with intraventricular hemorrhage* [IVH]) and original Fisher Grading Scales (1 = *no SAH* to 4 = *diffuse SAH with IVH*) are radiographic-based scores that quantify the diffusion of a hemorrhage. They also have been widely studied as predictors of outcome. The newer modified Fisher Scale differs from the original Fisher Scale in range and gradation definitions. Higher original Fisher scores are associated with higher mortality rates and poor 1-year functional outcomes (Bian et al., 2012; Duan et al., 2016; Solanki, Pandey, & Rao, 2016). Low modified Fisher scores are associated with excellent outcomes at 1 year post injury (mRS score 0-1) in univariate analysis (Pegoli, Mandrekar, Rabinstein, & Lanzino, 2015) but not in multivariate models. Other studies have investigated correlations between scores and poor functional outcome (defined by mRS), mortality, shunt-dependent hydrocephalus, hemorrhage, and DCI (Lantigua et al., 2015; Mahaney, Todd, Bayman, Torner, & IHAST Investigators, 2012). Specifically among elderly populations, higher modified Fisher scores are associated with poor functional outcome at 1 year, as measured by mRS scores lower than 3 (Duan et al., 2016). Increased modified Fisher grades also are predictive of shunt-dependent hydrocephalus (Duan et al., 2016), risk for aneurysmal rebleed (Pegoli et al., 2015), acute neurological deterioration (Chalouhi et al., 2014; Mahaney et al., 2012), and DCI incidence (Crobeddu et al., 2012).

The WFNS score (1 = *best* to 5 = *worst*) is based on a combined grade indicating Glasgow Coma Scale score and functional motor deficit. WFNS score is a consistent indicator in both univariate and multivariate models of excellent outcomes after aSAH as measured by an mRS of 0 to 1 at 1 year (Pegoli et al., 2015). WFNS scores have demonstrated superior predictability for functional outcomes in some studies when compared to radiographic findings alone (Dengler et al., 2018). Although higher WFNS scores are not associated with a poor clinical course (Iosif et al., 2014), lower scores (between 1 and 3) are associated with lower DCI incidence (Crobeddu et al., 2012; Dengler et al., 2018).

Scoring metrics provide important information on the severity of neurological injury and should be considered as part of the overall clinical picture when making treatment decisions for patients with aSAH. When examining the predictive ability of scores, each metric is strongly associated with clinical course and long-term outcomes, particularly when paired with other predictors. H & H and original and modified Fisher scores remain strong predictors of complications such as rebleeding and need for retreatment, cerebral infarction, and DCI. Both scales also correlate with mortality and poor functional outcomes after discharge. WFNS scores are linked to incidence of delayed cerebral edema and functional outcomes after discharge.

Recommendation

- When paired with clinical indicators, the H & H, Fisher, and WFNS scales are important determinants of neurological injury severity and predictors of outcomes after aSAH (strong recommendation, high-quality evidence).

Palliative Care in aSAH

Does a palliative care consultation result in enhanced satisfaction for patients and families affected by aSAH?

The American Cancer Society defines palliative care as "care for adults and children with serious illness that focuses on relieving suffering and improving quality of life for patients and their families, but is not intended to cure the disease itself" (American Cancer Society, 2014). Unlike hospice or end-of-life care, palliative care is appropriate in the context of any serious illness regardless of stage or prognosis. Because of the high morbidity (57%) and mortality (20%) associated with aSAH (Brain Aneurysm Foundation, n.d.), patients and families may benefit from palliative care interventions. A 2015 study pointed to lower overall in-hospital mortality when compared to preadmission mortality rates in aSAH patients (Lantigua et al., 2015; Milinis et al., 2017). Also, aSAH survivors often live with substantial deficits. Palliative care optimally is provided in conjunction with, not in lieu of, disease-directed or life-prolonging treatment and should focus on symptom relief, effective communication about care goals, the alignment of treatment with patient/family preferences, emotional support, and planning for transitions (Burton & Payne, 2012; Creutzfeldt, Holloway, & Curtis, 2015; Frontera et al., 2015).

The literature on aSAH and palliative care is scant and dominated by publications focused on end-of-life care and prevalence. Existing research suggests that providers have difficulty discussing treatment options—inclusive of palliative treatments—in the setting of poor prognosis; this difficulty results in less than 4% of patients with aSAH having aggressive treatment withdrawn, compared to 15% of all patients in the neuro-ICU (Qureshi, Adil, & Suri, 2014). A national database study of American patients with stroke receiving inpatient care revealed that 8.8% of patients with aSAH received palliative care services (American Cancer Society, 2014). Palliative care typically was consulted only for individuals who were severely functionally impaired, lacked decision-making capacity, and had a high likelihood of in-hospital death (Holloway et al., 2010; Qureshi et al., 2014; Singh, Peters, Tirschwell, & Creutzfeldt, 2017).

Blacquiere and colleagues (Blacquiere, Bhimji, Meggison, Sinclair, & Sharma, 2013) published the sole study on palliative care and patient/family satisfaction in stroke care. This single-center study included only 15 patients, five of whom had hemorrhagic stroke or aSAH. These investigators found that family perception of palliative care revealed high satisfaction, especially related to pain management. Presently, there is insufficient evidence to support a recommendation regarding palliative care in the setting of aSAH. However, it is worth noting that the American Heart Association/American Stroke Association (AHA/ASA) has published a policy statement that advocates for palliative care services for patients with cardiovascular disease and their families (Braun et al., 2016) and much of the statement relies on research findings on other disease specialties.

Recommendation

- Palliative care should be considered for patients with aSAH if symptoms such as pain are poorly controlled or if the patient or family members are struggling with goals of care or coping (Good Practice Statement).

Management

What is the effect of cerebrospinal fluid (CSF) drainage for the management of acute hydrocephalus on hospital and functional outcomes after aSAH?

Symptomatic hydrocephalus is a common complication that occurs in about 20% of patients with aSAH within the first 3 days and is associated with neurological impairment and increased mortality (Mocco et al., 2006; Vivancos et al., 2014). Hydrocephalus is distinguished by the presence of intraventricular blood rather than cisternal blood, as detected with computed tomography (CT; Chen, Luo, Reis, Manaenko, & Zhang, 2017). Clinical signs include worsening level of consciousness, poorly reactive pupils, or impaired gaze, and diagnosis can be confirmed with a noncontrast head CT demonstrating increased ventricular size. Common pharmacological treatments for hydrocephalus include mannitol and acetazolamide. Treatment with external ventricular drainage (EVD) may be lifesaving and can improve consciousness and responsiveness shortly after placement (Chen et al., 2017; Salary et al., 2007).

Chronic hydrocephalus occurs in 8.9% to 48% of patients with aSAH (Connolly et al., 2012). CSF diversion with EVD and shunt placement for permanent CSF diversion are common and well-established practices for the management of hydrocephalus in aSAH and are recommended by both the AHA (Connolly et al., 2012) and European Stroke Organization (Steiner et al., 2013). A 2016 meta-analysis by Qian et al. (2016) analyzed studies that compared subjects who received any type of CSF drainage to subjects who did not receive CSF drainage and concluded that instituting some type of CSF drainage decreased the incidence of vasospasm and infarction related to vasospasm after aSAH (Qian et al., 2016). Subjects who underwent CSF drainage experienced lower mortality rates (3.3% vs. 8.3%) and improved long-term functional outcomes as measured by a Glasgow Outcomes Scale (GOS) score higher than 3 or mRS lower than 3 (80.9% vs. 63.5%). The complication rate associated with any type of CSF drainage was 5.3%, and complications included infection, headache, and hemorrhage (Qian et al., 2016). Despite the demonstrated benefits associated with CSF drainage, substantial variation remains regarding the method of CSF drainage, including lumbar drainage (LD) vs. EVD drainage and continuous vs. intermittent drainage. Qian et al. (2016) meta-analysis also included a comparison of LD vs. EVD drainage across studies (Qian et al., 2016). However, only two studies were included and results were equivocal, with neither approach demonstrating superior outcomes.

Only one RCT has compared the effectiveness of continuous and intermittent CSF drainage (Olson, Zomorodi, et al., 2013). This trial of 60 subjects with aSAH who were randomized to continuous CSF drainage or intermittent (on-demand) drainage was terminated early because of preliminary safety concerns. Findings indicated that those undergoing continuous CSF drainage experienced more complications including CSF leak, hemorrhage, inadvertent device removal, ventriculitis, and clogged EVDs (52.9% vs. 23.1%). Despite these complications, the drainage approach did not appear to influence the incidence of vasospasm; estimates were comparable between groups (Olson, Zomorodi, et al., 2013).

Studies also have compared continuous to intermittent CSF drainage after aSAH (Kim et al., 2011). A pilot study of 37 subjects undergoing intermittent or continuous approaches reported no difference in vasospasm incidence (Kim et al., 2011). Another study of 62 patients with traumatic brain injury (TBI) concluded that those with intermittent drainage experienced increased mean intracranial pressure (ICP) values and greater ICP burden (ICP > 20 mmHG) than patients in the continuous drainage group (Nwachuku et al., 2014). Another pilot observational study of 37 subjects concluded that both intermittent and continuous approaches resulted in similar incidence of vasospasm, ICP values, length of stay (LOS), volume of CSF drainage, and mRS upon discharge (Amato et al., 2011).

Studies also have investigated the use of LDs for CSF draining after aSAH. LD use in general has been documented as safe (Hoekema, Schmidt, & Ross, 2007), may decrease risk for ICH (Sun et al., 2014), and may improve clinical outcomes at discharge when compared to a no-drainage approach after aSAH (Al-Tamimi et al., 2012; Kwon et al., 2008; Park, Yang, & Seo, 2015). When comparing LD to EVD after aSAH, LD use may be associated with higher rates of infection and hydrocephalus (Sun et al., 2014). However, rates of vasospasm and chronic hydrocephalus, duration of catheter placement, and GOS scores at 2 months have been reported as being similar between groups (Sun et al., 2014). As a growing body of literature supports the use of CSF drainage after aSAH, variations in drainage approaches remain. No consistent, compelling evidence favors one CSF drainage approach over another after aSAH.

Recommendation

- CSF drainage after aSAH is recommended as a treatment option for management of acute hydrocephalus to improve patient outcomes (strong recommendation, moderate-quality evidence).

Which CSF management practices can decrease the incidence of hospital-acquired conditions (HACs)?

EVD management

Infection of the EVD system is a common complication for patients with aSAH. Rates of infection range from 1% to 40% (Camacho et al., 2011). Infection rates may vary widely because studies employ different definitions of EVD infection. Some authors use the Centers for Disease Control and Prevention definition that specifies positive CSF culture and laboratory findings and clinical symptoms (Scheithauer et al., 2009). Other authors define infection as simply positive CSF cultures (Arif et al., 2012). The lack of a standardized definition results in inconsistent reported rates.

CSF sampling

When caring for patients with aSAH and EVD, CSF cultures may be taken routinely or only if clinically indicated. One study compared infection rates based on the frequency of CSF sampling among 382 adults in the ICU. EVD-related infections were much less common and reduced to 3% when CSF samples were taken every third day when compared to more frequent sampling (Williams, Leslie, Dobb, Roberts, & van Heerden, 2011). Another study of 410 patients in the neuro ICU demonstrated higher infection risk with increased sampling frequency: each CSF sample was associated with an 8.3% increase in the risk for an EVD-related infection (Williamson et al., 2014). A retrospective study of 228 neurosurgical patients indicated that frequent CSF sampling was a substantial risk factor for infection, particularly when paired with increased duration of catheter use (Hoefnagel, Dammers, Ter Laak-Poort, & Avezaat, 2008). Despite these findings, a 2015 meta-analysis of 35 prospective and retrospective observational studies investigating risk factors for ventriculitis reported similar infection rates among studies when routine (10.8%) or clinically indicated (11.2%) sampling was performed (Ramanan, Lipman, Shorr, & Shankar, 2015). However, there was wide heterogeneity across studies for measurement of ventriculitis, study populations, and methodological rigor.

Recommendation

- CSF samples should be obtained from EVDs when clinically indicated rather than at frequent intervals (weak recommendation, low-quality evidence).

What effects do nursing interventions have on cerebral hemodynamics?

There is much variation regarding nursing care for patients with abnormal cerebral hemodynamics (Olson, McNett, Lewis, Riemen, & Bautista, 2013). Research suggests the effect of nursing interventions on cerebral hemodynamics on patients with neurologic illness (including patients with aSAH) also is variable and is influenced by the context in which the intervention was initiated (Olson, McNett, et al., 2013; Salary et al., 2007). Overall, nursing interventions appear to be safe, and although one study of 18 neurocritically ill patients suggested avoiding clustering nursing interventions to minimize the impact on ICP, the impact of these interventions on secondary insults is minimal (Nyholm, Steffansson, Frojd, & Enblad, 2014; Olson, McNett, et al., 2013). The research literature primarily focuses on nursing interventions in the critical care setting that include oral care, CPT, ETS, repositioning, HOB elevation, and early ambulation. In addition, research primarily documents outcomes for mixed neurocritically ill populations that include but are not limited to patients with aSAH.

Oral care

Oral care is a well-established intervention to reduce the risk for ventilator-acquired pneumonia. Observational studies have demonstrated that, despite wide variations in which registered nurses provide oral care, oral care is safe and does not substantially increase ICP in adults or children with neurological impairment (Prendergast, Hallberg, Jahnke, Kleiman, & Hagell, 2009; Tume, Baines, & Lisboa, 2011). Oral care is safe to use in mixed populations in the neurocritical care setting regardless of the duration, intensity, or type of product used (McNett & Olson, 2013; Nyholm et al., 2014; Prendergast, Hagell, & Hallberg, 2011; Prendergast et al., 2009; Szabo, Grap, Munro, Starkweather, & Merchant, 2014). Oral care may be associated with mild increases in ICP; however, elevations appear to be transient and do not result in neurologic deterioration or have an adverse impact on cerebral perfusion pressure (CPP) values (Nyholm et al., 2014; Szabo et al., 2014).

Recommendation

- Routine oral care for patients who require mechanical ventilation is recommended (strong recommendation, moderate-quality evidence).

CPT

CPT promotes pulmonary hygiene and improves oxygenation. Several studies have investigated severe neurologic injury and elevated ICP resulting from various disease processes, but no study solely included patients with aSAH. Two observational studies addressed the effect of CPT on cerebral hemodynamics in patients with neurological impairment resulting from multiple etiologies. CPT was found to be safe and decreased ICP in patients with an EVD (Dengler et al., 2018; Olson, McNett, et al., 2013; Olson, Thoyre, Turner, Bennett, & Graffagnino, 2007). Similarly, two pilot RCTs concluded that automated CPT did not have a significant effect on ICP (Olson, Bader, Dennis, Mahanes, & Riemen, 2010; Olson, Thoyre, Bennett, Stoner, & Graffagnino, 2009).

Recommendation

- CPT is recommended when clinically indicated and does not appear to adversely affect ICP (strong recommendation, moderate-quality evidence). This recommendation is extrapolated from data on patients with various disease etiologies; further studies are needed to validate these findings in patients with aSAH.

Positioning

Nurses change patient position frequently to maintain skin integrity. Several prospective observational studies on patients with neurological impairment have concluded that ICP with respect to position (lateral, supine, prone) varies from one patient to the next (Ledwith et al., 2010; Olson, McNett, et al., 2013; Salary et al., 2007). One study concluded that changing patient position reduces ICP within 5 minutes after the intervention in mixed neurocritical care populations (Olson, McNett, et al., 2013). However, another pilot study linked repositioning to sustained high ICP among 18 neurocritically ill patients, particularly when paired with other interventions (Nyholm et al., 2014). Another study found that baseline elevated ICP may be an important predisposing factor for the development of a secondary insult with repositioning (Nyholm, Howells, & Enblad, 2017). Benefits associated with repositioning, specifically avoidance of pressure injury and promotion of respiratory function, are important considerations that support the use of this intervention during the acute stage of illness and may mitigate the influence of transient, minor ICP increases.

Recommendations

- Patient repositioning during the acute stage of illness is recommended in the absence of increased ICP (strong recommendation, moderate-quality evidence).
- If intracranial hypertension is suspected, consider repositioning at intervals that do not coincide with other care activities (weak recommendation, low-quality evidence).

ETS

ETS is a common nursing intervention for patients who require mechanical ventilation. In the setting of mechanical ventilation with an EVD, studies have noted an association between ETS and ICP elevation (McNett & Olson, 2013). An observational study of children undergoing mechanical ventilation revealed that ICP values were substantially higher during ETS (Tume et al., 2011). Among patients in this study, 68% recovered to baseline ICP within 5 minutes and 52% achieved ICPs lower than baseline before the nursing intervention (Tume et al., 2011).

Observational studies of adults requiring mechanical ventilation and EVD concluded that ETS was safe and not correlated with higher ICP levels for most patients; this finding suggests that endotracheal stimulation and coughing are related to ICP peaks rather than suctioning (Olson, McNett, et al., 2013). An observational study and an RCT concluded that aerosolized lidocaine safely and effectively prevents ICP peaks that correlate with ETS. These studies included a mixed critical-care population and were not specific to patients with neurological impairment or with aSAH (Bilotta et al., 2008; Mathieu et al., 2013).

Recommendation

- ETS is safe to perform in short intervals for patients with aSAH, when clinically indicated (strong recommendation, moderate-quality evidence).

HOB elevation

A systematic review with a meta-analysis concluded that HOB elevation decreases ICP after craniotomy (Jiang et al., 2015). HOB elevation also is related to ICP reduction in patients with aSAH (Kim et al., 2014) and ambulatory neurosurgical patients (L. G. Petersen, Petersen, Andresen, Secher, & Juhler, 2016). The benefit of elevating the HOB also was evident in children with neurological impairment (Agbeko, Pearson, Peters, McNames, & Goldstein, 2012). One observational study concluded that this intervention also improves CPP (Ledwith et al., 2010) and reduces the effect of positive end-expiratory pressure on cerebral hemodynamics in patients requiring mechanical ventilation (Schulz-Stubner & Thiex, 2006). In patients with vasospasm, HOB elevation is safe and does not affect cerebral blood flow (CBF; Blissitt, Mitchell, Newell, Woods, & Belza, 2006; Kim et al., 2014; Kung et al., 2013; Ledwith et al., 2010; Zhang & Rabinstein, 2011). Petersen et al. (2016) found that HOB elevation is related to ICP reduction in patients with aSAH and ambulatory neurosurgical patients.

Recommendation

- Elevate the HOB to between 30 degrees and 45 degrees (strong recommendation, moderate-quality evidence).

How do physiotherapy and early ambulation affect cerebral hemodynamics?

Physiotherapy

Physiotherapy is an important intervention for critically ill, neurologically impaired patients. Few studies have examined the effects of bedside physical therapies on cerebral hemodynamics, and no studies have evaluated its influence on long-term outcomes. Two observational studies in a neurocritical care setting concluded that passive range of motion exercises did not result in significant changes in ICP, CPP, or brain tissue oxygenation, even in the setting of intracranial hypertension (Roth et al., 2013; Roth et al., 2017). Another retrospective study of 80 patients concluded that mild physical therapy as tolerated is safe for patients with aSAH after the aneurysm is secured and may be associated with a lower risk for symptomatic vasospasm (Riordan et al., 2015).

Recommendation

- Physiotherapy with passive range of motion or mild exercise is acceptable as clinically tolerated (weak recommendation, low-quality evidence).

Early ambulation

A 2013 Cochrane review concluded that there is insufficient evidence to recommend ambulation prior to coiling or clipping of an aneurysm, particularly when the risk for rebleeding is highest (Ma, Wang, & Liu, 2013). This review found no RCTs evaluating early vs. delayed mobilization for patients with aSAH. The identified studies dated as far back as 1960 and primarily reported the incidence and prevalence of morbidity and mortality after hemorrhage. However, data show that ambulation after aneurysm repair is safe, is feasible, and may be associated with improved ambulation at discharge (Karic, Roe, Nordenmark, Becker, & Sorteberg, 2016; Karic, Sorteberg, Haug Nordenmark, Becker, & Roe, 2015; Olkowski et al., 2013). Among elderly patients with aSAH, those who participated in an early ambulation program had better clinical outcomes as measured by GOS scores 30 days after discharge (Shimamura, Matsuda, Satou, Nakano, & Ohkuma, 2014).

Recommendation

- Early ambulation after aneurysmal repair is recommended as tolerated when clinically indicated (strong recommendation, moderate-quality evidence).

Circulation

How do euvolemia and hypervolemia influence hemodynamic response for patients with aSAH?

Triple H therapy (hypertension, hypervolemia, hemodilution) has been the cornerstone treatment for vasospasm occurring during the days and weeks after aSAH. Over time, prophylactic administration of hypertension, hypervolemia, or both became commonplace to prevent vasospasm. However, a growing body of literature suggests triple H therapy is an oversimplification of a complex interplay of variables and may not be associated with improved outcomes after aSAH.

Several studies have evaluated the role of fluid status in overall outcomes after aSAH and in vasospasm prevention. Two trials evaluated the impact of overall fluid status on outcomes after aSAH. One retrospective study of 356 patients with aSAH assessed fluid status at hospital day 3 and found that patients with positive fluid balance experienced no difference in mortality or new stroke development, but they had a higher occurrence of vasospasm and longer LOS than those with negative or neutral fluid balance (Martini et al., 2012). A second prospective observational trial of 288 patients found that positive fluid balance was linked to cardiopulmonary complications including pulmonary edema and left ventricular dysfunction. In this trial, hypervolemia was independently associated with poor functional outcomes (Kissoon et al., 2015). Three RCTs evaluated the role of hypervolemia or hypervolemia and hypertension early in the care of aSAH to prevent vasospasm (Egge et al., 2001; Lennihan et al., 2000; Togashi et al., 2015). These trials identified no decrease in vasospasm occurrence and worse neuropsychiatric outcomes in patients with augmented blood pressure in one study. Two observational studies suggest a poor correlation between overall fluid status and circulating blood volume (Hoff, van Dijk, Algra, Kalkman, & Rinkel, 2008; Joffe, Khandelwal, Hallman, & Treggiari, 2015).

Optimal CPP and CBF also is a source of debate when caring for patients with aSAH. Two retrospective studies used a calculated pressure reactivity index (PRx) to analyze actual and optimal CPP in patients with aSAH

(Bijlenga et al., 2010; Rasulo et al., 2012). Both studies concluded that elevated PRx values and increased time with suboptimal CPP were associated with worse outcomes. However, the optimal CPP target in patients with aSAH has yet to be defined and may vary depending on the patient and his or her vasospasm status. Fluid management for patients with aSAH should encompass a number of factors such as aneurysm status (secured or unsecured), vasospasm, DCI, cardiac function, cerebral salt wasting, syndrome of inappropriate antidiuretic hormone, and diabetes insipidus (Lo, Mendelow, Sacco, & Wong, 2015).

Recommendations

- Support euvolemic fluid status to optimize cerebral perfusion and oxygenation and minimize secondary insults (strong recommendation, moderate-quality evidence).
- Avoid prophylactic hypervolemic therapy (strong recommendation, high-quality evidence).

Venous Thromboembolism (VTE) Prophylaxis in aSAH

What is the comparative effectiveness of pharmacological prophylaxis vs. mechanical prophylaxis on inpatients with aSAH?

Following aSAH, patients often are limited by periods of decreased mobility that necessitate close monitoring for thromboembolic events and routine prophylactic interventions. In a retrospective analysis of 15,968 patients with aSAH, the overall rate of VTE (deep vein thromboembolism [DVT] or pulmonary embolism) was 4.4% (Kshettry et al., 2014; Salary et al., 2007), and VTE was associated with cardiopulmonary infection, ventriculostomy complications, and vasospasm. These complications nearly doubled LOS. Factors associated with higher risk for venous thromboembolic events included age older than 40, black race, male gender, a teaching hospital setting, congestive heart failure, coagulopathy, neurologic disorders, paralysis, fluid and electrolyte disorders, obesity, and weight loss (Kshettry et al., 2014).

Pharmacological prophylaxis by means of anticoagulation raises bleeding risk concern in the setting of acute intracranial hemorrhage. Studies evaluating the safety of prophylactic subcutaneous low-molecular weight heparin (LMWH) and unfractionated heparin (UFH) more than 24 hours after aneurysm obliteration have revealed infrequent bleeding complications or heparin-induced thrombocytopenia (Manoel et al., 2014; Olson, Bandle, & Calvo, 2015). In a retrospective review of 522 neurosurgical patients, UFH administration during the postoperative period was safe for patients with aSAH and did not appear to contribute to postoperative hemorrhage (Hacker et al., 2012). Risk for hemorrhage associated with the placement, management, and discontinuation of an EVD in patients with aSAH on pharmacological prophylaxis for DVT is another concern. A single-center retrospective study of 241 patients found a significantly reduced rate of DVT (7.5% vs. 18%), with low rates of ventriculostomy-associated hemorrhage, for patients receiving adjuvant chemo prophylaxis versus solitary sequential compression device (SCD) therapy (Zachariah et al., 2016).

Further research may support appropriate screening guidelines for the use of pharmacological prophylaxis. Many of the trials on pharmacological prophylaxis measured safety over efficacy, and it is worth noting that pharmacological agents often were used in conjunction with intermittent pneumatic compression (IPC) devices. No head-to-head studies comparing UFH and LMWH in aSAH have been conducted. Of note, LMWH has been associated with more bleeding than UFH (Siironen et al., 2003). Other UFH advantages include a shorter half-life and the ability to completely reverse its effect with protamine sulfate (Collen, Jackson, Shorr, & Moores, 2008; Manoel et al., 2014; Qaseem, Chou, Humphrey, Starkey, & Shekelle, 2011).

Mechanical prophylaxis by means of IPC devices combats VTE by reducing venous stasis and stimulating fibrinolytic activity by means of compressed air, which intermittently inflates and enhances venous return. ICP contraindications include severe CHF, severe skin problems on legs, severe peripheral vascular disease, and end-of-life status. IPCs may be removed 30 days after stroke if adverse events develop, hospital discharge occurs, or patients become independently mobile (Gould et al., 2012; Kinnier et al., 2016; Makic, Rauen, Watson, & Poteet, 2014).

Generally speaking, IPC devices are recommended in favor of anticoagulation prior to aneurysm occlusion, although potential bleeding complications must be considered. Neurocritical Care Society (NCS) guidelines (Nyquist et al., 2016) advocate for immediate initiation of IPC devices, but more data are needed to support the influence of timeliness on outcomes and efficacy. The American Association of Critical-Care Nurses (AACN) directs nurses to ensure these devices are in use at all times except during skin assessments and bathing ("Preventing Venous Thromboembolism in Adults," 2016). Recommendations from AHA/ASA (Connolly et al., 2012), NCS (Nyquist et al., 2016), the European Stroke Organization (Steiner et al., 2013), and the Canadian Stroke Best Practice Recommendations (Casaubon et al., 2015) clinical practice guidelines address the initiation of VTE prophylaxis for patients with aSAH.

Studies suggest that the application and maintenance of IPC devices at the bedside are notably inconsistent, although the effects on outcomes have not been studied.

In a prospective observational study investigating the use of IPC for patients in the ICU, user errors were observed in 49% of cases (Elpern, Killeen, Patel, & Senecal, 2013). The most common error included misapplication of the devices; of note, 15% of patients prescribed IPCs never received the therapy. Researchers recommend adequate training and routine surveillance of clinical performance (Dunn & Ramos, 2017; Elpern et al., 2013; H. Kim & Lee, 2015; Ritsema, Watson, Stiteler, & Nguyen, 2013). AACN and the American College of Chest Physicians have emphasized the importance of proper IPC device use by issuing practice alerts (Arabi et al., 2013; Makic et al., 2014).

Considering that mechanical prophylaxis is an effective intervention, nurses should ensure proper application and routine monitoring of IPC therapy, which includes measurement of leg circumference and selection of appropriate sleeves that fit and optimize comfort. Vigilance is needed to reapply devices after procedures, baths, transportation, etc. Education that emphasizes comprehension and promotes adherence should be provided to patients and their families (Dunn & Ramos, 2017). In some cases, multimodal prophylaxis will consist of both chemical and mechanical therapies. Nurses should ensure that both therapies are properly administered to most effectively reduce VTE risk.

Recommendations

- UFH and LMWH, sequential compression devices, and other appropriate measures should be employed to prevent DVT in patients with aSAH (strong recommendation, moderate-quality evidence).
- LMWH or UFH for prophylaxis should be withheld for patients with unprotected aneurysms who are expected to undergo surgery (strong recommendation, moderate-quality evidence).
- UFH for prophylaxis may be initiated 24 hours after aneurysm obliteration or in the setting of angiogram-negative aSAH (strong recommendation, moderate-quality evidence). Additional research is needed to understand how to tailor pharmacoprophylaxis to the management of EVDs in patients with aSAH.
- IPC devices should be routinely used at all times except when performing skin assessments and hygiene or during ambulation (strong recommendation, high-quality evidence).
- Careful attention should be paid to proper fit and use of IPCs (weak recommendation, low-quality evidence).

Nursing Care for Patients Undergoing Endovascular Therapy

What are the nursing care considerations regarding endovascular therapy for patients with aSAH?

Endovascular embolization has become a mainstay of aneurysm treatment during the last 30 years. More recently, endovascular flow diversion devices have offered a less invasive approach to aneurysm management. Also, endovascular procedures can counteract cerebral vasospasms. Vasospasm, DCI, and subsequent infarction are among the most feared complications for patients with aSAH because they can contribute to long-term disability. Angiographic evidence of vasospasm occurs in as many as 70% of patients with aSAH, whereas DCI occurs in about 32% to 46% of patients. Symptomatic vasospasm, the most common cause of DCI, usually develops 7 to 8 days after aSAH and warrants prevention, monitoring, and anticipation of corrective measures (Connolly et al., 2012).

Early detection of vasospasm and DCI is crucial to ensure prompt intervention. Follow-up communication that involves reporting of clinical deterioration despite first-line efforts to augment blood pressure in the setting of clinical vasospasm is equally important. Common symptoms of vasospasm include decreased level of consciousness, and new-onset focal neurological deficits may indicate symptomatic vasospasm. Other nonspecific behaviors reported by bedside nurses include restlessness, impulsivity, and other atypical behaviors that correlate with vasospasm diagnoses (Doerksen & Naimark, 2006).

Postintervention nursing considerations include an understanding of postprocedural hemodynamic parameters, anesthesia or sedation, medications and devices, blood vessels traversed and involved, procedural complications, and blood loss. In addition to neurological checks, frequent vital signs, site assessments, and distal pulse checks should be performed to assess for active bleeding, hematoma, or vascular complications. Specific procedural complications that include iatrogenic dissection and stroke, insertion-site hematoma, intracranial hemorrhage, and allergic reactions also should be considered while caring for patients after intra-arterial treatment or an aneurysm or vasospasm (Bautista, 2012; Froehler & Geocadin, 2007). Many nursing management practices for patients undergoing neuro-endovascular procedures are borrowed from cardiac catheterization traditions and lack rigorous evaluation. More research is needed to most effectively operationalize nursing monitoring and management of complications that occur during the perioperative endovascular period.

Recommendation

- Care for postintervention patients should include frequent vital sign monitoring; neurologic and endovascular site assessments; distal pulse checks; and monitoring for procedural complications including bleeding, hematoma, vascular complications, stroke, and signs of allergic reaction (weak recommendation, low-quality evidence).

Nutritional Support and Glycemic Control

What is the effect of enteral vs. parenteral nutrition on glycemic control and prevention of HACs for patients with aSAH?

The optimal method for delivering early nutritional support is the subject of an ongoing debate in the field of care for critically ill patients. Several studies address nutritional support during acute hospitalization after aSAH. Some evidence shows that patients with aSAH are undernourished and that negative energy balance is associated with outcomes. A single-center prospective observational study of 58 patients with aSAH receiving enteral nutrition found that even though enteral feeding commenced early during the hospital admission, most patients maintained a negative energy balance (Badjatia et al., 2015; Jaja et al., 2013). Patients who had a higher average negative energy balance were more likely to experience infections but not other complications. Cumulative energy balance did not correlate with overall mortality. A follow-up single-center prospective observational study of 229 patients found negative energy balance to be common and associated with infections during the first 2 weeks of hospitalization (Lantigua et al., 2015; Milinis et al., 2017). A single-center retrospective study of patients with severe aSAH and ventilator-acquired pneumonia (VAP) revealed that delayed enteral feeding was associated with higher rates of VAP (Cinotti et al., 2014). Although these studies outline an association between undernutrition and infection, little existing evidence guides optimal nutritional support for this population.

If oral enteral nutrition is ordered, an assessment is needed to prevent aspiration pneumonia. aSAH can affect patients' mechanical and cognitive ability to swallow properly, and this can lead to aspiration. Although the Canadian Stroke Best Practice Recommendations, AHA/ASA, European Stroke Organization, and accreditation and certification bodies do not address the effects of dysphagia in aSAH guidelines, the effects of dysphagia-related HACs cannot be dismissed. Study findings in the general stroke population demonstrate that dysphagia is associated with infection, which influences morbidity and mortality and LOS; a dysphagia screen may prevent these problems (Sellars et al., 2007; Titsworth et al., 2013; Yuan et al., 2015). One single-center retrospective study of 360 consecutive patients with aSAH found that 18% of patients were discharged with a gastric feeding tube because of a swallowing impairment (Abecassis et al., 2017). Another single-center retrospective study of 64 patients with aSAH undergoing a videofluoroscopic swallowing study revealed that nearly 50% of participants had oral phase abnormalities and delayed oral transit times—a higher rate than found in other stroke populations (Rhie et al., 2016). Although more studies of swallow dysfunction in the aSAH population are warranted, existing study findings suggest that dysphagia assessment is an important component of nursing care for patients with aSAH.

Although in-hospital hyperglycemia and glucose variability is associated with vasospasm, infection, worse functional outcomes, and mortality in patients with aSAH, no randomized data exist to guide aggressive management of hyperglycemia (Acosta Escribano, Herrero Meseguer, & Conejero Garcia-Quijada, 2011; Barletta, Figueroa, DeShane, Blau, & McAllen, 2013; L. Bian et al., 2013; Kurtz et al., 2014; L. Y. Lin, H. C. Lin, Lee, Ma, & Lin, 2007; Okazaki et al., 2018; Pasternak et al., 2008; Schlenk, Vajkoczy, & Sarrafzadeh, 2009; van Donkelaar et al., 2016). Several single-center studies suggest that plasma and cerebral glucose levels may not always correlate. This lack of correlation warrants further investigation of the role of tight glucose control and the interplay between nutritional support and serum and cerebral glucose levels.

Data on glucose control targets in mixed neurosurgical patient populations and the aSAH population are varied. A meta-analysis of mixed neurosurgical populations revealed that tight glycemic control was associated with lower postoperative infection rates (Ooi et al., 2012). Latorre et al. (2009) reported that good glycemic control (regardless of aggressive or standard management protocols) was associated with fewer poor outcomes (Latorre et al., 2009). One prospective study found that conventional glycemic control was associated with higher infection rates than aggressive glycemic control (Bilotta et al., 2007). Other studies revealed that aggressive glycemic control was not associated with improved mortality rates (Bilotta et al., 2007; Kramer, Roberts, & Zygun, 2012; Thiele et al., 2009; Ling, Li, & Gao, 2012). Several studies have shown that tight glycemic control is associated with serum and cerebral hypoglycemia and that energy imbalances are associated with poor outcomes (Badjatia et al., 2010; Badjatia et al., 2015; Kramer et al., 2012; Ling et al., 2012; Naidech et al., 2010; Prakash & Matta, 2008; Schmutzhard & Rabinstein, 2011). More research is needed to determine the ideal target for aggressive glucose control in patients with aSAH.

Recommendations

- Hypoglycemia must be corrected (strong recommendation, moderate-quality evidence).
- Dysphagia assessment should precede administration of oral nutrition and medication therapies (weak recommendation, low-quality evidence).
- Nutritional support should start as soon as clinically possible and meet the patient's energy needs (weak recommendation, low-quality evidence).

Pain Management

How do adjunctive pain management therapies affect efficacy, safety, and cerebral hemodynamics for patients with aSAH?

Most patients hospitalized with H & H grades 1, 2, or 3 aSAH (73%) describe severe headache lasting approximately 15 days, and 74% to 81% of patients with aSAH with angiogram-negative aSAH experience severe headaches (Morad, Tamargo, & Gottschalk, 2016; Glisic et al., 2016; Dorhout Mees, Bertens, van der Worp, Rinkel, & van den Bergh, 2010; Asadi-Noghabi, Gholizadeh, Zolfaghari, Mehran, & Sohrabi, 2015). According to one single-center prospective database study, the most commonly reported pain site is the head (76%), while neck and back pain (15%), limb and joint pain (15%), and eye pain also are reported (Morad et al., 2016). Long-term follow-up data indicate that headache may persist after aSAH for 2 to 9 years. In one study, headache was the second-leading cause of 30-day hospital readmission after aSAH (Glisic et al., 2016). Limited data exist on the course of headache after aSAH and pharmacological management. Researchers postulate that pain with aSAH occurs because of inflammatory by-products of hemolysis, which results in meningeal irritation in the subarachnoid space. Other investigators contend central pain sensitization mediated by N-methyl-D-aspartate receptors leads to hyperalgesia (Dorhout Mees et al., 2010; Glisic et al., 2016).

Because patients with aSAH often are limited by decreased consciousness or other physiological problems, they may be unable to report pain verbally. Nursing assessment should include behavioral measures that identify nonverbal pain indicators because poorly managed pain in patients with aSAH can lead to agitation or exacerbate hypertension and may elevate ICP—all of which increase the risk for aneurysm rebleeding. Pain assessments in nonverbal, critically ill patients should include observation of facial expressions, body movements, muscle tension, and ventilator compliance (Arif-Rahu & Grap, 2010; Asadi-Noghabi et al., 2015; Lindberg & Engstrom, 2011; Puntillo et al., 2004).

Few studies have explored the management of severe pain in aSAH. A single-center database study by Morad and colleagues found that pain persists despite substantial acetaminophen (APAP) and opioid use (Mocco et al., 2006; Morad et al., 2016). An international practice audit of pain management in neurocritical care units in France, the United States, Australia, and New Zealand found that APAP/paracetamol is the most common first-line pain management agent, with opioids commonly the second-line agent. Third-line use of gabapentin/pregabalin was reported specific to patients with aSAH (Zeiler, AlSubaie, Zeiler, Bernard, & Skrobik, 2016). No studies explored strategies to optimize pain management in the aSAH population.

Pain in the aSAH setting should be anticipated, assessed, and treated with consideration of the patient's level of consciousness and hemodynamic status. Pharmacologic pain interventions may help reduce systemic and intracranial hypertension, but analgesia may be a delicate matter for patients with hypotension. Pain and pain management are not addressed in AHA or NCS guidelines on aSAH; however, European guidelines advocate for the judicious use of analgesics in patients with aSAH (Steiner et al., 2013).

As a nonopioid agent, APAP is considered safe for aSAH treatment. Both oral and intravenous (IV) APAP preparations have been studied for onset and efficacy for patients with aSAH. In a retrospective study of 309 neurocritical care patients (involving 899 doses of APAP), the IV preparation yielded significantly faster (pain intensity difference 3 vs. 0 at 30 minutes post administration; $p < .003$) analgesic effects than oral APAP, but efficacy at 1 and 6 hours after administration was equal (Nichols, Nadpara, Taylor, & Brophy, 2016). Data on clinical outcomes remain unknown. Considering the cost difference between IV and oral APAP, it may be reasonable to reserve IV preparations for patients who need rapid pain control (Morad et al., 2016; Nichols et al., 2016).

Opiates may be used for analgesia and in combination with sedative-hypnotic agents to stabilize ICP. The cerebral physiological effects of opioids on patients in the neuro-ICU have been controversial, and hemodynamic consequences should be anticipated. Patients with aSAH and impaired autoregulation may be at further risk for dose-dependent decreases in mean arterial pressure (MAP), which may in turn decrease CPP and pose risk for DCI. In patients with neurological trauma, transient ICP elevations of up to 8 mmHg have been observed with the administration of IV fentanyl and sufentanil, with subsequent decreases in MAP and CPP by an average of 10 mmHg. These effects were witnessed to resolve after 15 minutes and did not involve cerebral ischemia (Rhoney & Parker, 2001). Other studies have similarly found that morphine and fentanyl yield mild increases in ICP and decreases in MAP without an effect on CBF for patients who experience neurotrauma (Keegan, 2008; Oddo et al., 2016; Rhoney & Parker, 2001). Further research involving patients with aSAH is needed to understand the impact of opioids on CBF.

Drug selection should involve consideration of potential adverse effects such as decreased level of alertness, hypotension, respiratory depression, and gastrointestinal dysmotility (Dhakal, Harriott, Capobianco, & Freeman, 2016). Opiate use raises concerns about potential masking of neurological exam findings, and caution should be exercised while acknowledging potential effects on neurology checks and side effects such as excessive sedation and delirium. The best analgesic agents for patients in the neuro-ICU should be short-acting and titratable with limited hemodynamic effects (Karabinis et al., 2004; B. S. Paul & Paul, 2013; Rhoney & Parker, 2001).

Nonsteroidal anti-inflammatory drugs generally are withheld during the acute phase of aSAH because of their antiplatelet effects. Although data are limited, potential complications include bleeding and cardiovascular effects. Other adjunctive agents include gabapentin, which enhances GABAergic signaling and modulates calcium channels. Gabapentin appears to be well tolerated and can be administered safely to patients with aSAH. More data are needed to test the efficacy of gabapentin on pain management. Limited preliminary data show that it can decrease opiate analgesic requirements (Dhakal et al., 2015; Zeiler et al., 2016). Triptans and serotonin 1B/1D receptor agonists, which commonly are used for migraine treatment, have been identified as effective analgesics but should be avoided because of the risk for vasoconstriction and vasospasm (Dhakal et al., 2016).

Nursing research has explored complementary nonpharmacological pain therapies, but high-quality data supporting the use of specific interventions, especially for critically ill people, are limited, and outcomes data are not available. One adjuvant therapy incorporates sleep quality because inadequate sleep has been linked to decreased immunity, healing, and pain tolerance and other physiological problems. Nurses can help reduce sleep disruption by controlling noise, adjusting lighting, adhering to quiet-time protocols, and thoughtfully timing routine care such as laboratory tests, imaging, electrocardiograms, and hygiene (Tracy & Chlan, 2011). Because of the critical state of many patients with aSAH, physical assessments including vital signs and ventilator management should be performed as scheduled and more frequently if clinically indicated. Noise reduction with the use of ear plugs has been associated with improved sleep for volunteers exposed to simulated ICU noises in a laboratory setting, and some researchers noted longer sleep duration of 1 hour for patients who received a brief back massage (Sessler, Grap, & Brophy, 2001). Multidisciplinary pain control approaches such as addressing underlying physiological needs and managing analgesia and sedation to foster a healing environment are recommended to enhance patient comfort.

Recommendations

- Pain assessments that take into account nonverbal behaviors should be performed with every vital sign check for all patients including those who are nonverbal, unconscious, cognitively impaired, or require mechanical ventilation (weak recommendation, low-quality evidence).
- APAP and opiates are preferred agents for headache treatment (Good Practice Statement).
- Opiate selection should involve consideration of potential onset, duration, titratability, and side effects including effects on hemodynamics and neurological status (Good Practice Statement).
- A healing environment may be created with nonpharmacological measures that promote adequate sleep, reduce excessive noise, and involve appropriate use of lighting and conscientious coordination of routine care (Good Practice Statement).

The Role of Seizure Prophylaxis in aSAH

The incidence of seizure after aSAH varies from 8% to 35% and is more common in the setting of ruptured anterior circulation aneurysms, ICH, thicker cisternal clots, rebleeding, ischemic infarct, and history of hypertension (Bhalla et al., 2016; Claassen et al., 2013). Seizure frequently is observed at the time of aneurysm rupture rather than during hospitalization and may be a symptom or a cause of rebleeding for an untreated aneurysm (Lanzino, D'Urso, Suarez, & Participants in the International Multi-Disciplinary Consensus Conference on the Critical Care Management of Subarachnoid Hemorrhage, 2011). Adverse effects associated with seizure can include hypertension, lactic acidosis, hyperthermia, respiratory compromise, pulmonary aspiration or edema, rhabdomyolysis, self-injury, and irreversible neurological damage, especially if effects last longer than 30 minutes; seizure with aSAH is associated with an unfavorable prognosis (Wagner, Johnson, & Hardin-Pierce, 2011). If the seizure occurs prior to aneurysm treatment, hyperemia may increase the risk for aneurysm rerupture.

Traditionally, patients with aSAH were started on anticonvulsant (antiepileptic drug [AED]) therapy upon admission and for months or years afterward. In a retrospective review of 353 patients with aSAH, 10% sustained clinical and electrographic seizures that most often occurred within 24 hours of the event. These investigators found that AED therapy did not significantly decrease seizure activity (Panczykowski et al., 2016). However, a 2000 retrospective cohort study (Butzkueven et al., 2000), a 2005 prospective observational study (Naidech et al., 2005), and a 2007 meta-analysis (Rosengart, Schultheiss, Tolentino, & Macdonald, 2007) of several RCTs suggested that patients treated with antiseizure drugs had more complications and no difference in seizure risk. This was of particular concern for patients receiving phenytoin, a common AED for inpatient seizure prophylaxis. Anticonvulsant therapy is independently associated with worse outcomes and may increase inpatient complications (Lo, Mendelow, Sacco, & Wong, 2015; Naidech et al., 2005; Rosengart et al., 2007). The benefits of prophylactic AEDs have not been well established, and the association between seizures and functional outcomes is not well known. Some studies have demonstrated no impact of seizure on clinical outcomes; however, status epilepticus is a strong predictor of poor outcomes (Butzkueven et al., 2000). In 2007, a retrospective review of 453 patients evaluated the truncation of AED duration in aSAH to 3 days after hospital admission with aSAH compared to administration throughout the hospitalization. Investigators found no difference in seizure rates and fewer adverse events with shorter AED prophylaxis (Chumnanvej, Dunn, & Kim, 2007). Also, many practitioners have moved away from the use of phenytoin for seizure prophylaxis and have incorporated other AEDs. Another retrospective analysis of 442 patients revealed more in-hospital seizures among patients treated with a 3-day course of seizure prophylaxis compared to a 7-day course (Murphy-Human, Welch, Zipfel, Diringer, & Dhar, 2011), and a follow-up RCT suggested a trend toward fewer in-hospital seizures among patients treated with a 7-day course of levetiracetam instead of a 3-day course, although this trial was too underpowered to draw a firm conclusion (Human et al., 2017).

Nonconvulsive seizures, for which activity only can be diagnosed by electroencephalogram (EEG), have been reported in about 13% of patients with aSAH (Claassen et al., 2013). Electrographic seizures have been observed in patients demonstrating symptoms of diminished alertness, unresponsiveness, nystagmus, facial twitching, confusion, blank staring, aphasia, humming while unresponsive to verbal commands, automatisms (lip smacking, fumbling with fingers) and childlike behaviors. Diagnoses often are delayed, and assessment for nonconvulsive seizure activity should be included in routine neurology checks because such findings are linked to higher mortality (Young, Jordan, & Doig, 1996).

Continuous EEG monitoring may be indicated for patients with aSAH with unexplained and persistent altered consciousness to evaluate for nonconvulsive seizure activity because sporadic EEGs may not capture abnormal electrical activity (Claassen et al., 2013; Young et al., 1996). For patients with aSAH who are in a coma, continuous EEG monitoring provides objective information on cerebral function and response to stimuli and treatments. Continuous monitoring is indicated for patients with aSAH who are comatose, yet the effects of treatment for these patients are less clear. Abnormal EEG patterns such as slower frequencies have been detected prior to episodes of DCI; further studies may suggest EEG utility for ischemia detection (Claassen et al., 2013; Young et al., 1996). Because many nonconvulsive seizures go unrecognized, nurses are needed to detect subclinical seizure activity and initiate neuroprotective seizure control. Nurses who recognize ischemic electrographic patterns for patients with aSAH can help promote appropriate treatment plans for patients at risk for DCI and infarction.

Recommendations

- Seizure prophylaxis prior to aneurysm treatment may be considered (Good Practice Statement). There is insufficient evidence to generate recommendations on optimal agents and duration of therapy.
- Routine neurological checks should include an assessment of nonconvulsive and other seizure activity (weak recommendation, low-quality evidence).

Thermodynamics

Does maintaining normothermia improve outcomes?

No RCTs examining the effects of normothermia on aSAH outcomes are available in the literature. A number of studies concluded that hyperthermia (body temperature >38.3 °C) is associated with increased DCI, poorer functional outcomes, increased cerebral metabolic distress, and mortality (Douds et al., 2012; Kramer, Pegoli, Mandrekar, Lanzino, & Rabinstein, 2017; Springer et al., 2009; Suehiro et al., 2016; Todd et al., 2009; G. Zhang, Zhang, & Qin, 2011). In a small retrospective study of patients with aSAH undergoing microdialysis, induced normothermia decreased cerebral metabolic distress in patients with refractory fever (Oddo et al., 2009). One small retrospective study and a second small prospective cohort study found that aggressive fever control, defined by the addition of external (gel wraps, circulating blankets) or intravascular temperature–modulating devices adjuvant to traditional APAP every 4 to 6 hours, was associated with a better mRS of 3 or lower at 6 and 12 months, respectively (Badjatia et al., 2010; Fischer et al., 2015). No well-designed trial has evaluated the target temperature, duration, or mode of providing normothermia in patients with aSAH. Regardless, the AHA/ASA (Connolly et al., 2012) and the Canadian Stroke Best Practice Recommendations (Casaubon et al., 2015) advocate normothermia.

The effects of therapeutic hypothermia (TH) for neurological protection after aSAH are considerably less definitive than the benefits documented after cardiac arrest; TBI; and, to a lesser extent, acute ischemic stroke. The use of TH for neurological protection after aSAH warrants further investigation. The largest RCT studying intraoperative hypothermia for aSAH revealed that intraoperative hypothermia did not provide benefits compared to normothermia (Anderson et al., 2006; Hindman, Bayman, Pfisterer, Torner, & Todd, 2010; Todd, Hindman, Clarke, & Torner, 2005). Further research on TH during hospital stays also was inconclusive. In one study, TH was found to decrease the severity of macrovascular spasm and DCI in poor-grade hemorrhage (Kuramatsu et al., 2015). In this same study, marked and only marginally insignificant differences (66.7 vs. 33.3%, $p = 0.06$) in favorable outcomes (mRS < 3) for the hypothermic group were documented (Kuramatsu et al., 2015). Another small institutional study by Seule et al. (2014) documented a decrease in CBF velocities in patients treated with hypothermia. Francoeur and Mayer (Francoeur & Mayer, 2016) classify TH as a second-tier rescue therapy based on the research of Seule and Nagao (Nagao et al., 2003; Seule et al., 2014). Two small studies concluded that TH does not influence patient outcomes (Anei, Sakai, Iihara, & Nagata, 2010; Karnatovskaia, Lee, Festic, Kramer, & Freeman, 2014), but outcomes were limited to discharge or 3-month mRS and did not account for possible longer-term benefits seen in normothermia studies.

Recommendation

- Measures to promote normothermia after aSAH are recommended (weak recommendation, low-level evidence).

References

Abdullah, J. M., & Husin, A. (2011). Intravascular hypothermia for acute hemorrhagic stroke: A pilot study. *Acta Neurochir Suppl, 111*, 421-424. doi: 10.1007/978-3-7091-0693-8_72

Abecassis, I. J., Morton, R. P., McGrath, L., Hanson, J., Xue, A. H., Kelly, C. M., . . . Kim, L. J. (2017). Respiratory and swallowing outcomes based on aneurysm location in 360 patients with subarachnoid hemorrhage. *World Neurosurg, 105*, 108-114. doi: 10.1016/j.wneu.2017.05.110

Acosta Escribano, J., Herrero Meseguer, I., & Conejero Garcia-Quijada, R. (2011). Guidelines for specialized nutritional and metabolic support in the critically-ill patient: Update. Consensus SEMICYUC-SENPE: Neurocritical patient. *Nutr Hosp, 26 Suppl 2*, 72-75. doi: 10.1590/s0212-16112011000800016

Agbeko, R. S., Pearson, S., Peters, M. J., McNames, J., & Goldstein, B. (2012). Intracranial pressure and cerebral perfusion pressure responses to head elevation changes in pediatric traumatic brain injury. *Pediatr Crit Care Med, 13*(1), e39-47. doi: 10.1097/PCC.0b013e31820ac2ad

Al-Tamimi, Y. Z., Bhargava, D., Feltbower, R. G., Hall, G., Goddard, A. J., Quinn, A. C., & Ross, S. A. (2012). Lumbar drainage of cerebrospinal fluid after aneurysmal subarachnoid hemorrhage: A prospective, randomized, controlled trial (LUMAS). *Stroke, 43*(3), 677-682. doi: 10.1161/STROKEAHA.111.625731

Amato, A., Britz, G. W., James, M. L., Graffagnino, C., Zomorodi, A. R., Zomorodi, M. E., & Olson, D. M. (2011). An observational pilot study of CSF diversion in subarachnoid haemorrhage. *Nurs Crit Care, 16*(5), 252-260. doi: 10.1111/j.1478-5153.2010.00444.x

American Cancer Society. (2014). A guide to palliative or supportive care. Retrieved April 11, 2018.

Anderson, S. W., Todd, M. M., Hindman, B. J., Clarke, W. R., Torner, J. C., Tranel, D., . . . Samra, S. (2006). Effects of intraoperative hypothermia on neuropsychological outcomes after intracranial aneurysm surgery. *Ann Neurol, 60*(5), 518-527. doi: 10.1002/ana.21018

Anei, R., Sakai, H., Iihara, K., & Nagata, I. (2010). Effectiveness of brain hypothermia treatment in patients with severe subarachnoid hemorrhage: Comparisons at a single facility. *Neurol Med Chir (Tokyo), 50*(10), 879-883.

Arabi, Y. M., Khedr, M., Dara, S. I., Dhar, G. S., Bhat, S. A., Tamim, H. M., & Afesh, L. Y. (2013). Use of intermittent pneumatic compression and not graduated compression stockings is associated with lower incident VTE in critically ill patients: A multiple propensity scores adjusted analysis. *Chest, 144*(1), 152-159. doi: 10.1378/chest.12-2028

Arif-Rahu, M., & Grap, M. J. (2010). Facial expression and pain in the critically ill non-communicative patient: State of science review. *Intensive Crit Care Nurs, 26*(6), 343-352. doi: 10.1016/j.iccn.2010.08.007

Arif, S., Bhat, A. R., Wani, M. A., Raina, T., Ramzan, A., Kirmani, A.,. . . Bari, S. (2012). Infective and non-infective complications of external ventricular drainage. *JK Practitioner, 17*(1-3), 27-32.

Asadi-Noghabi, A. A., Gholizadeh, M., Zolfaghari, M., Mehran, A., & Sohrabi, M. (2015). Nurses use of critical care pain observational tool in patients with low consciousness. *Oman Med J, 30*(4), 276-282. doi: 10.5001/omj.2015.55

Badjatia, N., Fernandez, L., Schlossberg, M. J., Schmidt, J. M., Claassen, J., Lee, K., . . . Rosenbaum, M. (2010). Relationship between energy balance and complications after subarachnoid hemorrhage. *JPEN J Parenter Enteral Nutr, 34*(1), 64-69. doi: 10.1177/0148607109348797

Badjatia, N., Monahan, A., Carpenter, A., Zimmerman, J., Schmidt, J. M., Claassen, J., . . . Seres, D. (2015). Inflammation, negative nitrogen balance, and outcome after aneurysmal subarachnoid hemorrhage. *Neurology, 84*(7), 680-687. doi: 10.1212/WNL.0000000000001259

Barletta, J. F., Figueroa, B. E., DeShane, R., Blau, S. A., & McAllen, K. J. (2013). High glucose variability increases cerebral infarction in patients with spontaneous subarachnoid hemorrhage. *J Crit Care, 28*(5), 798-803. doi: 10.1016/j.jcrc.2012.12.012

Bautista, C. (2012). Unresolved issues in the management of aneurysmal subarachnoid hemorrhage. *AACN Adv Crit Care, 23*(2), 175-185. doi: 10.1097/NCI.0b013e31824ebcfa

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., . . . American Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2017). Heart disease and stroke statistics-2017 update: A report from the American Heart Association. *Circulation, 135*(10), e146-e603. doi: 10.1161/CIR.0000000000000485

Bhalla, D., Lotfalinezhad, E., Timalsina, U., Kapoor, S., Kumar, K. S., Abdelrahman, A., . . . Irmansyah, I. (2016). A comprehensive review of epilepsy in the Arab world. *Seizure, 34*, 54-59. doi: 10.1016/j.seizure.2015.12.002

Bian, L., Liu, L., Wang, C., Hussain, M., Yuan, Y., Liu, G., . . . Zhao, X. (2013). Hyperglycemia within day 14 of aneurysmal subarachnoid hemorrhage predicts 1-year mortality. *Clin Neurol Neurosurg, 115*(7), 959-964. doi: 10.1016/j.clineuro.2012.09.026

Bian, L. H., Liu, Y. F., Nichols, L. T., Wang, C. X., Wang, Y. L., Liu, G. F., . . . Zhao, X. Q. (2012). Epidemiology of subarachnoid hemorrhage, patterns of management, and outcomes in China: A hospital-based multicenter prospective study. *CNS Neurosci Ther, 18*(11), 895-902. doi: 10.1111/cns.12001

Bijlenga, P., Czosnyka, M., Budohoski, K. P., Soehle, M., Pickard, J. D., Kirkpatrick, P. J., & Smielewski, P. (2010). "Optimal cerebral perfusion pressure" in poor grade patients after subarachnoid hemorrhage. *Neurocrit Care, 13*(1), 17-23. doi: 10.1007/s12028-010-9362-1

Bilotta, F., Branca, G., Lam, A., Cuzzone, V., Doronzio, A., & Rosa, G. (2008). Endotracheal lidocaine in preventing endotracheal suctioning-induced changes in cerebral hemodynamics in patients with severe head trauma. *Neurocrit Care, 8*(2), 241-246. doi: 10.1007/s12028-007-9012-4

Bilotta, F., Spinelli, A., Giovannini, F., Doronzio, A., Delfini, R., & Rosa, G. (2007). The effect of intensive insulin therapy on infection rate, vasospasm, neurologic outcome, and mortality in neurointensive care unit after intracranial aneurysm clipping in patients with acute subarachnoid hemorrhage: A randomized prospective pilot trial. *J Neurosurg Anesthesiol, 19*(3), 156-160. doi: 10.1097/ANA.0b013e3180338e69

Blacquiere, D., Bhimji, K., Meggison, H., Sinclair, J., & Sharma, M. (2013). Satisfaction with palliative care after stroke: A prospective cohort study. *Stroke, 44*(9), 2617-2619. doi: 10.1161/strokeaha.113.001992

Blissitt, P. A., Mitchell, P. H., Newell, D. W., Woods, S. L., & Belza, B. (2006). Cerebrovascular dynamics with head-of-bed elevation in patients with mild or moderate vasospasm after aneurysmal subarachnoid hemorrhage. *Am J Crit Care, 15*(2), 206-216.

Brain Aneurysm Foundation. Brain aneurysm statistics and facts. Retrieved April 11, 2018, from https://www.bafound.org/about-brain-aneurysms/brain-aneurysm-basics/brain-aneurysm-statistics-and-facts/

Braun, L. T., Grady, K. L., Kutner, J. S., Adler, E., Berlinger, N., Boss, R., . . . American Heart Association Advocacy Coordinating Committee. (2016). Palliative care and cardiovascular disease and stroke: A policy statement from the American Heart Association/American Stroke Association. *Circulation, 134*(11), e198-225. doi: 10.1161/CIR.0000000000000438

Burton, C. R., & Payne, S. (2012). Integrating palliative care within acute stroke services: developing a programme theory of patient and family needs, preferences and staff perspectives. *BMC Palliat Care, 11*, 22. doi: 10.1186/1472-684X-11-22

Butzkueven, H., Evans, A. H., Pitman, A., Leopold, C., Jolley, D. J., Kaye, A. H., . . . Davis, S. M. (2000). Onset seizures independently predict poor outcome after subarachnoid hemorrhage. *Neurology, 55*(9), 1315-1320.

Camacho, E. F., Boszczowski, I., Basso, M., Jeng, B. C., Freire, M. P., Guimaraes, T., . . . Costa, S. F. (2011). Infection rate and risk factors associated with infections related to external ventricular drain. *Infection, 39*(1), 47-51. doi: 10.1007/s15010-010-0073-5

Casaubon, L. K., Boulanger, J. M., Blacquiere, D., Boucher, S., Brown, K., Goddard, T., . . . Stroke Foundation of Canada Canadian Stroke Best Practices Advisory Committee. (2015). Canadian stroke best practice recommendations: Hyperacute stroke care guidelines, update 2015. *Int J Stroke, 10*(6), 924-940. doi: 10.1111/ijs.12551

Chalouhi, N., Tjoumakaris, S., Thakkar, V., Theofanis, T., Hammer, C., Hasan, D., . . . Jabbour, P. (2014). Endovascular management of cerebral vasospasm following aneurysm rupture: outcomes and predictors in 116 patients. *Clin Neurol Neurosurg, 118*, 26-31. doi: 10.1016/j.clineuro.2013.12.012

Chen, S., Luo, J., Reis, C., Manaenko, A., & Zhang, J. (2017). Hydrocephalus after subarachnoid hemorrhage: Pathophysiology, diagnosis, and treatment. *Biomed Res Int, 2017*, 8584753. doi: 10.1155/2017/8584753

Chumnanvej, S., Dunn, I. F., & Kim, D. H. (2007). Three-day phenytoin prophylaxis is adequate after subarachnoid hemorrhage. *Neurosurgery, 60*(1), 99-102; discussion 102-103. doi: 10.1227/01.NEU.0000249207.66225.D9

Cinotti, R., Dordonnat-Moynard, A., Feuillet, F., Roquilly, A., Rondeau, N., Lepelletier, D., . . . Asehnoune, K. (2014). Risk factors and pathogens involved in early ventilator-acquired pneumonia in patients with severe subarachnoid hemorrhage. *Eur J Clin Microbiol Infect Dis, 33*(5), 823-830. doi: 10.1007/s10096-013-2020-8

Claassen, J., Taccone, F. S., Horn, P., Holtkamp, M., Stocchetti, N., Oddo, M., & Neurointensive Care Section of the European Society of Intensive Care Medicine. (2013). Recommendations on the use of EEG monitoring in critically ill patients: Consensus statement from the neurointensive care section of the ESICM. *Intensive Care Med, 39*(8), 1337-1351. doi: 10.1007/s00134-013-2938-4

Cohen, M. D., & Hilligoss, P. B. (2010). The published literature on handoffs in hospitals: Deficiencies identified in an extensive review. *Qual Saf Health Care, 19*(6), 493-497. doi: 10.1136/qshc.2009.033480

Collen, J. F., Jackson, J. L., Shorr, A. F., & Moores, L. K. (2008). Prevention of venous thromboembolism in neurosurgery: A metaanalysis. *Chest, 134*(2), 237-249. doi: 10.1378/chest.08-0023

Connolly, E. S. Jr., Rabinstein, A. A., Carhuapoma, J. R., Derdeyn, C. P., Dion, J., Higashida, R. T., . . . Council on Clinical Cardiology. (2012). Guidelines for the management of aneurysmal subarachnoid hemorrhage: A guideline for healthcare professionals from the American Heart Association/american Stroke Association. *Stroke, 43*(6), 1711-1737. doi: 10.1161/STR.0b013e3182587839

Creutzfeldt, C. J., Holloway, R. G., & Curtis, J. R. (2015). Palliative care: A core competency for stroke neurologists. *Stroke, 46*(9), 2714-2719. doi: 10.1161/STROKEAHA.115.008224

Crobeddu, E., Mittal, M. K., Dupont, S., Wijdicks, E. F., Lanzino, G., & Rabinstein, A. A. (2012). Predicting the lack of development of delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage. *Stroke, 43*(3), 697-701. doi: 10.1161/STROKEAHA.111.638403

Dengler, N. F., Sommerfeld, J., Diesing, D., Vajkoczy, P., & Wolf, S. (2018). Prediction of cerebral infarction and patient outcome in aneurysmal subarachnoid hemorrhage: Comparison of new and established radiographic, clinical and combined scores. *Eur J Neurol, 25*(1), 111-119. doi: 10.1111/ene.13471

Dhakal, L. P., Harriott, A. M., Capobianco, D. J., & Freeman, W. D. (2016). Headache and Its Approach in Today's NeuroIntensive Care Unit. *Neurocrit Care, 25*(2), 320-334. doi: 10.1007/s12028-016-0260-z

Dhakal, L. P., Hodge, D. O., Nagel, J., Mayes, M., Richie, A., Ng, L. K., & Freeman, W. D. (2015). Safety and tolerability of gabapentin for aneurysmal subarachnoid hemorrhage (sah) headache and meningismus. *Neurocrit Care, 22*(3), 414-421. doi: 10.1007/s12028-014-0086-5

Diebolt, E., Sims, S., Conners, J., & Lee, V. (2014). Stroke patient length of stay benchmarking and analysis (P2.129). *Neurology, 82*(10 Supplement).

Doerksen, K., & Naimark, B. (2006). Nonspecific behaviors as early indications of cerebral vasospasm. *J Neurosci Nurs, 38*(6), 409-415.

Dorhout Mees, S. M., Bertens, D., van der Worp, H. B., Rinkel, G. J., & van den Bergh, W. M. (2010). Magnesium and headache after aneurysmal subarachnoid haemorrhage. *J Neurol Neurosurg Psychiatry, 81*(5), 490-493. doi: 10.1136/jnnp.2009.181404

Dorman Wagner, K., Johnson, K. L., & Hardin-Pierce, M. G. (2011). *High acuity nursing* (5th ed.). Upper Saddle River, NJ: Pearson Education Inc.

Douds, G. L., Tadzong, B., Agarwal, A. D., Krishnamurthy, S., Lehman, E. B., & Cockroft, K. M. (2012). Influence of fever and hospital-acquired infection on the incidence of delayed neurological deficit and poor outcome after aneurysmal subarachnoid hemorrhage. *Neurol Res Int, 2012*, 479865. doi: 10.1155/2012/479865

Duan, G., Yang, P., Li, Q., Zuo, Q., Zhang, L., Hong, B., . . . Huang, Q. (2016). Prognosis predicting score for endovascular treatment of aneurysmal subarachnoid hemorrhage: A risk modeling study for individual elderly patients. *Medicine (Baltimore), 95*(7), e2686. doi: 10.1097/MD.0000000000002686

Dunn, N., & Ramos, R. (2017). Preventing venous thromboembolism: The role of nursing with intermittent pneumatic compression. *Am J Crit Care, 26*(2), 164-167. doi: 10.4037/ajcc2017504

Egge, A., Waterloo, K., Sjoholm, H., Solberg, T., Ingebrigtsen, T., & Romner, B. (2001). Prophylactic hyperdynamic postoperative fluid therapy after aneurysmal subarachnoid hemorrhage: A clinical, prospective, randomized, controlled study. *Neurosurgery, 49*(3), 593-605; discussion 605-596.

Elpern, E., Killeen, K., Patel, G., & Senecal, P. A. (2013). The application of intermittent pneumatic compression devices for thromboprophylaxis: An observational study found frequent errors in the application of these mechanical devices in ICUs. *Am J Nurs, 113*(4), 30-36; quiz 37. doi: 10.1097/01.naj.0000428736.48428.10

Fischer, M., Lackner, P., Beer, R., Helbok, R., Pfausler, B., Schneider, D., . . . Broessner, G. (2015). Cooling activity is associated with neurological outcome in patients with severe cerebrovascular disease undergoing endovascular temperature control. *Neurocrit Care, 23*(2), 205-209. doi: 10.1007/s12028-015-0122-0

Francoeur, C. L., & Mayer, S. A. (2016). Management of delayed cerebral ischemia after subarachnoid hemorrhage. *Crit Care, 20*(1), 277. doi: 10.1186/s13054-016-1447-6

Froehler, M. T., & Geocadin, R. G. (2007). Postprocedure intensive care management of stroke patients. *Endovascular Today, 81-84*.

Frontera, J. A., Curtis, J. R., Nelson, J. E., Campbell, M., Gabriel, M., Mosenthal, A. C., . . . Improving Palliative Care in the ICU Project Advisory Board. (2015). Integrating palliative care into the care of neurocritically ill patients: A report from the improving palliative care in the ICU Project Advisory Board and the Center to Advance Palliative Care. *Crit Care Med, 43*(9), 1964-1977. doi: 10.1097/CCM.0000000000001131

Glisic, E. K., Gardiner, L., Josti, L., Dermanelian, E., Ridel, S., Dziodzio, J., . . . Seder, D. B. (2016). Inadequacy of headache management after subarachnoid hemorrhage. *Am J Crit Care, 25*(2), 136-143. doi: 10.4037/ajcc2016486

Gould, M. K., Garcia, D. A., Wren, S. M., Karanicolas, P. J., Arcelus, J. I., Heit, J. A., & Samama, C. M. (2012). Prevention of VTE in nonorthopedic surgical patients: Antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. *Chest, 141*(2 Suppl), e227S-e277S. doi: 10.1378/chest.11-2297

Hacker, R. I., Ritter, G., Nelson, C., Knobel, D., Gupta, R., Hopkins, K., . . . Barrera, R. (2012). Subcutaneous heparin does not increase postoperative complications in neurosurgical patients: An institutional experience. *J Crit Care, 27*(3), 250-254. doi: 10.1016/j.jcrc.2011.09.005

Hindman, B. J., Bayman, E. O., Pfisterer, W. K., Torner, J. C., & Todd, M. M. (2010). No association between intraoperative hypothermia or supplemental protective drug and neurologic outcomes in patients undergoing temporary clipping during cerebral aneurysm surgery: Findings from the Intraoperative Hypothermia for Aneurysm Surgery Trial. *Anesthesiology, 112*(1), 86-101. doi: 10.1097/ALN.0b013e3181c5e28f

Hoefnagel, D., Dammers, R., Ter Laak-Poort, M. P., & Avezaat, C. J. (2008). Risk factors for infections related to external ventricular drainage. *Acta Neurochir (Wien), 150*(3), 209-214; discussion 214. doi: 10.1007/s00701-007-1458-9

Hoekema, D., Schmidt, R. H., & Ross, I. (2007). Lumbar drainage for subarachnoid hemorrhage: technical considerations and safety analysis. *Neurocrit Care, 7*(1), 3-9. doi: 10.1007/s12028-007-0047-3

Hoff, R. G., van Dijk, G. W., Algra, A., Kalkman, C. J., & Rinkel, G. J. (2008). Fluid balance and blood volume measurement after aneurysmal subarachnoid hemorrhage. *Neurocrit Care, 8*(3), 391-397. doi: 10.1007/s12028-007-9043-x

Holloway, R. G., Ladwig, S., Robb, J., Kelly, A., Nielsen, E., & Quill, T. E. (2010). Palliative care consultations in hospitalized stroke patients. *J Palliat Med, 13*(4), 407-412. doi: 10.1089/jpm.2009.0278

Human, T., Diringer, M. N., Allen, M., Zipfel, G. J., Chicoine, M., Dacey, R., & Dhar, R. (2017). A randomized trial of brief versus extended seizure prophylaxis after aneurysmal subarachnoid hemorrhage. *Neurocrit Care*. doi: 10.1007/s12028-017-0440-5

Iosif, C., Di Maria, F., Sourour, N., Degos, V., Bonneville, F., Biondi, A., . . . Clarencon, F. (2014). Is a high initial World Federation of Neurosurgery (WFNS) grade really associated with a poor clinical outcome in elderly patients with ruptured intracranial aneurysms treated with coiling? *J Neurointerv Surg, 6*(4), 286-290. doi: 10.1136/neurintsurg-2013-010711

Jaja, B. N., Cusimano, M. D., Etminan, N., Hanggi, D., Hasan, D., Ilodigwe, D., . . . Macdonald, R. L. (2013). Clinical prediction models for aneurysmal subarachnoid hemorrhage: a systematic review. *Neurocrit Care, 18*(1), 143-153. doi: 10.1007/s12028-012-9792-z

Jiang, Y., Ye, Z., You, C., Hu, X., Liu, Y., Li, H., . . . Li, J. P. (2015). Systematic review of decreased intracranial pressure with optimal head elevation in postcraniotomy patients: A meta-analysis. *J Adv Nurs, 71*(10), 2237-2246. doi: 10.1111/jan.12679

Joffe, A. M., Khandelwal, N., Hallman, M. R., & Treggiari, M. M. (2015). Assessment of circulating blood volume with fluid administration targeting euvolemia or hypervolemia. *Neurocrit Care, 22*(1), 82-88. doi: 10.1007/s12028-014-9993-8

Karabinis, A., Mandragos, K., Stergiopoulos, S., Komnos, A., Soukup, J., Speelberg, B., & Kirkham, A. J. (2004). Safety and efficacy of analgesia-based sedation with remifentanil versus standard hypnotic-based regimens in intensive care unit patients with brain injuries: a randomised, controlled trial [ISRCTN50308308]. *Crit Care, 8*(4), R268-280. doi: 10.1186/cc2896

Karic, T., Roe, C., Nordenmark, T. H., Becker, F., & Sorteberg, A. (2016). Impact of early mobilization and rehabilitation on global functional outcome one year after aneurysmal subarachnoid hemorrhage. *J Rehabil Med, 48*(8), 676-682. doi: 10.2340/16501977-2121

Karic, T., Sorteberg, A., Haug Nordenmark, T., Becker, F., & Roe, C. (2015). Early rehabilitation in patients with acute aneurysmal subarachnoid hemorrhage. *Disabil Rehabil, 37*(16), 1446-1454. doi: 10.3109/09638288.2014.966162

Karnatovskaia, L. V., Lee, A. S., Festic, E., Kramer, C. L., & Freeman, W. D. (2014). Effect of prolonged therapeutic hypothermia on intracranial pressure, organ function, and hospital outcomes among patients with aneurysmal subarachnoid hemorrhage. *Neurocrit Care, 21*(3), 451-461. doi: 10.1007/s12028-014-9989-4

Keegan, M. T. (2008). Sedation in the neurologic intensive care unit. *Curr Treat Options Neurol, 10*(2), 111-125.

Kim, G. S., Amato, A., James, M. L., Britz, G. W., Zomorodi, A., Graffagnino, C., . . . Olson, D. M. (2011). Continuous and intermittent CSF diversion after subarachnoid hemorrhage: A pilot study. *Neurocrit Care, 14*(1), 68-72. doi: 10.1007/s12028-010-9401-y

Kim, H., & Lee, E. S. (2015). Major difficulties and information needs recognised by nurses in applying graduated compression stocking and intermittent pneumatic compression for deep vein thrombosis prophylaxis. *J Clin Nurs, 24*(1-2), 308-311. doi: 10.1111/jocn.12610

Kim, M. N., Edlow, B. L., Durduran, T., Frangos, S., Mesquita, R. C., Levine, J. M., . . . Detre, J. A. (2014). Continuous optical monitoring of cerebral hemodynamics during head-of-bed manipulation in brain-injured adults. *Neurocrit Care, 20*(3), 443-453. doi: 10.1007/s12028-013-9849-7

Kinnier, C. V., Ju, M. H., Kmiecik, T., Barnard, C., Halverson, T., Yang, A. D., . . . Bilimoria, K. Y. (2016). Development of a novel composite process measure for venous thromboembolism prophylaxis. *Med Care, 54*(2), 210-217. doi: 10.1097/mlr.0000000000000474

Kissoon, N. R., Mandrekar, J. N., Fugate, J. E., Lanzino, G., Wijdicks, E. F., & Rabinstein, A. A. (2015). Positive fluid balance is associated with poor outcomes in subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis, 24*(10), 2245-2251. doi: 10.1016/j.jstrokecerebrovasdis.2015.05.027

Kramer, A. H., Roberts, D. J., & Zygun, D. A. (2012). Optimal glycemic control in neurocritical care patients: a systematic review and meta-analysis. *Crit Care, 16*(5), R203. doi: 10.1186/cc11812

Kramer, C. L., Pegoli, M., Mandrekar, J., Lanzino, G., & Rabinstein, A. A. (2017). Refining the association of fever with functional outcome in aneurysmal subarachnoid hemorrhage. *Neurocrit Care, 26*(1), 41-47. doi: 10.1007/s12028-016-0281-7

Kshettry, V. R., Rosenbaum, B. P., Seicean, A., Kelly, M. L., Schiltz, N. K., & Weil, R. J. (2014). Incidence and risk factors associated with in-hospital venous thromboembolism after aneurysmal subarachnoid hemorrhage. *J Clin Neurosci, 21*(2), 282-286. doi: 10.1016/j.jocn.2013.07.003

Kung, D. K., Chalouhi, N., Jabbour, P. M., Starke, R. M., Dumont, A. S., Winn, H. R., . . . Hasan, D. M. (2013). Cerebral blood flow dynamics and head-of-bed changes in the setting of subarachnoid hemorrhage. *Biomed Res Int, 2013*, 640638. doi: 10.1155/2013/640638

Kuramatsu, J. B., Kollmar, R., Gerner, S. T., Madzar, D., Pisarcikova, A., Staykov, D., . . . Huttner, H. B. (2015). Is hypothermia helpful in severe subarachnoid hemorrhage? An exploratory study on macro vascular spasm, delayed cerebral infarction and functional outcome after prolonged hypothermia. *Cerebrovasc Dis, 40*(5-6), 228-235. doi: 10.1159/000439178

Kurtz, P., Claassen, J., Helbok, R., Schmidt, J., Fernandez, L., Presciutti, M., . . . Mayer, S. A. (2014). Systemic glucose variability predicts cerebral metabolic distress and mortality after subarachnoid hemorrhage: A retrospective observational study. *Crit Care, 18*(3), R89. doi: 10.1186/cc13857

Kwon, O. Y., Kim, Y. J., Kim, Y. J., Cho, C. S., Lee, S. K., & Cho, M. K. (2008). The utility and benefits of external lumbar CSF drainage after endovascular coiling on aneurysmal subarachnoid hemorrhage. *J Korean Neurosurg Soc, 43*(6), 281-287. doi: 10.3340/jkns.2008.43.6.281

Lantigua, H., Ortega-Gutierrez, S., Schmidt, J. M., Lee, K., Badjatia, N., Agarwal, S., . . . Mayer, S. A. (2015). Subarachnoid hemorrhage: Who dies, and why? *Crit Care, 19*, 309. doi: 10.1186/s13054-015-1036-0

Lanzino, G., D'Urso, P. I., Suarez, J., & Participants in the International Multi-Disciplinary Consensus Conference on the Critical Care Management of Subarachnoid Hemorrhage. (2011). Seizures and anticonvulsants after aneurysmal subarachnoid hemorrhage. *Neurocrit Care, 15*(2), 247-256. doi: 10.1007/s12028-011-9584-x

Latorre, J. G., Chou, S. H., Nogueira, R. G., Singhal, A. B., Carter, B. S., Ogilvy, C. S., & Rordorf, G. A. (2009). Effective glycemic control with aggressive hyperglycemia management is associated with improved outcome in aneurysmal subarachnoid hemorrhage. *Stroke, 40*(5), 1644-1652. doi: 10.1161/STROKEAHA.108.535534

Ledwith, M. B., Bloom, S., Maloney-Wilensky, E., Coyle, B., Polomano, R. C., & Le Roux, P. D. (2010). Effect of body position on cerebral oxygenation and physiologic parameters in patients with acute neurological conditions. *J Neurosci Nurs, 42*(5), 280-287.

Lennihan, L., Mayer, S. A., Fink, M. E., Beckford, A., Paik, M. C., Zhang, H., . . . Solomon, R. A. (2000). Effect of hypervolemic therapy on cerebral blood flow after subarachnoid hemorrhage: A randomized controlled trial. *Stroke, 31*(2), 383-391.

Lin, L. Y., Lin, H. C., Lee, P. C., Ma, W. Y., & Lin, H. D. (2007). Hyperglycemia correlates with outcomes in patients receiving total parenteral nutrition. *Am J Med Sci, 333*(5), 261-265. doi: 10.1097/MAJ.0b013e3180536b26

Lindberg, J. O., & Engstrom, A. (2011). Critical care nurses' experiences: "A good relationship with the patient is a prerequisite for successful pain relief management." *Pain Manag Nurs, 12*(3), 163-172. doi: 10.1016/j.pmn.2010.03.009

Ling, Y., Li, X., & Gao, X. (2012). Intensive versus conventional glucose control in critically ill patients: a meta-analysis of randomized controlled trials. *Eur J Intern Med, 23*(6), 564-574. doi: 10.1016/j.ejim.2012.02.013

Lo, E., Mendelow, A., Sacco, R., & Wong, L. (2015). *Stroke: Pathophysiology, diagnosis, and management* (6th ed.). New York: Elsevier.

Ma, Z., Wang, Q., & Liu, M. (2013). Early versus delayed mobilisation for aneurysmal subarachnoid haemorrhage. *Cochrane Database Syst Rev*(5), CD008346. doi: 10.1002/14651858.CD008346.pub2

Mahaney, K. B., Todd, M. M., Bayman, E. O., Torner, J. C., & IHAST Investigators. (2012). Acute postoperative neurological deterioration associated with surgery for ruptured intracranial aneurysm: Incidence, predictors, and outcomes. *J Neurosurg, 116*(6), 1267-1278. doi: 10.3171/2012.1.JNS111277

Makic, M. B., Rauen, C., Watson, R., & Poteet, A. W. (2014). Examining the evidence to guide practice: Challenging practice habits. *Crit Care Nurse, 34*(2), 28-45; quiz 46. doi: 10.4037/ccn2014262

Manoel, A. L., Turkel-Parrella, D., Germans, M., Kouzmina, E., Almendra Pda, S., Marotta, T., . . . Abrahamson, S. (2014). Safety of early pharmacological thromboprophylaxis after subarachnoid hemorrhage. *Can J Neurol Sci, 41*(5), 554-561. doi: 10.1017/cjn.2014.16

Martini, R. P., Deem, S., Brown, M., Souter, M. J., Yanez, N. D., Daniel, S., & Treggiari, M. M. (2012). The association between fluid balance and outcomes after subarachnoid hemorrhage. *Neurocrit Care, 17*(2), 191-198. doi: 10.1007/s12028-011-9573-0

Mathieu, A., Guillon, A., Leyre, S., Martin, F., Fusciardi, J., & Laffon, M. (2013). Aerosolized lidocaine during invasive mechanical ventilation: In vitro characterization and clinical efficiency to prevent systemic and cerebral hemodynamic changes induced by endotracheal suctioning in head-injured patients. *J Neurosurg Anesthesiol, 25*(1), 8-15. doi: 10.1097/ANA.0b013e31826a75b1

McNett, M. M., & Olson, D. M. (2013). Evidence to guide nursing interventions for critically ill neurologically impaired patients with ICP monitoring. *J Neurosci Nurs, 45*(3), 120-123. doi: 10.1097/JNN.0b013e3182901f0a

Milinis, K., Thapar, A., O'Neill, K., & Davies, A. H. (2017). History of aneurysmal spontaneous subarachnoid hemorrhage. *Stroke, 48*(10), e280-e283. doi: 10.1161/STROKEAHA.117.017282

Mocco, J., Ransom, E. R., Komotar, R. J., Sergot, P. B., Ostapkovich, N., Schmidt, J. M., . . . Connolly, E. S., Jr. (2006). Long-term domain-specific improvement following poor grade aneurysmal subarachnoid hemorrhage. *J Neurol, 253*(10), 1278-1284. doi: 10.1007/s00415-006-0179-y

Morad, A. H., Tamargo, R. J., & Gottschalk, A. (2016). The Longitudinal course of pain and analgesic therapy following aneurysmal subarachnoid hemorrhage: A cohort study. *Headache, 56*(10), 1617-1625. doi: 10.1111/head.12908

Murphy-Human, T., Welch, E., Zipfel, G., Diringer, M. N., & Dhar, R. (2011). Comparison of short-duration levetiracetam with extended-course phenytoin for seizure prophylaxis after subarachnoid hemorrhage. *World Neurosurg, 75*(2), 269-274. doi: 10.1016/j.wneu.2010.09.002

Nagao, S., Irie, K., Kawai, N., Nakamura, T., Kunishio, K., & Matsumoto, Y. (2003). The use of mild hypothermia for patients with severe vasospasm: a preliminary report. *J Clin Neurosci, 10*(2), 208-212.

Naidech, A. M., Kreiter, K. T., Janjua, N., Ostapkovich, N., Parra, A., Commichau, C., . . . Fitzsimmons, B. F. (2005). Phenytoin exposure is associated with functional and cognitive disability after subarachnoid hemorrhage. *Stroke, 36*(3), 583-587. doi: 10.1161/01.STR.0000141936.36596.1e

Naidech, A. M., Levasseur, K., Liebling, S., Garg, R. K., Shapiro, M., Ault, M. L., . . . Batjer, H. H. (2010). Moderate hypoglycemia is associated with vasospasm, cerebral infarction, and 3-month disability after subarachnoid hemorrhage. *Neurocrit Care, 12*(2), 181-187. doi: 10.1007/s12028-009-9311-z

Nichols, D. C., Nadpara, P. A., Taylor, P. D., & Brophy, G. M. (2016). Intravenous versus oral acetaminophen for pain control in neurocritical care patients. *Neurocrit Care, 25*(3), 400-406. doi: 10.1007/s12028-016-0289-z

Nielsen, A. K., Jeppesen, A. N., Kirkegaard, H., & Hvas, A. M. (2016). Changes in coagulation during therapeutic hypothermia in cardiac arrest patients. *Resuscitation, 98*, 85-90. doi: 10.1016/j.resuscitation.2015.11.007

Nwachuku, E. L., Puccio, A. M., Fetzick, A., Scruggs, B., Chang, Y. F., Shutter, L. A., & Okonkwo, D. O. (2014). Intermittent versus continuous cerebrospinal fluid drainage management in adult severe traumatic brain injury: Assessment of intracranial pressure burden. *Neurocrit Care, 20*(1), 49-53. doi: 10.1007/s12028-013-9885-3

Nyholm, L., Howells, T., & Enblad, P. (2017). Predictive factors that may contribute to secondary insults with nursing interventions in adults with traumatic brain injury. *J Neurosci Nurs, 49*(1), 49-55. doi: 10.1097/JNN.0000000000000260

Nyholm, L., Steffansson, E., Frojd, C., & Enblad, P. (2014). Secondary insults related to nursing interventions in neurointensive care: A descriptive pilot study. *J Neurosci Nurs, 46*(5), 285-291. doi: 10.1097/JNN.0000000000000077

Nyquist, P., Bautista, C., Jichici, D., Burns, J., Chhangani, S., DeFilippis, M., . . . Meyer, K. (2016). Prophylaxis of venous thrombosis in neurocritical care patients: An evidence-based guideline: A statement for healthcare professionals from the Neurocritical Care Society. *Neurocrit Care, 24*(1), 47-60. doi: 10.1007/s12028-015-0221-y

Oddo, M., Crippa, I. A., Mehta, S., Menon, D., Payen, J. F., Taccone, F. S., & Citerio, G. (2016). Optimizing sedation in patients with acute brain injury. *Crit Care, 20*(1), 128. doi: 10.1186/s13054-016-1294-5

Oddo, M., Frangos, S., Milby, A., Chen, I., Maloney-Wilensky, E., Murtrie, E. M., . . . Levine, J. M. (2009). Induced normothermia attenuates cerebral metabolic distress in patients with aneurysmal subarachnoid hemorrhage and refractory fever. *Stroke, 40*(5), 1913-1916. doi: 10.1161/STROKEAHA.108.534115

Okazaki, T., Hifumi, T., Kawakita, K., Shishido, H., Ogawa, D., Okauchi, M., . . . Kuroda, Y. (2018). Blood glucose variability: A strong independent predictor of neurological outcomes in aneurysmal subarachnoid hemorrhage. *J Intensive Care Med, 33*(3), 189-195. doi: 10.1177/0885066616669328

Olkowski, B. F., Devine, M. A., Slotnick, L. E., Veznedaroglu, E., Liebman, K. M., Arcaro, M. L., & Binning, M. J. (2013). Safety and feasibility of an early mobilization program for patients with aneurysmal subarachnoid hemorrhage. *Phys Ther, 93*(2), 208-215. doi: 10.2522/ptj.20110334

Olson, D. M., Bader, M. K., Dennis, C., Mahanes, D., & Riemen, K. (2010). Multicenter pilot study: Safety of automated chest percussion in patients at risk for intracranial hypertension. *J Neurosci Nurs, 42*(3), 119-127.

Olson, D. M., McNett, M. M., Lewis, L. S., Riemen, K. E., & Bautista, C. (2013). Effects of nursing interventions on intracranial pressure. *Am J Crit Care, 22*(5), 431-438. doi: 10.4037/ajcc2013751

Olson, D. M., Thoyre, S. M., Bennett, S. N., Stoner, J. B., & Graffagnino, C. (2009). Effect of mechanical chest percussion on intracranial pressure: A pilot study. *Am J Crit Care, 18*(4), 330-335. doi: 10.4037/ajcc2009523

Olson, D. M., Thoyre, S. M., Turner, D. A., Bennett, S., & Graffagnino, C. (2007). Changes in intracranial pressure associated with chest physiotherapy. *Neurocrit Care, 6*(2), 100-103. doi: 10.1007/s12028-007-0015-y

Olson, D. M., Zomorodi, M., Britz, G. W., Zomorodi, A. R., Amato, A., & Graffagnino, C. (2013). Continuous cerebral spinal fluid drainage associated with complications in patients admitted with subarachnoid hemorrhage. *J Neurosurg, 119*(4), 974-980. doi: 10.3171/2013.6.JNS122403

Olson, E. J., Bandle, J., & Calvo, R. Y. (2015). Heparin versus enoxaparin for prevention of venous thromboembolism after trauma: A randomized noninferiority trial. *The Journal of Trauma and Acute Care Surgery, 79*(6), 961-969. doi: 10.1097/TA.0000000000000750.

Ooi, Y. C., Dagi, T. F., Maltenfort, M., Rincon, F., Vibbert, M., Jabbour, P., . . . Jallo, J. (2012). Tight glycemic control reduces infection and improves neurological outcome in critically ill neurosurgical and neurological patients. *Neurosurgery, 71*(3), 692-702; discussion 702. doi: 10.1227/NEU.0b013e3182631eb4

Panczykowski, D., Pease, M., Zhao, Y., Weiner, G., Ares, W., Crago, E., . . . Ducruet, A. F. (2016). Prophylactic antiepileptics and seizure incidence following subarachnoid hemorrhage: A propensity score-matched analysis. *Stroke, 47*(7), 1754-1760. doi: 10.1161/STROKEAHA.116.013766

Park, S., Yang, N., & Seo, E. (2015). The effectiveness of lumbar cerebrospinal fluid drainage to reduce the cerebral vasospasm after surgical clipping for aneurysmal subarachnoid hemorrhage. *J Korean Neurosurg Soc, 57*(3), 167-173. doi: 10.3340/jkns.2015.57.3.167

Pasternak, J. J., McGregor, D. G., Schroeder, D. R., Lanier, W. L., Shi, Q., Hindman, B. J., . . . Todd, M. M. (2008). Hyperglycemia in patients undergoing cerebral aneurysm surgery: Its association with long-term gross neurologic and neuropsychological function. *Mayo Clin Proc, 83*(4), 406-417. doi: 10.4065/83.4.406

Paul, B. S., & Paul, G. (2013). Sedation in neurological intensive care unit. *Ann Indian Acad Neurol, 16*(2), 194-202. doi: 10.4103/0972-2327.112465

Pegoli, M., Mandrekar, J., Rabinstein, A. A., & Lanzino, G. (2015). Predictors of excellent functional outcome in aneurysmal subarachnoid hemorrhage. *J Neurosurg, 122*(2), 414-418. doi: 10.3171/2014.10.JNS14290

Petersen, L. G., Petersen, J. C., Andresen, M., Secher, N. H., & Juhler, M. (2016). Postural influence on intracranial and cerebral perfusion pressure in ambulatory neurosurgical patients. *Am J Physiol Regul Integr Comp Physiol, 310*(1), R100-104. doi: 10.1152/ajpregu.00302.2015

Prakash, A., & Matta, B. F. (2008). Hyperglycaemia and neurological injury. *Curr Opin Anaesthesiol, 21*(5), 565-569. doi: 10.1097/ACO.0b013e32830f44e4

Prendergast, V., Hagell, P., & Hallberg, I. R. (2011). Electric versus manual tooth brushing among neuroscience ICU patients: Is it safe? *Neurocrit Care, 14*(2), 281-286. doi: 10.1007/s12028-011-9502-2

Prendergast, V., Hallberg, I. R., Jahnke, H., Kleiman, C., & Hagell, P. (2009). Oral health, ventilator-associated pneumonia, and intracranial pressure in intubated patients in a neuroscience intensive care unit. *Am J Crit Care, 18*(4), 368-376. doi: 10.4037/ajcc2009621

Preventing Venous Thromboembolism in Adults. (2016). *Crit Care Nurse, 36*(5), e20-e23. doi: 10.4037/ccn2016638

Puntillo, K. A., Morris, A. B., Thompson, C. L., Stanik-Hutt, J., White, C. A., & Wild, L. R. (2004). Pain behaviors observed during six common procedures: results from Thunder Project II. *Crit Care Med, 32*(2), 421-427. doi: 10.1097/01.ccm.0000108875.35298.d2

Qaseem, A., Chou, R., Humphrey, L. L., Starkey, M., & Shekelle, P. (2011). Venous thromboembolism prophylaxis in hospitalized patients: A clinical practice guideline from the American College of Physicians. *Ann Intern Med, 155*(9), 625-632. doi: 10.7326/0003-4819-155-9-201111010-00011

Qian, C., Yu, X., Chen, J., Gu, C., Wang, L., Chen, G., & Dai, Y. (2016). Effect of the drainage of cerebrospinal fluid in patients with aneurismal subarachnoid hemorrhage: A meta-analysis. *Medicine (Baltimore), 95*(41), e5140. doi: 10.1097/MD.0000000000005140

Qureshi, A. I., Adil, M. M., & Suri, M. F. (2014). Rate of use and determinants of withdrawal of care among patients with subarachnoid hemorrhage in the United States. *World Neurosurg, 82*(5), e579-584. doi: 10.1016/j.wneu.2014.07.008

Ramanan, M., Lipman, J., Shorr, A., & Shankar, A. (2015). A meta-analysis of ventriculostomy-associated cerebrospinal fluid infections. *BMC Infect Dis, 15*, 3. doi: 10.1186/s12879-014-0712-z

Rasulo, F. A., Girardini, A., Lavinio, A., De Peri, E., Stefini, R., Cenzato, M., . . . Latronico, N. (2012). Are optimal cerebral perfusion pressure and cerebrovascular autoregulation related to long-term outcome in patients with aneurysmal subarachnoid hemorrhage? *J Neurosurg Anesthesiol, 24*(1), 3-8. doi: 10.1097/ANA.0b013e318224030a

Rhie, S. H., Choi, J. W., Jeon, S. J., Kang, S. D., Joo, M. C., & Kim, M. S. (2016). Characteristics of patients with aneurysmal subarachnoid hemorrhage and risk factors related to dysphagia. *Ann Rehabil Med, 40*(6), 1024-1032. doi: 10.5535/arm.2016.40.6.1024

Rhoney, D. H., & Parker, D., Jr. (2001). Use of sedative and analgesic agents in neurotrauma patients: Effects on cerebral physiology. *Neurol Res, 23*(2-3), 237-259. doi: 10.1179/016164101101198398

Riordan, M. A., Kyle, M., Dedeo, C., Villwock, M. R., Bauer, M., Vallano, M. L., & Deshaies, E. M. (2015). Mild exercise reduces cerebral vasospasm after aneurysm subarachnoid hemorrhage: A retrospective clinical study and correlation with laboratory investigation. *Acta Neurochir Suppl, 120*, 55-61. doi: 10.1007/978-3-319-04981-6_10

Ritsema, D. F., Watson, J. M., Stiteler, A. P., & Nguyen, M. M. (2013). Sequential compression devices in postoperative urologic patients: An observational trial and survey study on the influence of patient and hospital factors on compliance. *BMC Urol, 13*, 20. doi: 10.1186/1471-2490-13-20

Rosengart, A. J., Schultheiss, K. E., Tolentino, J., & Macdonald, R. L. (2007). Prognostic factors for outcome in patients with aneurysmal subarachnoid hemorrhage. *Stroke, 38*(8), 2315-2321. doi: 10.1161/strokeaha.107.484360

Roth, C., Stitz, H., Kalhout, A., Kleffmann, J., Deinsberger, W., & Ferbert, A. (2013). Effect of early physiotherapy on intracranial pressure and cerebral perfusion pressure. *Neurocrit Care, 18*(1), 33-38. doi: 10.1007/s12028-012-9799-5

Roth, C., Stitz, H., Kleffmann, J., Kaestner, S., Deinsberger, W., Ferbert, A., & Gehling, M. (2017). Early physiotherapy by passive range of motion does not affect partial brain tissue oxygenation in neurocritical care patients. *J Neurol Surg A Cent Eur Neurosurg, 78*(1), 42-45. doi: 10.1055/s-0036-1592160

Salary, M., Quigley, M. R., & Wilberger, J. E., Jr. (2007). Relation among aneurysm size, amount of subarachnoid blood, and clinical outcome. *J Neurosurg, 107*(1), 13-17. doi: 10.3171/JNS-07/07/0013

Scheithauer, S., Burgel, U., Ryang, Y. M., Haase, G., Schiefer, J., Koch, S., . . . Lemmen, S. (2009). Prospective surveillance of drain associated meningitis/ventriculitis in a neurosurgery and neurological intensive care unit. *J Neurol Neurosurg Psychiatry, 80*(12), 1381-1385. doi: 10.1136/jnnp.2008.165357

Schlenk, F., Vajkoczy, P., & Sarrafzadeh, A. (2009). Inpatient hyperglycemia following aneurysmal subarachnoid hemorrhage: relation to cerebral metabolism and outcome. *Neurocrit Care, 11*(1), 56-63. doi: 10.1007/s12028-009-9222-z

Schmutzhard, E., & Rabinstein, A. A. (2011). Spontaneous subarachnoid hemorrhage and glucose management. *Neurocrit Care, 15*(2), 281-286. doi: 10.1007/s12028-011-9601-0

Schulz-Stubner, S., & Thiex, R. (2006). Raising the head-of-bed by 30 degrees reduces ICP and improves CPP without compromising cardiac output in euvolemic patients with traumatic brain injury and subarachnoid haemorrhage: A practice audit. *Eur J Anaesthesiol, 23*(2), 177-180. doi: 10.1017/S0265021505232118

Sellars, C., Bowie, L., Bagg, J., Sweeney, M. P., Miller, H., Tilston, J., . . . Stott, D. J. (2007). Risk factors for chest infection in acute stroke: A prospective cohort study. *Stroke, 38*(8), 2284-2291. doi: 10.1161/strokeaha.106.478156

Sessler, C. N., Grap, M. J., & Brophy, G. M. (2001). Multidisciplinary management of sedation and analgesia in critical care. *Semin Respir Crit Care Med, 22*(2), 211-226. doi: 10.1055/s-2001-13834

Seule, M., Muroi, C., Mink, S., Yonekawa, Y., & Keller, E. (2009). Therapeutic hypothermia in patients with aneurysmal subarachnoid hemorrhage, refractory intracranial hypertension, or cerebral vasospasm. *Neurosurgery, 64*(1), 86-92. doi: 10.1227/01.NEU.0000336312.32773.A0

Seule, M., Muroi, C., Sikorski, C., Hugelshofer, M., Winkler, K., & Keller, E. (2014). Therapeutic hypothermia reduces middle cerebral artery flow velocity in patients with severe aneurysmal subarachnoid hemorrhage. *Neurocrit Care, 20*(2), 255-262. doi: 10.1007/s12028-013-9927-x

Shimamura, N., Matsuda, N., Satou, J., Nakano, T., & Ohkuma, H. (2014). Early ambulation produces favorable outcome and nondemential state in aneurysmal subarachnoid hemorrhage patients older than 70 years of age. *World Neurosurg, 81*(2), 330-334. doi: 10.1016/j.wneu.2012.12.007

Siironen, J., Juvela, S., Varis, J., Porras, M., Poussa, K., Ilveskero, S., . . . Lassila, R. (2003). No effect of enoxaparin on outcome of aneurysmal subarachnoid hemorrhage: A randomized, double-blind, placebo-controlled clinical trial. *J Neurosurg, 99*(6), 953-959. doi: 10.3171/jns.2003.99.6.0953

Singh, T., Peters, S. R., Tirschwell, D. L., & Creutzfeldt, C. J. (2017). Palliative care for hospitalized patients with stroke: Results from the 2010 to 2012 national inpatient sample. *Stroke, 48*(9), 2534-2540. doi: 10.1161/STROKEAHA.117.016893

Solanki, C., Pandey, P., & Rao, K. V. (2016). Predictors of aneurysmal rebleed before definitive surgical or endovascular management. *Acta Neurochir (Wien), 158*(6), 1037-1044. doi: 10.1007/s00701-016-2784-6

Springer, M. V., Schmidt, J. M., Wartenberg, K. E., Frontera, J. A., Badjatia, N., & Mayer, S. A. (2009). Predictors of global cognitive impairment 1 year after subarachnoid hemorrhage. *Neurosurgery, 65*(6), 1043-1050; discussion 1050-1041. doi: 10.1227/01.NEU.0000359317.15269.20

Steiner, T., Juvela, S., Unterberg, A., Jung, C., Forsting, M., Rinkel, G., & European Stroke Organization. (2013). European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. *Cerebrovasc Dis, 35*(2), 93-112. doi: 10.1159/000346087

Suehiro, E., Sadahiro, H., Goto, H., Oku, T., Oka, F., Fujiyama, Y., . . . Suzuki, M. (2016). Importance of early postoperative body temperature management for treatment of subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis, 25*(6), 1482-1488. doi: 10.1016/j.jstrokecerebrovasdis.2016.01.053

Sun, C., Du, H., Yin, L., He, M., Tian, Y., & Li, H. (2014). Choice for the removal of bloody cerebrospinal fluid in postcoiling aneurysmal subarachnoid hemorrhage: External ventricular drainage or lumbar drainage? *Turk Neurosurg, 24*(5), 737-744. doi: 10.5137/1019-5149.JTN.9837-13.2

Szabo, C. M., Grap, M. J., Munro, C. L., Starkweather, A., & Merchant, R. E. (2014). The effect of oral care on intracranial pressure in critically ill adults. *J Neurosci Nurs, 46*(6), 321-329. doi: 10.1097/JNN.0000000000000092

Thiele, R. H., Pouratian, N., Zuo, Z., Scalzo, D. C., Dobbs, H. A., Dumont, A. S., . . . Nemergut, E. C. (2009). Strict glucose control does not affect mortality after aneurysmal subarachnoid hemorrhage. *Anesthesiology, 110*(3), 603-610. doi: 10.1097/ALN.0b013e318198006a

Titsworth, W. L., Abram, J., Fullerton, A., Hester, J., Guin, P., Waters, M. F., & Mocco, J. (2013). Prospective quality initiative to maximize dysphagia screening reduces hospital-acquired pneumonia prevalence in patients with stroke. *Stroke, 44*(11), 3154-3160. doi: 10.1161/strokeaha.111.000204

Todd, M. M., Hindman, B. J., Clarke, W. R., & Torner, J. C. (2005). Mild intraoperative hypothermia during surgery for intracranial aneurysm. *N Engl J Med, 352*(2), 135-145. doi: 10.1056/NEJMoa040975

Todd, M. M., Hindman, B. J., Clarke, W. R., Torner, J. C., Weeks, J. B., Bayman, E. O., . . . IHAST Investigators. (2009). Perioperative fever and outcome in surgical patients with aneurysmal subarachnoid hemorrhage. *Neurosurgery, 64*(5), 897-908; discussion 908. doi: 10.1227/01.NEU.0000341903.11527.2F

Togashi, K., Joffe, A. M., Sekhar, L., Kim, L., Lam, A., Yanez, D., . . . Treggiari, M. M. (2015). Randomized pilot trial of intensive management of blood pressure or volume expansion in subarachnoid hemorrhage (IMPROVES). *Neurosurgery, 76*(2), 125-134; discussion 134-125; quiz 135. doi: 10.1227/neu.0000000000000592

Tracy, M. F., & Chlan, L. (2011). Nonpharmacological interventions to manage common symptoms in patients receiving mechanical ventilation. *Crit Care Nurse, 31*(3), 19-28. doi: 10.4037/ccn2011653

Tume, L. N., Baines, P. B., & Lisboa, P. J. (2011). The effect of nursing interventions on the intracranial pressure in paediatric traumatic brain injury. *Nurs Crit Care, 16*(2), 77-84. doi: 10.1111/j.1478-5153.2010.00412.x

van Donkelaar, C. E., Dijkland, S. A., van den Bergh, W. M., Bakker, J., Dippel, D. W., Nijsten, M. W., & van der Jagt, M. (2016). Early circulating lactate and glucose levels after aneurysmal subarachnoid hemorrhage correlate with poor outcome and delayed cerebral ischemia: A two-center cohort study. *Crit Care Med, 44*(5), 966-972. doi: 10.1097/ccm.0000000000001569

Vivancos, J., Gilo, F., Frutos, R., Maestre, J., Garcia-Pastor, A., Quintana, F., . . . Tejada, J. (2014). Clinical management guidelines for subarachnoid haemorrhage. Diagnosis and treatment. *Neurologia, 29*(6), 353-370. doi: 10.1016/j.nrl.2012.07.009

Williams, T. A., Leslie, G. D., Dobb, G. J., Roberts, B., & van Heerden, P. V. (2011). Decrease in proven ventriculitis by reducing the frequency of cerebrospinal fluid sampling from extraventricular drains. *J Neurosurg, 115*(5), 1040-1046. doi: 10.3171/2011.6.JNS11167

Williamson, R. A., Phillips-Bute, B. G., McDonagh, D. L., Gray, M. C., Zomorodi, A. R., Olson, D. M., . . . James, M. L. (2014). Predictors of extraventricular drain-associated bacterial ventriculitis. *J Crit Care, 29*(1), 77-82. doi: 10.1016/j.jcrc.2013.08.012

Young, G. B., Jordan, K. G., & Doig, G. S. (1996). An assessment of nonconvulsive seizures in the intensive care unit using continuous EEG monitoring: An investigation of variables associated with mortality. *Neurology, 47*(1), 83-89.

Yu, H., Zhan, R., Wen, L., Shen, J., & Fan, Z. (2014). The relationship between risk factors and prognostic factors in patients with shunt-dependent hydrocephalus after aneurysmal subarachnoid hemorrhage. *J Craniofac Surg, 25*(3), 902-906. doi: 10.1097/scs.0000000000000561

Yuan, M. Z., Li, F., Tian, X., Wang, W., Jia, M., Wang, X. F., & Liu, G. W. (2015). Risk factors for lung infection in stroke patients: a meta-analysis of observational studies. *Expert Rev Anti Infect Ther, 13*(10), 1289-1298. doi: 10.1586/14787210.2015.1085302

Zachariah, J., Snyder, K. A., Graffeo, C. S., Khanal, D. R., Lanzino, G., Wijdicks, E. F., & Rabinstein, A. A. (2016). Risk of ventriculostomy-associated hemorrhage in patients with aneurysmal subarachnoid hemorrhage treated with anticoagulant thromboprophylaxis. *Neurocrit Care, 25*(2), 224-229. doi: 10.1007/s12028-016-0262-x

Zeiler, F. A., AlSubaie, F., Zeiler, K., Bernard, F., & Skrobik, Y. (2016). Analgesia in neurocritical care: An international survey and practice audit. *Crit Care Med, 44*(5), 973-980. doi: 10.1097/CCM.0000000000001602

Zhang, G., Zhang, J. H., & Qin, X. (2011). Fever increased in-hospital mortality after subarachnoid hemorrhage. *Acta Neurochir Suppl, 110*(Pt 1), 239-243. doi: 10.1007/978-3-7091-0353-1_42

Zhang, Y., & Rabinstein, A. A. (2011). Lower head of the bed position does not change blood flow velocity in subarachnoid hemorrhage. *Neurocrit Care, 14*(1), 73-76. doi: 10.1007/s12028-010-9444-0

Morbidity and Mortality						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Bian et al., 2012)	651	Prospective cohort	Patients with SAH within 14 days of onset	Examined whether securing aneurysm reduces long-term mortality and factors affecting prognosis after aSAH	H & H, Fisher, and GCS scores at admission are predictive of 1-year mortality.	Moderate
(Chalouhi et al., 2014)	116	Retrospective	Patients with aSAH with varied endovascular RXs for vasospasm	Retreatment and poor outcome (neurological recovery, pre and post procedure hypodensity, GOS)	H & H grade was predictive of need for retreatment and poor outcome (neurological recovery, pre and post procedure hypodensity, GOS).	Low
(Dengler, Sommerfeld, Diesing, Vajkoczy, & Wolf, 2018)	423	Retrospective	aSAH	New infarct, functional outcome by mRS	Modified Fisher and Barrows Neurological Institute grade predicted cerebral infarct ($p \leq 0.001$, $p \leq 0.001$). Modified Fisher and Barrows Neurological Institute grade predicted poor outcome ($p \leq 0.001$, $p \leq 0.001$). H & H and WFNS predicted cerebral infarct ($p \leq 0.001$, $p \leq 0.001$). H & H and WFNS predicted poor outcome ($p \leq 0.001$, $p \leq 0.001$). Combined VASOGRADE and HAIR scores predicted cerebral infarct ($p \leq 0.001$, $p \leq 0.001$). Combined VASOGRADE and HAIR scores predicted poor outcome ($p < 0.001$, $p \leq 0.001$).	Moderate
(Duan et al., 2016)	520	Prospective observational longitudinal	Patients older than age 60 with aSAH who underwent endovascular RX	Functional status post intervention and death	H & H and Fisher scales were predictive of poor outcome (mRS $\geq$ 3)	Moderate
(Mocco et al., 2006)	98	Prospective	Those with poor H & H grade and aSAH	12-month outcomes	Age younger than 65 years ($p < 0.001$), hyperglycemia ($p < 0.001$), H & H grade 5 ($p < 0.001$), and aneurysm 13 mm < ($p < 0.001$) predicted poor outcome. Computed Prognosis Score combining age, blood glucose, H & H grade, and aneurysm size predicted outcomes for poor-grade aSAH	Moderate
(Salary, Quigley, & Wilberger, 2007)	133	Retrospective	Consecutive aSAH patients	GOS at 6 months	Age, admission H & H grade, and clinical vasospasm were related to outcomes ($p < 0.0001$)	Moderate

Morbidity and Mortality *continued*

Reference	Sample Size	Study Design	Population	Endpoint	Findings	Quality
(Yu, Zhan, Wen, Shen, & Fan, 2014)	202	Retrospective	Patients with ruptured aSAH admitted to the neurosurgery department at Zhejiang University. Excluding trauma, preexisting neurological deficits, age-related brain atrophy, death fewer than 7 days postictal	Shunt insertion	Independent shunt predictors Age H & H grade GCS score Nosocomial meningitis EVD duration The mFisher score did not independently predict outcomes (GOS or mRS) or shunt dependence.	Low
(Lantigua et al., 2015)	1,200	Prospective	Consecutive patients with SAH at CUSAHOP between July 1996 and January 2009	Mortality	Admission mortality predictors Age Loss of consciousness at ictus GCS Large aneurysm size APACHE II physiologic subscore Modified Fisher Scale score Hospital complication predictors Rebleeding Global cerebral edema Hypernatremia Clinical signs of brainstem herniation SBP < 90 mmHg treated with pressors Pulmonary edema Myocardial ischemia Hepatic failure DCI did not predict mortality.	High
(Solanki, Pandey, & Rao, 2016)	889 (99 rebleeds)	Prospective and retrospective	Retrospective and consecutive patients with aSAH who rebled, excluding those taking antiplatelets or anticoagulants or with untreated aneurysms	Rebleed	Predictors of rebleed Known hypertension (p=0.023), Admit DBP > 90 mmHg (p=0.008) Loss of consciousness (p=0.013) Seizures at first ictus (p=0.002) History of warning HA (p=0.005) Higher 3/4 Fisher grade (p<0.001) Multiple aneurysms (p=0.021) Irregular aneurysm surface (p = 0.002)	Moderate

Morbidity and Mortality *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Pegoli, Mandrekar, Rabinstein, & Lanzino, 2015)	373	Retrospective	Patients with aSAH admitted between 2001 and 2013	Excellent functional outcome (mRS ≤ 1)	Univariate excellent outcome predictors: better WFNS; better modified Fisher grade; and absence of ICH, IVH, and symptomatic hydrocephalus; aneurysm treatment with coil embolization; absence of symptomatic vasospasm, DCI, and radiological infarction; absence of in-hospital seizures; lack of need for CSF diversion; fewer hours with fever; less severe anemia; and absence of transfusion	Moderate
(Mahaney, Todd, Bayman, Torner, & IHAST Investigators, 2012)	1,001	Secondary analysis of clinical trial data from SAH study	SAH	Neurological deterioration	Admit Fisher scale score was significantly associated with occurrence of postoperative neurological deterioration at 24 hours.	Moderate
(Crobeddu et al., 2012)	307	Retrospective	Patients older than 16 years with acute aSAH	Determine which clinical and radiographic variables are associated with DCI	Older age (particularly 68 years or older) and good clinical (WFNS I-III) and radiological (modified Fisher 1-2) grades at presentation can reliably identify patients who will not develop DCI. These patients require less monitoring because they are at low risk for DCI and can be moved out of the ICU after obliteration of the aneurysm.	Low
(Iosif et al., 2014)	59	Observational cohort	Patients age 70 or older with aSAH who underwent endovascular treatment	Technical feasibility, complication profile, and clinical outcomes (Cohen & Hilligoss, 2010) of elderly patients with SAH treated with endovascular therapy	WFNS score was not predictive of GOS. Low WFNS scores between 1-3 were associated with decreased incidence of DCI.	Low

Palliative Care						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Burton & Payne, 2012)	191	Survey and interview	Patients who sustained stroke	Family engagement regarding prognoses and treatment plan, palliative care, and intervention timing	Palliative care needs range from clinical (whether patients are being actively treated and prognostic uncertainty) to service (leadership, specialty status, and neurological focus).	Moderate
(Qureshi, Adil, & Suri, 2014)	266,067	Retrospective	All patients with SAH entered into the NIS database between 2002 and 2010	Withdrawal of care vs. nonwithdrawal of care	Withdrawal of care was instituted for 8,912 (3.4%) patients. Rates of withdrawal increased over time: 1% in 2002 to 6.4% in 2010. Withdrawal was more likely for patients with severe functional loss who were older (70 ± 16 years vs. 57 ± 17 years), white, and female. Medicare/Medicare payor sources were associated with higher rates of care withdrawal (63.3% vs 43.8%, p <0.0001).	High
(Singh, Peters, Tirschwell, & Creutzfeldt, 2017)	395,411	Retrospective database review	All stroke patients entered into the NIS database between 2010 and 2012	PCE	PCEs increased over time ($p <$ 0.05). Age ($p < 0.001$), gender ($p <$ 0.001), ethnicity ($p < 0.001$), and stroke type ($p < 0.001$) were associated with PCE use. Smaller and for-profit hospitals had lower use ($p < 0.001$). PCE was associated with shorter LOS ($p < 0.001$).	High
(Holloway et al., 2010)	101	Retrospective case review	All stroke palliative care consults over a 3-year period (2005-2007) N = 1,085 Stroke N = 101	Compare stroke demographics with other consult demographic types	Patients with stroke who had a PC consult were more functionally impaired, less likely to have capacity, and more likely to die in the hospital and have fewer traditional symptom burdens commonly seen in the PC consultation service. The most common trajectory to death was withdrawal of mechanical ventilation, but this varied by type of stroke. Common treatments discussed during these consultations included mechanical ventilation, artificial nutrition, and tracheostomy; antibiotics, intravenous fluids, and various neurosurgical procedures were less frequently discussed.	Moderate
(Blacquiere, Bhimji, Meggison, Sinclair, & Sharma, 2013)	15	Prospective nonintervention study	All families of patients who died from a stroke over a 1-year period	Evaluate family satisfaction quantitatively after palliative care in acute stroke	Families who sought palliative care had higher rates of satisfaction.	Low

CSF Drainage and Hydrocephalus

Reference	Sample size	Study Design	Population	Endpoint	Findings	Quality
(Qian et al., 2016)	Outcomes N = 808 Vasospasms N = 519	Meta-analysis	aSAH	GOS mRS	Patients with CSF drainage had better outcomes ($p < 0.01$). CSF drainage was associated with fewer angiographic and symptomatic vasospasms ($p < 0.01$). CSF drainage was associated with fewer vasospasm-related cerebral infarcts ($p = 0.03$).	Moderate
(Olson, Zomorodi, et al., 2013)	60	RCT	aSAH with EVD	Vasospasm complications (CSF leak, hemorrhage, self-device removal, ventriculitis, nonpatent EVD, loss of consciousness, mortality, mRS score	An open EVD was associated with more complications ($p < 0.02$). There was no difference in vasospasm or mortality between open EVD and intermittent-open populations with EVDs.	Moderate
(Kim et al., 2011)	37	Prospective observational	aSAH with EVD	Primary: Vasospasm Secondary: Highest ICP, daily CSF output, ICU LOS, mRS	There was no significance regarding vasospasms between continuous and intermittent drain groups. There was no significance in highest ICP, daily CSF output, ICU LOS, and mRS between continuous and intermittent drain groups.	Low
(Nwachuku et al., 2014)	62	Retrospectively matched prospective cohort	TBI	ICU LOS CSF infection Hypothermia RX Neurosurgical intervention Survival status ICP burden	There was no significant ISS, ICU LOS, CSF infection, hypothermia RX, neurosurgical intervention, or survival status between continuous and intermittent drain groups. Patients with closed-intermittent drainage had significantly higher ICP burden ($p = 0.0002$).	Low
(Amato et al., 2011)	37	Prospective cohort	SAH	Vasospasm, LOS, highest average ICP, total CSF drained, and mRS upon discharge in continuous monitoring vs. CSF drainage groups	No significant difference in both groups regarding vasospasm, LOS, highest average ICP, total CSF drained, and mRS discharge between groups	Low
(Hoekema, Schmidt, & Ross, 2007)	25	Retrospective	SAH	Vasospasm, VPS insertion, and evidence of neurologic dysfunction (motor power weakness and changes in reflexes)	For every 25 subjects, one developed neurologic damage. Lumbar drainage is safe for patients with SAH.	Low

CSF Drainage and Hydrocephalus *continued*						
Reference	**Sample size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Sun et al., 2014)	148	Prospective controlled	SAH	Ratios of ICH, vasospasm, infection, duration of catheter placement, hydrocephalus, and GOS after 2 months	Hemorrhage LD: 0 EVD: 7 ($p = 0.014$) Ischemia due to vasospasm LD: 7 EVD: 6 ($p = 0.954$, not significant) Infection Positive cultures in LD: 13 EVD: 11 ($p = 0.437$, not significant) Duration LD: 11.3 ± 2.11 days EVD: 10.7 ± 1.88 ($p = 0.086$, not significant) Chronic hydrocephalus LD: 10 EVD: 9 ($p = 0.831$, not significant) GOS LD: 4.0 ± 0.79 ($p = 0.291$, not significant) EVD: 4.2 ± 0.83	Moderate
(Al-Tamimi et al., 2012)	210	RCT	SAH	Primary: DIND changes in GCS: 1 motor score or 2 eye/verbal scores or new neurologic deterioration 96 hours post initial bleed Secondary: mRS 4, 5, or 6 at day 10 and 6 months Infarction confirmed by MRI or CT VPS prevalence	LD of CSF after aneurysmal SAH reduced the prevalence of DIND and improved early clinical outcome. DIND Control: 35.2% Study: 21.0% ($p = 0.021$) mRS 4, 5, or 6 at Day 10 Control: 62.5% Study: 44.8% ($p = 0.009$) mRS 4, 5, or 6 at 6 months Control: 18.6% Study: 19.8% ($p = 0.83$) Infarction confirmed by MRI or CT Control: 70% Study: 64% ($p = 0.816$) VPS Control: 7.6% Study: 5.7% ($p = 0.58$)	High

CSF Drainage and Hydrocephalus *continued*						
Reference	**Sample size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Kwon et al., 2008)	107	Retrospective	SAH	Vasospasm GOS score at 1 month to 6 months VPS ICU LOS, total LOS Mortality rates	Vasospasm (%) $p < 0.001$ LD: 23.4 Non-LD: 63.3 GOS score at 1 month to 6 months (%) $p < 0.001$ I LD: 2.1 Non-LD: 15 II LD: 2.1 Non-LD: 20 III LD: 0 Non-LD: 5 IV LD: 23.4 Non-LD: 23.3 V LD: 72.3 Non-LD: 36.7 VPS (%) $p = 0.172$ LD: 17 Non-LD: 8.3 ICU LOS (days): $p = 0.955$ LD: 18.8 Non-LD: 18.6 Total hospital stay (days): $p = 0.105$ LD: 33.6 Non-LD: 24.3 Mortality (%): $p = 0.04$ LD: 2.1 Non-LD: 15 Post coiling, LD seems to reduce vasospasm	Low
(Park, Yang, & Seo, 2015)	234	Prospective, nonrandomized	SAH	Vasospasm Angioplasty Cerebral infarction GOS at discharge GOS score at 6 months Mortality	Vasospasm: LD: 19% Non-LD: 42% Angioplasty LD: 17% Non-LD: 38% Cerebral infarctions 54% of each group, respectively GOS score 5- at 6-month LD: 69% Non-LD group: 58% Mortality rate LD: 5% Non-LD: 10%	Moderate

EVD Management						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Camacho et al., 2011)	119	Prospective	ICH SAH TBI Brain tumor	HAI	Length of the EVD left in place was related to an increase in infection rate.	Low
(Scheithauer et al., 2009)	1,333	Prospective	Patients admitted to the neurosurgical and neurological ICU between January 5, 2005, and December 31, 2006 (inclusive of SAH: 55%)	HAI	No significant difference in infection among patients with EVDs for more or less than 9 days	Moderate
(Arif, 2012)	104	Retrospective	SAH IVH	HAI	EVD use longer than 8 days: 16/19 infection EVD use less than 4 days: 3/19 infection	Low
(Williams, Leslie, Dobb, Roberts, & van Heerden, 2011)	382	Prospective	Patients with EVDs (SAH: 37.8%, TBI: 32.2%)	HAI: Daily CSF sampling vs. every-third-day CSF sampling	Preintervention daily sampling ventriculitis: 9.7% Postintervention q3days sampling ventriculitis: 3.4% ($p = 0.02$)	Moderate
(Williamson et al., 2014)	420	Retrospective	Patients with EVDs	HAI: Monitored frequency of CSF sampling	Much higher HAI risk was associated with frequent CSF sampling	Moderate
(Hoefnagel, Dammers, Ter Laak-Poort, & Avezaat, 2008)	228	Retrospective	Patients who underwent external CSF diversion procedures between January 1993 and April 2005 at a single center	CSF infection (+ culture)	Frequency of CSF sampling ($p < 0.001$) and number of catheter days ($p < 0.001$) were associated with infection.	Moderate

Nursing Intervention Effects on Cerebral Hemodynamics						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Olson, McNett, Lewis, Riemen, & Bautista, 2013)	1,593	Secondary analysis of SIM City (Study of ICP Monitoring in Critically Ill; prospective multicenter observational data)	Nurse-patient dyads at 16 hospitals across the United States	ICP 1 min and 5 min after nursing interventions	Within 5 min, limiting stimulation did not result in ICP changes in 84% of patients. ICP levels were lower in 5.8% of observations. ICP levels were higher in 5.8% of observations. Odds of observing lower ICP after 5 min is 1.03 (95% CI = 0.87-1.12; $p = .76$). Odds of observing lower ICP after 5 min is 1.47 (95% CI = 1.24-1.75; $p < 0.001$).	High
(Nyholm, Steffansson, Frojd, & Enblad, 2014)	18	Prospective cohort	Acute SDH, epidural hematoma, ICH, SAH	ICP, CPP, and SBP after nursing interventions	Oral care resulted in ICP > 20 in 4% of encounters. Authors concluded that oral care did not significantly correlate with a secondary insult.	Very low
(Prendergast, Hallberg, Jahnke, Kleiman, & Hagell, 2009)	45 patients and 879 incidents of oral care	Prospective	Intubated patients in the neuro-ICU	VAP ICP	Oral health (assessed by the OAG) declined with increased ventilator days. There was no statistical difference in ICP measures before, during, and after oral care ($p = 0.24$).	Moderate
(Tume, Baines, & Lisboa, 2011)	25	Prospective observational cohort	Children with TBI	ICP (post nursing intervention)	ICP rise associated with oral care Averages 1-5 mmHg Standard deviation 2-5 These differences were not statistically or clinically significant.	Very low
(Prendergast, Hagell, & Hallberg, 2011)	47	RCT	Hemorrhagic stroke	ICP and CPP measurement before, during, and after oral care Manual oral care vs. electric oral care	There is no significant difference in ICP and CPP readings of the two groups. ICP levels initially increased an average of 1.7 mmHg during oral care but decreased by an average of 2.1 mmHg after oral care.	Moderate
(Szabo, Grap, Munro, Starkweather, & Merchant, 2014)	23	Prospective	TBI SAH ICH IVH BT Craniectomy	ICP and CPP 5 min before, after, and 5 min after oral care	Significant mean increase of 2.13 mmHg in ICP. CPP was not affected. The changes in ICP were not clinically significant. Intensity had no effect on ICP and CPP.	Low

Nursing Intervention Effects on Cerebral Hemodynamics *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Olson, Thoyre, Turner, Bennett, & Graffagnino, 2007)	1	Case study	TBI	ICP and MAP	ICP mean values: Before: -25.63, SD = 8.4 During: -23.0, SD = 9.57 After: 17.30, SD = 7.15: CPT was statistically related to changes in ICP. MAP was not significantly related.	Very low
(Olson, Bader, Dennis, Mahanes, & Riemen, 2010)	30	Prospective case series cohort	TBI SAH ICH HC	ICP before, during, and after chest physiotherapy	Before CPT: M = 13.94 mmHg, SD = 6.40 mmHg During CPT: M = 14.38 mmHg, SD = 6.61 mmHg After CPT: M = 13.7 mmHg, SD = 6.47 mmHg. There was no significant difference controlling for participants and comparing individual ICP values before and during CPT (p = .15), before and after CPT (p = .50) and during and after CPT (p = .67)	Low
(Olson, Thoyre, Bennett, Stoner, & Graffagnino, 2009)	28	RCT	SAH ICH TBI BT IS	ICP before, during, and after chest physiotherapy	Mean ICP: For the control group, before 14.4, during 15.0, and after 15.9 mmHg For the intervention group, before 13.6, during 13.7, and after 14.2 mmHg ICP did not differ significantly between the groups.	Moderate
(Ledwith et al., 2010)	33	Prospective	TBI	ICP, CPP, and PbtO2 after 12 different body positions	ICP response to positioning differs from one patient to the next.	Very low
(Nyholm, Howells, & Enblad, 2017)	28 patients	Prospective case series cohort	TBI	ICP after nursing interventions	Patients with a baseline ICP of ≥ 15 mmHg had a 4.7-times-higher risk for an ICP insult after a nursing intervention. ICP insults were observed when changing a patient's position from lateral to supine (16%) vs. vice versa (4%).	Low
(Bilotta et al., 2008)	41	Prospective observational cohort	Head trauma	HR, MAP, CPP, ICP, EtCO2, SpO2, and V_{MCA} T1 at baseline; T2 at 5 minutes; and T3 10 minutes after endotracheal lidocaine instillation	Lidocaine via ET tube effectively and safely prevents endotracheal suctioning-related systemic and cerebral hemodynamic changes (in particular, the associated increase in ICP and CPP reduction). The optimal dose according to this study was 1.7 ± 0.3 mg/kg.	Low

Nursing Intervention Effects on Cerebral Hemodynamics *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Mathieu et al., 2013)	15	RCT	TBI	MAP, HR, ICP, CPP, BIS, PaCO2, DTC, Vmca, Pmax, Pav, Spo2	0.9% NaCl group: ETS caused a significant elevation in ICP for 5 minutes; the peak was at 1 minute (variation of ICP: 6 ± 2 mmHg, $p < 0.01$) with a significant reduction of CPP (variation of CPP: 2 ± 2 mmHg, $p < 0.01$). Lidocaine group: Variation in ICP: 1 ± 1 mmHg, $p < 0.05$) Variation in CPP: -1 ± 1 mmHg, $p < 0.05$ Aerosolized lidocaine (2 mg/kg) can prevent ETS-related increases in ICP without modifying systemic and cerebral hemodynamics in deeply sedated patients.	Moderate
(Jiang et al., 2015)	237	Patients who underwent craniotomy	Quantitative systematic review with meta-analysis	ICP at different HOB elevations of 0, 10, 15, 30, and 45 degrees	ICP at 30 degrees was not significantly different than at 45 degrees. ICP at 30 and 45 degrees is significantly lower than at 10 and 15 degrees.	High
(Petersen, Petersen, Andresen, Secher, & Juhler, 2016)	9	Prospective cohort	Mixed neurosurgical	MAP, ICP, and CPP in response to 10-degree and 20-degree head-up and head-down tilt and standing (90 degrees)	10-degree and 20-degree head-up tilt reduced ICP to 4.8 (± 3.6) and 1.3 (± 3.6) mmHg. ICP reached -2.4 (± 4.2) mmHg in the standing position. Subsequent MAP changes maintained CPP at 77 (± 7) mmHg in different body positions.	Very low
(Agbeko, Pearson, Peters, McNames, & Goldstein, 2012)	8	Prospective randomized interventional cohort study	Children with TBI	ICP and CPP at different HOB angles	HOB elevation is related to ICP reduction. The higher the baseline ICP, the lower the magnitude of reduction. CPP response was negligible.	Very low
(Schulz-Stubner & Thiex, 2006)	10	Prospective cohort	TBI SAH	Percentage of change from baseline of ICP, CPP, SVR, and CBF at different HOB and PEEP levels	A significant increase in ICP with a reduction in CPP was related to a position change from 30 degrees to flat. At HOB 0 degrees and PEEP = 10 cm H_2O increased ICP, with no significant effect on CPP At HOB 30 degrees and PEEP increase from 5 to 10 cm H_2O, no significant changes in ICP or CPP occured.	Very low

Nursing Intervention Effects on Cerebral Hemodynamics *continued*

Reference	Sample Size	Study Design	Population	Endpoint	Findings	Quality
(Blissitt, Mitchell, Newell, Woods, & Belza, 2006)	20	Prospective cohort	Patients with SAH and vasospasms	CBF at different HOB elevations at 20 and 45 degrees with the same patients	No significant change in CBF at different HOB elevations. ICP significantly and consistently decreased when the HOB was elevated; CPP significantly increased.	Very low
(M. N. Kim et al., 2014)	20	Prospective cohort	Adults with brain injury and healthy adults and matched volunteers	Relative CBF, concurrent changes in oxy- ($\Delta HbO2$) deoxy-(ΔHb), total hemoglobin concentrations (ΔTHC) from left/right frontal cortices were monitored for 5 min at 30 degrees and then at 0 degrees HOB	DCS, measurement of CBF, and near-infrared spectroscopy for measurement of cerebral oxy- and deoxy-hemoglobin concentrations detected differences in CBF and oxygenation.	Very low
(Kung et al., 2013)	13	Prospective cohort	SAH	CBF at different HOB elevations	Changing HOB elevation did not significantly affect CBF. This study suggests that HOB elevation and early mobilization should be considered in patients with vasospasm	Low
(Y. Zhang & Rabinstein, 2011)	19	Prospective cohort	SAH post coiling or embolization	MFV with HOB positions at (30-45) and then at (0-15)	HOB does not significantly affect MFV. HOB elevation had no significant effect on ICP.	Low
(Roth et al., 2017)	n = 10 RXs = 25	Prospective	Neuro-ICU	ICP CPP ptiO2 Baseline, minute, during RX, 15 mins post	Mean ICP, CPP, and ptiO2 did not change significantly comparatively for before, during, and after passive range of motion	Very low
(Roth et al., 2013)	84	Case series prospective cohort	SAH TBI ICH IS BT CA Gunshot Brain edema	ICP CPP MAP HR	Mean ICP before treatment was 11.5 ± 5.1 mmHg with a significant decrease of 1 mmHg during therapy. Persistent ICP reduction after therapy was seen in patients with ICP > 20 mmHg	Low
(Riordan et al., 2015)	80 patients 18 rodents	Retrospective cohort	SAH	Patient control group vs. exercise group Radiographic cerebral vasospasm and symptomatic cerebral vasospasm	Early mobilization with mild exercise significantly reduced the odds of developing symptomatic cerebral vasospasm.	Low
(Olkowski et al., 2013)	25	Retrospective case series cohort	SAH	30-day mortality rate Adverse events	Adverse events occurred during 5.9% of early mobilization training sessions. Mean day-to-start ambulation was 3.2 days (SD = 1.3) after SAH. Early mobilization in SAH was feasible and safe.	Low

Nursing Intervention Effects on Cerebral Hemodynamics *continued*

Reference	Sample Size	Study Design	Population	Endpoint	Findings	Quality
(Karic, Roe, Nordenmark, Becker, & Sorteberg, 2016)	168	Prospective controlled interventional study	SAH	mRS and GOS extended	In poor-grade scenarios, early rehabilitation increased the chance for a better outcome (adjusted OR, = 2.33; 95% CI, 1.04-5.2; $p = 0.039$).	Moderate
(Karic, Sorteberg, Haug Nordenmark, Becker, & Roe, 2015)	37	Prospective observational	aSAH	Discharge ambulatory status	67% of poor-grade vs. 78% of good-grade patients could ambulate by discharge.	Low
(Shimamura, Matsuda, Satou, Nakano, & Ohkuma, 2014)	71	Retrospective case series cohort	SAH	GOS or dementia at 30 days after SAH	GOS at 30 days after the SAH (# of cases) Good Recovery (25) Moderate Disability (22) Severly Disabled (17) Vegetative Survival (2) Dead (5) Revised Hasegawa Dementia Scale (# of cases) 30-21 (27) 20-11 (8) 10-0 (36) Early ambulation correlated with favorable GOS and postoperative nondemential state.	Low

Euvolemia						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Martini et al., 2012)	356	Retrospective	Adults with aSAH	Fluid balance GCS score H & H grade	Patients with positive fluid balance had worse admission GCS scores and H & H grade. There was no difference in mortality or new infarct (adjusted OR: 1.47, 95% CI: 0.85-2.54) between patients with positive and negative fluid balance. Positive fluid balance was associated with increased odds of TCD vasospasm (adjusted OR 2.25, 95% CI: 1.37-3.71) and increased LOS.	Moderate
(Kissoon et al., 2015)	288	Prospective	Infants with aSAH admitted to the NICU between October 2001 and June 2011	DCI	Greater positive net fluid balance is independently associated with a worse functional outcome mRS.	Moderate
(Togashi et al., 2015)	20	Prospective randomized pilot trial	SAH	Protocol adherence to normo or hypervolemia (Nielsen, Jeppesen, Kirkegaard, & Hvas) and CBP or ABP; retention to follow-up	mRS scores: No differences in normo and hypervolemia groups and no differences in CBP and ABP groups. Neuropsychological scores: no differences in NV and HV groups with worse scores in the ABP vs. CBP groups ($p = .04$).	Very low
(Egge et al., 2001)	32	RCT	aSAH	Clinical outcomes, clinically evident and TCD-evident vasospasm, complication, cost, SPECT	There was no difference in 1 year GOS between patients who received full hypervolemic hypertensive hemodilution with therapy vs. normokalemic fluidotherapy. No differences were observed between the groups with respect to clinical or TCD-evident vasospasm. Costs were higher and complications were more frequent for the hyperdynamic therapy group. No differences were evident by SPECT.	High
(Lennihan et al., 2000)	82	RCT	SAH	CBF measured serially during the first 14 days and blood volume measured at baseline and postop day 3. Secondary measures included frequency of symptomatic vasospasm, medical and neurological complications, MAP, pulmonary artery diastolic pressure, central venous pressure, hematocrit level, 24-hour fluid intake, 24-hour net fluid balance, and GOS.	Hypervolemia after aneurysm surgery did not increase blood volume or CBF compared with normal volemic treatment. Fluid administration may be important to avoid hypovolemia after SAH; however, prophylactic therapy does not confer additional benefit. Among subjects, 66% were functionally independent on days 14, 27, and 41. One person in each group died, three people died during hospitalization after completion of the study. GOS scores were not significantly different between the two groups at day 14 or 3 months. There was little change in outcome between 3, 6, and 12 months after SAH.	Moderate
(Joffe, Khandelwal, Hallman, & Treggiari, 2015)	39	Prospective clinical trial	SAH	Circulating blood volume	There was no difference in circulating blood volume between hypervolemia and normovolemia groups ($p < 0.72$).	Very low

Euvolemia *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Hoff, van Dijk, Algra, Kalkman, & Rinkel, 2008)	50	Prospective	aSAH	CBV measurements with normovolemia intervention	Of the 265 CBV measurements, 52% were in the normovolemic range of 60-80 ml/kg; 29% indicated hypovolemia with CBV < 60 ml/kg; and 19% indicated hypervolemia with CBV > 80 ml/kg. There was no association between CBV and daily fluid balance or CBV and cumulative fluid balance. This raises doubt regarding whether fluid management guided by fluid balance is effective in maintaining normokalemia.	Moderate
(Bijlenga et al., 2010)	42	Retrospective analysis	SAH	mRS at 3 months after SAH	Pressure reactivity index equal to or below 0 during the first 48 hours after SAH correlated with survival at 3 months. Positive predictive value was 87.5%. The pressure reactivity index also increased during periods of vasospasm, indicating a loss of autoregulation during that time. In patients with heart rate SAH, optimal CPP shifts toward higher values during vasospasm.	Moderate
(Rasulo et al., 2012)	29	Retrospective analysis of prospectively recorded data	aSAH	Cerebrovascular autoregulation as reflected by CPP GCS Level of disability Death	Good 6-month outcomes were 15 (51.7%; good recovery 7 [24.1%], moderate disability 8 [27.6%]) Poor 6-month outcomes were 14 (48.3%) GCS score and duration of impaired cerebrovascular autoregulation were independent predictors of long-term outcome Other variables (vasospasm, pupillary light response, HTN, and brain CT features) were not predictive of outcomes.	Moderate

VTE Prophylaxis						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Kshettry et al., 2014)	15,968	Retrospective	aSAH	Primary outcomes: DVT and PE Secondary outcomes: Inpatient mortality, discharge disposition, LOS, and total hospital charges	Overall rates of VTE, DVT, and PE were 4.4%, 3.5%, and 1.2%, respectively. Patients who underwent clipping vs. coiling had similar BTE rates. Transesophageal echocardiogram results were associated with pulmonary/cardiac complications, infectious complications, ventriculostomy, and vasospasm. Mean LOS and total inflation-adjusted hospital charges nearly doubled for patients with VTE.	High
(Manoel et al., 2014)	174	Retrospective cohort analysis	SAH	Thrombocytopenia or new ICH	The initiation of pharmacological thrombo-prophylaxis within 24 hours may be safe after aneurysm securement.	Moderate
(Olson et al., 2015).	495	Randomized	Trauma patients in the ICU	DVT by duplex or PE by CT angiography Subcutaneous heparin 5,000 U every 8 hours vs. enoxaparin 30 mg every 12 hours	A regimen of UFH every 8 hours may be noninferior to enoxaparin every 12 hours for the prevention of VTE following trauma. Given UFH's cost advantage, the use of UFH for VTE prophylaxis may offer better value.	High
(Hacker et al., 2012)	522	Retrospective	Neurosurgical	Postoperative hemorrhage, heparin-induced thrombocytopenia, PE, DVT, LOS	Administration of subcutaneous UFH dosed according to risk for thromboembolism does not appear to contribute postoperative hemorrhage among neurosurgical patients.	Moderate
(Zachariah et al., 2016)	241	Retrospective	aSAH with EVDs	PE, DVT within 30 days of admission	Adjuvant chemotherapy prophylaxis (7.5%) was associated with fewer VTEs than lone SCDs (18%). The incidence of cerebral hemorrhage is rare (1/10 therapeutic anticoagulant patients).	Moderate
(Siironen et al., 2003)	170	Randomized, double-blind, single-center clinical trial	aSAH	Unfavorable outcome: GOS and mRS	Enoxaparin had no effect on aSAH outcome. LMWH was associated with increased intracranial bleeding complications and had no beneficial effect on neurological outcome.	Moderate
(Collen, Jackson, Shorr, & Moores, 2008)	7,779	Meta-analysis	Published RCTs and prospective clinical trials of VTE prophylaxis in neurosurgical patients	DVT	LMWH and ICDs are effective in reducing the rate of DVT (LMWH: RR, 0.60; 95% CI, 0.44-0.81; ICD: RR, 0.41; 95% CI, 0.21-0.78). There was no statistical difference in the rate of ICH between therapy with LMWH and nonpharmacologic methods (RR, 1.97; 95% CI, 0.64 to 6.09).	High
(Kinnier et al., 2016)	786	Retrospective	Surgery patients at an academic medical center between May 2012 and December 2013	Adherence to ordered VTE prophylaxis: ambulation, SCDs, chemoprophylaxis	Adherence to ordered VTE prophylaxis is high for the first 24 hours postop (99.7%) but not thereafter (42.5%; $p < 0.001$).	Moderate
(Elpern, Killeen, Patel, & Senecal, 2013)	966 observations	Prospective, observational study	ICU patients who received IPCs	Adherence to ordered IPC devices	Errors in the application of IPC devices were found in 49% of observations. Errors and misapplications included machine not at bedside, pump not working, sleeves not applied, and pump not within recommended pressure range. No IPC prophylaxis: 15% of total observations	Moderate

VTE Prophylaxis *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(M. N. Kim et al., 2014)	29	Descriptive	Nurses caring for 147 patients in the SICU	Nursing concerns: impairment of assessment ability and difficulty with fit/application/ maintenance, skin impairment, patient discomfort, appropriate application. Other problems are listed in the publication.	To ensure appropriate use of DVT prophylactic devices among SICU nurses, systematic education based on information needs should be undertaken.	Low
(Arabi et al., 2013)	798	Prospective cohort analysis	ICU patients	Lower extremity DVT, PE, or both. Secondary endpoint: hospital mortality	VTE occurred in 7.1% of patients and rolled. IPC use was associated with a significantly fewer VTE incidents compared with no mechanical thromboprophylaxis. Graduated compression stockings were not associated with decreased VTE incidents.	Moderate

Endovascular Considerations						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Doerksen & Naimark, 2006)	30 nurses 60 patients	Descriptive quantitative	30 neurosurgical unit nurses, observing 60 patients	Vasospasm nonspecific behavior categories: restless, impulsive, strange, other (atypical behaviors that did not fit into other categories)	83% (24/29 patients) who exhibited nonspecific behaviors had vasospasms.	Very low

Glycemic Control						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Badjatia et al., 2010)	58	Prospective	Poor-grade SAH	Infectious complications	Negative energy balance was associated with infectious complications.	Low
(Badjatia et al., 2015)	229	Prospective	aSAH	HAI	Negative nitrogen balance associated with underfeeding increases risk for HAIs and poor mRS.	Moderate
(Cinotti et al., 2014)	193	Retrospective	Patients with mechanical ventilation and aSAH	Early VAP	Early enteral nutrition was a modifiable risk factor for VAP.	Moderate
(Yuan et al., 2015)	n/a	Meta-analysis	Stroke	Lung infection	23 risk factors for lung infections were identified. The top five, ranked by OR (95% CI), were multiple vertebrobasilar stroke, 22.99 (4.04, 130.83); NIHSS score > 15 points, 14.63 (8.54, 25.08); mechanical ventilation, 10.20 (7.15, 14.57); nasogastric tube use, 9.87 (6.21, 15.70); and dysphagia, 7.50 (2.60, 21.65). Preventive measures should be taken against these risk factors to reduce incidence of lung infection.	High
(Titsworth et al., 2013)	2,334	Prospective	Stroke (all)	Dysphagia protocol compliance and pneumonia	Dysphagia screens were associated with decreased prevalence of pneumonia.	Moderate
(Sellars et al., 2007)	412	Prospective	All stroke (ischemic, 94.9%)	Pneumonia	Pneumonia after stroke is associated with older age, dysarthria, severity of poststroke disability, cognitive impairment, and an abnormal water swallow test result. Simple assessment of these variables could be used to identify patients at high risk for pneumonia after stroke.	Moderate
(Abecassis et al., 2017)	360	Retrospective	SAH	Respiratory and swallow function at 1 year and most recent clinical follow-up	Aneurysm location had little effect on immediate and long-term swallow and respiratory function.	
(Rhie et al., 2016)	64	Prospective	Patients with first ruptured aSAH	Videofluoroscopic Dysphagia Scale score	Oral transit time was delayed in 46.8% of the patients. Residues in the vallecular space and/or pyriform sinus were found in 50% of patients. Pharyngeal transit time was prolonged in 39% of patients.	Low
(van Donkelaar et al., 2016)	258	Retrospective	Patients with aSAH admitted to the ICU who survived > 48 hours	DCI infarct, mRS > 3, 3-month mortality	High glucose was associated with DCI; high lactate was associated with mRS > 3 or 3-month mortality.	High
(Kurtz et al., 2014)	28	Prospective	Coma and SAH	Inpatient mortality and metabolic distress; LPR	Increased glucose variability is associated with cerebral metabolic distress and mortality.	Very low
(Barletta, Figueroa, DeShane, Blau, & McAllen, 2013)	42	Retrospective	SAH, H & H grade ≥ 3	Cerebral infarction	Glucose variability is a significant predictor of cerebral infarction.	Low
(L. Bian et al., 2013)	239	Prospective	aSAH without diabetes	1-year mortality	Higher admission and general glucose levels were associated with 1-year mortality.	High

Glycemic Control *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Lin, Lin, Lee, Ma, & Lin, 2007)	457	Retrospective	Inpatients receiving total parenteral nutrition during 2004	Infection, cardiac complications, renal failure, respiratory failure, death	Hyperglycemia increased the odds ratios for infection, cardiac complications, renal failure, respiratory failure, and death.	Moderate
(Okazaki et al., 2018)	122	Retrospective	aSAH	Poor neurological outcomes	Increased glucose variability was associated with poor neurological outcomes.	Moderate
(Pasternak et al., 2008)	1,000	Retrospective	Patients with aSAH enrolled in the IHAST	NIH stroke score, ICU LOS	Intraoperative hyperglycemia was associated with long-term changes in cognition and gross neurologic function and increased ICU LOS.	High
(Schlenk, Vajkoczy, & Sarrafzadeh, 2009)	178	Prospective	aSAH	12-month GOS	Blood glucose level > 7.8 mmol (135 mg/dl) was associated with low GOS and mortality.	Moderate
(Ooi et al., 2012)	1,459	Meta-analysis	Neurological and neurosurgical patients undergoing insulin protocols	Infection, neurological outcome, hypoglycemia, mortality	Tight glycemic control decreased infection risk and improved neurological outcome despite increased hypoglycemic incidents.	High
(Latorre et al., 2009)	332	Prospective	aSAH with hyperglycemia	mRS ≥ 4 at 3-6 months, vasospasms	AHM resulted in good glucose control and decreased the odds of poor outcomes. AHM did not affect rate of vasospasms.	Moderate
(Bilotta et al., 2007)	78	Prospective	Clipped aSAH	Infection, vasospasms, 6-month mRS >3 and mortality	Patients using conventional glycemic control had a higher infection rate than patients receiving IIT. There was no benefit regarding vasospasms, neurological outcome, and mortality at 6 months for patients undergoing IIT.	Low
(A. H. Kramer, Roberts, & Zygun, 2012)	1,248	Meta-analysis	Neurocritical care	Mortality, poor neurological outcomes (mRS ≥ 3, GOS 1-3, hypoglycemia)	IIT did not decrease mortality but increased risk for hypoglycemia.	Moderate
(Thiele et al., 2009)	834	Retrospective	aSAH	Hypoglycemia, mortality	SGC did not affect mortality and increased incidence of hypoglycemia. Hypoglycemia was highly associated with mortality, but overall mortality was unaffected possibly due to positive effects of SGC being offset by hypoglycemic effects.	Low
(Ling, Li, & Gao, 2012)	13,978	Meta-analysis	Critically ill patients in an RCT that compared intensive glucose control to target glucose < 6.1 mmol (110 mg/dl)	Inpatient 90- and 180-day mortality, sepsis, new need dialysis	Intensive glucose control did not improve mortality and was associated with risk of hypoglycemia.	High
(Naidech et al., 2010)	172	Prospective	SAH	Vasospasm, cerebral infarct, and mRS ≥ 3	Hypoglycemia was associated with vasospasm, cerebral infarct, and mRS ≥ 3; increased glucose variability was associated with worse neurological outcome.	Moderate

Pain Management						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Morad, Tamargo, & Gottschalk, 2016)	46	Prospective	aSAH	Pain score, location, and analgesic consumption	Headache rate was 76%. Despite analgesia, pain reported by conscious patients with SAH often is severe and persists throughout hospitalization.	Low
(Glisic et al., 2016)	77	Retrospective observation	aSAH	Duration and pattern of headaches and pain severity; analgesics used	Headache after aSAH often was severe, necessitating multiple opioid and nonopioid analgesics. Many patients reported persistent headaches and inadequate pain control.	Low
(Dorhout Mees, Bertens, van der Worp, Rinkel, & van den Bergh, 2010)	108	RCT	aSAH cohort of the MASH-2 trial	HA level (11-point scale)	Elevated serum magnesium levels were associated with slightly less severe HA and less opioid use.	Moderate
(Asadi-Noghabi, Gholizadeh, Zolfaghari, Mehran, & Sohrabi, 2015)	106	Prospective	ICU nurses	Critical care pain observational tool (CPOT) pain assessment checklist for RNs Pain diagnosis Report pain to physician Administration of pharmacologic medications or nonpharmacologic interventions Document pain Document pain relief measures (meds) Document pain relief measures (non-meds) Evaluate pain-relief measures Reevaluate relief measures	There was no change in recorded pain levels. Use of the CPOT increased nurses' sensitivity to pain in patients with low consciousness level.	Low
(Dorhout Mees et al., 2010)	108	RCT	aSAH cohort of the MASH-2 trial	HA level (11-point scale)	Elevated serum magnesium levels were associated with slightly less severe HA and less opioid use.	Moderate
(Puntillo et al., 2004)	5,957	Prospective descriptive	Inpatients undergoing turning, CVC insertion, wound drain removal, wound care, ET suctioning, femoral sheath removal	Exhibited pain behaviors pre-, intra-, and post-procedure	In addition to verbal responses, facial expressions (grimace, frown, wince, eyes closed, eyes wide open with raised eyebrows, clenched teeth, wide-open mouth, grin/smile) and body movement (no movement, rigid, arching, clenched fist, shaking, withdrawing, splinting, flailing, pick/touching site, restlessness, rubbing/massaging, repetitive movements, defensive grabbing, pushing, guarding) can be used to assess pain.	Moderate

Pain Management *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Lindberg & Engstrom, 2011)	6	Qualitative	ICU nurses in northern Sweden	Pain assessment and treatment skill descriptions	Interviews yielded a primary theme: "A good relationship with the patient is a prerequisite for successful pain relief management." Subcategories: Understanding the unique patient's experience of pain Succeeding or failing to relieve pain Trusting the patient and getting physician help Treating pain when a pharmacologic agent is not sufficient	Very low
(Nichols, Nadpara, Taylor, & Brophy, 2016)	309	Retrospective	Neurocritical care	Pain intensity with IV vs. PO APAP	IV APAP was more effective than PO APAP in relieving pain within 30 minutes of dosing, but the difference was not sustained over 6 hours. No differences were noted in opioid use between IV and PO APAP within 6 hours before and 6 hours after dosing.	Low
(Dhakal et al., 2015)	53	Retrospective	Patients with aSAH treated with GBP	Safety and tolerability of GBP	GBP was safe and tolerable in this population.	Very low
(Karabinis et al., 2004)	161	Prospective	Patients with acute brain injury in 17 hospitals across 6 countries	ICP SAS score 1-3 Pain Intensity scale (significant ≥ 3)	Fentanyl and morphine groups were optimally sedated significantly longer than the remifentanil group. Patients who received remifentanil were extubated significantly earlier than the morphine group ($p < 0.001$).	Low
(Zeiler, AlSubaie, Zeiler, Bernard, & Skrobik, 2016)	173	Survey	173	Prescribed analgesia in neuro-ICUs	APAP is the most common first-line analgesic (49.1% of patients). Opiates are second-line treatment for 31.5% of patients. However, 33% of patients received no second agent. In the 2.3% with demyelinating disease, gabapentin was the most likely second analgesic (50%).	Moderate

Seizure Management						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Panczykowski et al., 2016)	353	Retrospective	SAH	Seizures pre and post prophylactic AED use	Prophylactic AEDs do not significantly reduce risk for seizure.	Moderate
(Rosengart, Schultheiss, Tolentino, & Macdonald, 2007)	3,552	Retrospective from prospective randomized double-blind, placebo-controlled trials conducted in 162 neurosurgical centers and 21 countries between 1991 and 1997	SAH	GOS Morbidity	AED use was associated with: Worse GOS (OR 1.56, $p = 0.003$) Vasospasm (OR 1.87, $p < 0.001$) Neurological deterioration (OR 1.33, $p = 0.04$) Elevated temperature (OR 1.36, $p = 0.03$)	High
(Naidech et al., 2005)	527	Prospective	Patients with SAH in the ICU	Poor functional outcome (mRS ≥ 4) Cognitive status (assessed with TICS)	PHT burden was associated with poor functional outcome at 14 days (OR, 1.5 per quartile; $p < 0.001$) PHT burden was not associated with poor functional outcome at 3 months ($p = 0.09$). PHT burden was associated with TICS scores at discharge ($p < 0.001$) and 3 months ($p = 0.003$).	Moderate
(Butzkueven et al., 2000)	412	Retrospective	SAH	Seizure GOS	6-week GOS predictors: Initial GCS of < 6 (OR, 13.7; $p < 0.01$) Onset seizure (OR, 7.8; p = 0.04) Late seizure predictors: Rebleeding (OR, 94; $p < 0.01$) Onset seizures (OR, 27; $p < 0.01$)	Moderate
(Chumnanvej, Dunn, & Kim, 2007)	453	Prospective	SAH	Seizure rate PHT complications	There was no difference in the seizure rate between entire hospitalization prophylaxis and 3-day prophylaxis patients. Hospitalization SZ rates ($p = 0.603$) included entire admission PHT (1.3%) and 3-day admnistration (1.9%). Follow-up SZ rates ($p < 0.573$) include entire admission PHT (5.6%) and 3-day administration (4.6%). 3-day PHT regime significantly ($p = 0.002$) reduced PHT complications.	Moderate
(Murphy-Human, Welch, Zipfel, Diringer, & Dhar, 2011)	442	Prospective	SAH	Seizure rate	There was a difference in seizure rate for 3-day levetiracetam (8.3%) vs. 7-day PTH (3.4%; $p = 0.03$).	Moderate
(Young, Jordan, & Doig, 1996)	49	Retrospective	Nonconvulsive seizures	Mortality	Variables associated with mortality Seizure duration ($p = 0.0057$) Delay to diagnosis ($p = 0.0351$)	Low

Thermodynamic						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(C. L. Kramer, Pegoli, Mandrekar, Lanzino, & Rabinstein, 2017)	584	Retrospective	aSAH	Poor outcome: 6- to 12-month mRS ≥ 3	Early-onset fever and number of fever hours are associated with poor functional outcome.	High
(Suehiro et al., 2016)	62	Prospective	aSAH	DCI, poor outcome: GOS (Cohen & Hilligoss, 2010) score ≤ 3	Fever during the early postoperative period is associated with DCI and poor outcome.	Moderate
(Douds et al., 2012)	186	Retrospective	aSAH	DND Good outcome: GOS > 3	Fever was associated with DND. Number of days of fever and number of infections were negatively associated with good outcomes.	High
(G. Zhang, Zhang, & Qin, 2011)	155	Prospective	aSAH	In-hospital mortality	Fever was associated with increased in-hospital mortality risk.	Moderate
(Springer et al., 2009)	232	Prospective	aSAH	Cognitive impairment (score ≤ 30), Barthel Index score, Lawton Scale score, Sickness Impact Profile	Cognitive impairment at 3 months was 27% and at 12 months was 21%. Risk factors for cognitive impairment at 12 months: anemia treated with transfusion (AOR, 3.4; $p = 0.006$), any temperature level higher than 38.6 °C (AOR, 2.7; $p = 0.016$), and DCI (AOR, 3.6; $p = 0.01$). Among patients with cognitive impairment at 3 months, improvement at 1 year occurred in 34% and was associated with > 12 years of education and the absence of fever higher than 38.6 °C during hospitalization ($p = 0.015$).	Moderate
(Todd et al., 2009)	1,000	Prospective	Clipped aSAH, WFNS grades 1-3, subgroup of the IHAST trial population	Primary: GOS score. Secondary: Rankin disability score, NIHSS, Barthel Index score, neuropsychological testing (composite and any tests), current place of residence (home, acute care hospital, chronic care, or rehabilitation)	Fever with and without infection is associated with worsened outcome in clipped aSAH. Fever also was associated with infection, but a causative factor vs. marker for other events could not be identified.	High
(Oddo et al., 2009)	18	Retrospective	Patients with aSAH undergoing cerebral microdialysis and ICP monitoring	Poor GOS score (≤ 3)	Fever control is associated with decreased cerebral metabolic distress (LPR > 40) in both poor- and good-outcome patient groups regardless of ICP.	Very low
(Fischer et al., 2015)	51	Retrospective	Patients in the ICU with severe cerebrovascular disease (H & H grade 3-5, spontaneous ICH with GCS score < 10, cerebral infarct with NIHSS > 15 requiring ICU)	Favorable neurological outcome (mRS 0-2)	Patients with lower endovascular cool bath temperatures reflecting high cooling activity had a more favorable neurological outcome at 180 days (mRS 0-2) than patients with low cooling activity ($p < 0.05$). No significant correlation between cool bath temperature and inflammatory markers was found.	Low

Thermodynamic *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Badjatia et al., 2010)	120	Prospective	Febrile (temperature > 38.3 °C), nontrauma patients with SAH admitted to Columbia University Medical Center	Tracheostomy, ICU LOS, hospital LOS, mRS	AFC was associated with a higher rate of tracheostomy and longer ICU LOS, but no difference was found in average hospital LOS vs. conventional fever control. AFC was associated with improved outcomes at 12 months. Missing data on 3 of 40 AFC patients were carried through from existing 3-month data to 12-month data.	Very low
(Abdullah & Husin, 2011)	24	Prospective	Intracranial hemorrhage	6- and 12-month mRS	There was a statistically significant improvement at 6-month and 1-year follow-up using the mRS score in the hypothermia group.	Very low
(Hindman, Bayman, Pfisterer, Torner, & Todd, 2010)	441	Prospective	Patients with aSAH who underwent temporary clipping	24-hour (short-term) and 3-month (long-term) GOS score	Intraoperative hypothermia did not affect short- or long-term outcomes.	Moderate
(Anderson et al., 2006)	1,000	Prospective	Patients with aSAH who underwent clipping and bled ≤ 14 days prior to surgery, WFNS grades 1-3, IHAST trial	Primary: GOS score. Secondary: Composite score from Benton visual retention, controlled oral word association, Rey-Osterrieth Complex Figure, grooved pegboard, trial-making tests, Mini-Mental State Examination	There was no difference in the incidence of impairment between hypothermic and normothermic groups.	High
(Todd, Hindman, Clarke, & Torner, 2005)	1,000	Prospective	Patients with aSAH who underwent clipping and bled ≤ 14 days prior to surgery. WFNS grades 1-3 (good grade). IHAST trial	Primary: GOS score	Intraoperative hypothermia did not improve neurologic outcome after craniotomy among patients with good-grade SAH.	High
(Kuramatsu et al., 2015)	36	Prospective	Poor-grade (H & H grade > 3 and WFNS score > 3) patients with SAH	Macro vascular spasm, DCI, 6-month mRS (favorable outcome, mRS 0-2)	Early and prolonged TH was associated with a reduced degree of macrovascular spasm and significantly decreased DCI occurrence. Favorable outcome was twice as frequent in TH-treated patients (66.7 vs. 33.3% of non-TH, but p = 0.06).	Very low
(Seule et al., 2014)	20	Retrospective	Patients with SAH admitted to the ICU at University Hospital Zurich	Cerebral blood flow velocity	Therapeutic hypothermia decreased Doppler blood flow velocity in both intracranial hypertension and DCI cases.	Very low
(Seule, Muroi, Mink, Yonekawa, & Keller, 2009)	100	Prospective	Consecutively admitted patients with SAH who developed increased ICPs	Good functional outcomes: GOS score 4-5	Prolonged systemic hypothermia may be considered as a last-resort option for a carefully selected group of patients with SAH and increased ICPs or vasospasms who are resistant to conventional treatment. Complications associated with hypothermia require elaborate protocols in general ICU management.	Very low

Thermodynamic *continued*						
Reference	**Sample Size**	**Study Design**	**Population**	**Endpoint**	**Findings**	**Quality**
(Karnatovskaia, Lee, Festic, Kramer, & Freeman, 2014)	35	Retrospective	Patients with aSAH who underwent decompressive hemicraniectomy and/or prolonged hypothermia for refractory ICP	Mortality, mRS, days of mechanical ventilation, hospital LOS	There was difference in mRS and mortality for patients who underwent TH. TH was associated with longer mechanical ventilation and hospital LOS.	Very low
(Anei, Sakai, Iihara, & Nagata, 2010)	120	Retrospective	aSAH	Transfer or 3-month mRS	TH did not improve outcomes.	Very low

Table Abbreviations Key

AED, anti-epileptic drug; AFC, advanced fever control; AHM, aggressive hyperglycemia management; AOR, adjusted odds ratio; APAP, acetaminophen; aSAH, aneurysmal subarachnoid hemorrhage; BIS, bispectral index; BT, brain trauma; CBF, cerebral blood flow; CBP, concurrent conventional blood pressure; CI, confidence interval; CPOT, Critical Care Pain Observation Tool; CPP, cerebral perfusion pressure; CPT, chest physiotherapy; CSF, cerebrospinal fluid; CUSAHOP, Columbia University Subarachnoid Hemorrhage Outcomes Project; DBP, diastolic blood pressure; DCI, delayed cerebral ischemia; DCS, diffuse correlation spectroscopy; DIND, delayed ischemic neurological deficit; DND, delayed neurological deficit; DTC, transcranial doppler; DVT, deep vein thrombosis; ETCO2, end-tidal CO_2; ETS, endotracheal suctioning; EVD, external ventricular drainage; FY, fiscal year; GBP, gabapentin; GCS, Glasgow Coma Scale; GOS, Glasgow Outcomes Scale; H & H, Hunt and Hess; HA, headache; HAI, hospital-acquired infection; HC, hydrocephalus; HOB, head of bed; HR, heart rate; ICD, intermittent compression device; ICH, intracerebral hemorrhage; ICP, intracranial pressure; ICU, intensive care unit; IHAST, Intraoperative Hypothermia and Aneurysm Surgery Trial; IIT, intensive insulin therapy; IPC, intermittent pneumatic compression; IS, ischemic stroke; ISS, Injury Severity Score; IV, intravenous; IVH, intraventricular hemorrhage; LD, lumbar drainage; LMWH, low-molecular weight heparin; LOS, length of stay; LPR, lactate/pyruvate ratio; MAP, mean arterial pressure; MASH-2, Magnesium for Aneurysmal Subarachnoid Haemorrhage Trial; MFV, mean flow velocity; mRS, Modified Rankin Scale; NICU, neonatal intensive care unit; NIHSS, NIH Stroke Scale; NIS, National Inpatient Sample; OAG, Oral Assessment Guide; PaCO2, partial pressure of CO_2; PbtO2, brain tissue oxygen; PC, palliative care; PCE, palliative care encounter; PE, pulmonary embolism; PEEP, positive end-expiratory pressure; PHT, phenytoin; Pmax, maximal pressure; PO, by mouth; RCT, randomized controlled trial; SAH, subarachnoid hemorrhage; SBP, systolic blood pressure; SCD, sequential compression device; SDH, subdural hematoma; SGC, strict glucose control; SICU, surgical intensive care unit; SVR, systemic vascular resistance; TBI, traumatic brain injury; TCD, transcranial Doppler; TH, therapeutic hypothermia; TICS, Telephone Interview for Cognitive Status; UFH, unfractionated heparin; VAP, ventilator-acquired pneumonia; VMCA, mean velocity of cerebral artery blood flow; VPS, ventriculoperitoneal shunt; VTE, venous thromboembolism; WFNS, World Federation of Neurologic Surgeons Scale.