Nursing Care of the Patient with Aneurysmal Subarachnoid Hemorrhage

A Clinical Practice Guideline

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Introduction

Aneurysmal subarachnoid hemorrhage (aSAH), a cerebrovascular event, occurs when blood enters the subarachnoid space due to cerebral aneurysm rupture. Although aSAH is often grouped with other cerebrovascular diseases such as ischemic stroke and intracerebral hemorrhage (ICH), the treatment and care of aSAH differs from other strokes.[1,2] The unique nature of aSAH necessitates specific nursing considerations and interventions. This clinical practice guideline (CPG) addresses care considerations based on evidence from seminal publications and the most recent decade of research (2013-2022) at the outset of the writing group. The specific methodology by which this and all AANN CPGs and evidence-based clinical reviews are conducted is detailed in the AANN CPG methodology manuscript.[3] Refer to Appendix I for MeSH search terms.

Background

Worldwide, stroke is among the top three causes of morbidity and mortality.[4,5] Hemorrhagic stroke accounts for 37.6% of all stroke types (ICH 27.9%, subarachnoid hemorrhage [SAH] 9.7%) and morbidity (64.5 million disability-adjusted life years [DALYs]/annum) exceeds that of ischemic stroke (59.1 million DALYs/annum).[5] Additionally, hemorrhagic stroke mortality (2.8 million/annum) exceeds ischemic stroke mortality (2.7 million/annum).[5] These statistics demonstrate that while hemorrhagic strokes account for a minority of all strokes, death and disability exceeds that of the more prevalent ischemic stroke (62.4% of all strokes).

In the United States, stroke statistics differ from reported international numbers. A 2024 US-specific, cross-sectional analysis of the most recent Global Burden of Disease study reported a quintuple increase in aSAH incidence of 15% (0.07/0.46 million),[6] up from a historically reported 3%.[7] Given the intensive care needs and long-term disability associated with aSAH,[5,8-12] coordinated expert nursing care is crucial to ensure optimal outcomes.[13-17]

Results

Morbidity and Mortality

Which aSAH scales (H&H, Fisher, mFisher, WFNS, mRS, NIHSS) help predict recovery and treatment efficacy during hospitalization and in the long-term?

Multiple scales are used to grade aSAH severity. Common severity classifications include the Hunt and Hess (H&H), original Fisher, modified Fisher (mFisher), and World Federation of Neurosurgical Societies (WFNS) scales. They are used to predict morbidity and mortality in both the short- (acute phase) and long-term. Morbidity and functional outcomes are frequently documented via the modified Rankin Scale (mRS), which quantifies level of disability. The National Institutes of Health Stroke Scale (NIHSS), commonly used for ischemic stroke and ICH severity assessment, is understudied in the setting of aSAH, and more research is needed to determine its predictive effectiveness in this population.

There were no studies that assessed the inter-rater reliability of any of the scales. Nor were there any primary assessments of the scales' predictive abilities. The literature principally employed these scales as predictors for various primary outcomes, rather than as primary endpoints themselves.

Hunt and Hess Scale

The Hunt and Hess grading scale is a five-point categorical scale that increases in severity from asymptomatic to deep coma. Published research focuses on the relationships between H&H grades and different outcome indicators. One prospective study (N=1,200) found H&H grade predictive of in-hospital death.[18] Grade 1 had an associated mortality rate of 3.5% and grade 5 a rate as high as 70.5%. Higher H&H grade on admission and prior to aneurysm treatment was also found predictive of death up to 3 years post injury. Hunt and Hess grades of at least 4 were associated with death at 3 months (N=476; $P<.0001$)[19] and 12 months (N=609; $P<.001$).[20] A cohort study of high-grade aSAH (H&H 4, n=145; H&H 5, n=124) patients found the cumulative survival rate at 12 months for grade 4 was 63% and only 27% for grade 5 patients; results were similar at 3 years (61% and 26%, respectively; P values not reported).[21]

In a multivariate analysis (N=116), a higher H&H grade was associated with increased need for endovascular

vasospasm retreatment (P=.02).[22] A higher H&H grade was also found to be associated with increased incidence of delayed cerebral ischemia (DCI) in aSAH patients with hydrocephalus on admission (N=227; P=.000)[23] and cerebral infarction (N=423, P=.001; N=225, P=.048).[24,25] A meta-analysis of 2,470 patients concluded a higher H&H grade was associated with increased rebleeding risk (P<.0001).[26] One cohort study of 202 patients found higher H&H grades to be predictive of shunt-dependent hydrocephalus (P<.001) as well as a worse Glasgow Outcome Scale (GOS) score (P=.048).[27]

In multiple risk prediction models, inclusion of the H&H scale showed improved predictive ability for functional outcomes compared to radiographic findings alone.[24] A prospective cohort study (N=297) found higher admission H&H grades increased the likelihood of mechanical ventilation duration greater than 48 hours in grade 4 aSAH (P<.001) and greater than 7 days in grade 5 aSAH (P<.001).[28] Hunt and Hess grade 3 was associated with extubation failure compared to grades 1 and 2 (N=107; P=.005).[29] Higher H&H grades were found to be predictive of poor outcomes in general (N=62; P=.022),[30] and multiple studies found associations between higher H&H grades and decreased functional outcomes (mRS>2)[23,31-33] and (mRS>3).[20,24,34-36] Lower intensive care unit (ICU) admission H&H grades were associated with better mRS scores at 12 months in aSAH patients receiving specialized neurorehabilitation (N=250; P<.005).[37]

Fisher Scales

The original Fisher grading scale and mFisher scale are radiographic-based scoring methods that quantify the diffusion of a hemorrhage. The original Fisher scale comprises 4 points, ranging from 1 (no SAH) to 4 (diffuse SAH with intraventricular hemorrhage [IVH]), with no difference in IVH designations between scores 2 and 3. The mFisher scale comprises 5 points, ranging from 0 (no SAH) to 4 (diffuse SAH with IVH), and differentiates between with IVH and without IVH within successive scores. Both are widely studied as outcomes predictors.

Higher original Fisher grades were found predictive of aneurysmal rebleed risk and mortality (P=.0004 and P<.001, respectively),[26,38] and findings from a prospective observational study (N=476) evidenced an association between an original Fisher grade greater than 4 and mortality at 3 months (P<.0001).[19] A low original Fisher grade was reported predictive of favorable (mRS≤2) functional outcomes (N=104; P<.001),[39] and in patients older than 60 years with H&H grades 4-5, prognosis was better for those with original Fisher grades 1-2 (N=104; P=.025).[40] Two studies (N=121, N=926) reported a higher original Fisher grade was associated with poor functional outcomes (mRS≥3; P=.005 and P=.000).[32,35] Additionally, the original Fisher grade was found predictive of language deficits in anterior circulation aSAH patients presenting with an H&H grade less than 4, with one cross-sectional retrospective study (N=248) showing original Fisher grades 1-2, compared to control, were more likely to have deficits in written comprehension (P<.001), oral reading (P=.028), and semantic and phonologic fluency (P<.001).[41] In the same study, original Fisher grades 3-4, compared to control, were more likely to experience the same deficits as grades 1-2 (P<.001), with the addition of difficulty naming (P=.004).[41] The original Fisher grades 3-4, compared to grades 1-2, had an increased likelihood of language deficits including oral comprehension (P=.006), repetition (P=.031), naming (P=.033), semantic fluency (P=.003; P=.007), and phonologic fluency (P=.010).[41] Original Fisher grades 3-4 also were associated with higher fever burden (N=194; P>.026).[34] One retrospective cohort (N=118) found a lower original Fisher grade was associated with increased likelihood of undergoing early surgery (N=118; P=.04).[42]

Similar to the original Fisher, higher mFisher grades showed increased risk of aneurysmal rebleed and mortality (P<.001),[43] and the findings from a national database study (N=1,200) showed mFisher was predictive of in-hospital death (P=.03)[18] Additionally, in adults older than 60 years, higher mFisher scores predicted poor functional outcome at 1 year (mRS<3; P=0).[36] One retrospective chart review (N=373) revealed a lower mFisher grade was associated with excellent outcomes (mRS 0-1; P<.01) and a higher mFisher grade with outcomes other than excellent (mRS>1; P<.01) at 1 year post ictus in univariate analysis but not in multivariate models.[44] A prospective cohort study (N=297) found mFisher grade 4 on admission was associated with increased likelihood of mechanical ventilation duration greater than 48 hours and greater than 7 days compared to those presenting as grade 3 (P<.001).[28] Lastly, one retrospective study (N=202) found higher mFisher grades predictive of shunt-dependent hydrocephalus (P<.001).[27]

World Federation of Neurosurgical Societies

The World Federation of Neurosurgical Societies grading scale score (1=best, 5=worst) is derived by combining the Glasgow Coma Scale (GCS) score with motor deficit scores. Studies demonstrate WFNS grades as predictors of favorable[39,42] and unfavorable functional outcomes, including death,[32,40,45-49] inability to follow commands,[48] and the likelihood of early surgery.[42] A retrospective study (N=381) evidenced WFNS grades 1-3 after neurologic resuscitation were predictive of excellent outcomes (mRS 0-1; P<.0001) at 1 year.[44] Another study found the WFNS scale was associated with unfavorable outcomes (N=423; P<.001) and cerebral infarction (P≤.001) and was found superior in predicting functional outcomes compared to radiographic findings alone.[24]

Modified Rankin Scale

The mRS is another categorical severity scale (0=no disability, 6=death). Its effectiveness in assessing functional outcomes in the acute setting is well established in stroke populations. Long-term outcome mRS associations have been studied less, and the search decade only yielded two studies. One prospective study of 250 aSAH survivors discharged to a specialized neurorehabilitation center found lower hospital discharge mRS score was associated with improved mRS at 12 months (P<.001).[37] The second prospective study (N=168) evidenced an association between mRS 3-5 and increasing numbers of cognitive domain deficits present at 1 year post aSAH (P=.002).[48]

Recommendation

The H&H, original and mFisher, WFNS, and mRS scales are important predictors of neurological insult severity, complications, and outcomes after aSAH. Since assessment scores are commonly referred to during routine clinical care, nurses should be familiar with the various scales and their implications (strong recommendation, high-quality evidence).

Palliative Care

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

Palliative care is defined as "an interdisciplinary care delivery system designed to anticipate, prevent, and manage physical, psychological, social, and spiritual suffering to optimize quality of life for patients, their families, and caregivers."[50] Because aSAH has high mortality, high short- and long-term morbidity, and loss of productivity despite maximal treatment,[1] patients and families may benefit from palliative care interventions. Palliative care optimally occurs in conjunction with, not in lieu of, disease-directed or life-prolonging treatment and focuses on symptom relief, effective communication about care goals, the alignment of treatment with patient and family preferences, emotional support, and planning for transitions.[51,52]

The literature on aSAH patient and family satisfaction and palliative care was scant, and most evidence supporting the benefits of integrating palliative care was derived from oncology studies. Research suggested that providers struggle with discussing treatment options, including palliative care, in the setting of poor prognosis.[53] Three studies were found related to palliative care and patient and family satisfaction in stroke care. A longitudinal cohort study (N=91) implemented a daily palliative care needs checklist and examined family perspectives of ICU care and long-term family and patient outcomes. Families of patients who died outside the hospital were shown to have decreased satisfaction with care and decision making as the mRS score increased. Higher satisfaction was seen in families of patients who died in the hospital, possibly due to increased attention from the healthcare team.[51,54] A cross-sectional survey of stroke surrogate decision makers (N=79; aSAH not reported) found family perceptions of the end-of-life care provided to their loved ones were generally high.[55] One qualitative stroke study (N=15) found family perception of palliative care was positive, chiefly related to satisfaction with pain and dyspnea management, provision of information, and facilitated decision making.[56] Of note, the joint American Heart Association (AHA) and American Stroke Association (ASA) policy statement includes recommendations for palliative care for stroke patients.[53]

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).

Cerebrospinal Fluid Management

Past aSAH CPGs have included cerebrospinal fluid (CSF) management. This iteration of the aSAH CPG defers CSF management to the Evidence-Based Clinical Review: Intracranial Monitoring[57] and the External Ventricular Drain Monitoring,[58] Intraparenchymal Monitoring,[59] and Brain Tissue Oxygenation ($PbtO_2$) Monitoring[60] quick guides, as they cover CSF issues in detail.

Nursing Interventions

What effects do nursing interventions have on cerebral hemodynamics?

Management of the aSAH patient during the neurocritical care phase is recognized as one of the most critical components impacting short- and long-term patient outcomes.[2] Medical and pharmacological interventions are readily described across the literature, but there is a lack of research validating the clinical nursing care of these complex patients. Studying nursing care is feasible but requires synchronization and detailed analysis to isolate the effect of specific nursing care on patients' intracranial pressure (ICP).[61,62] Of note, research primarily documents outcomes for mixed neurocritically ill populations that may include but are not limited to aSAH.

Oral Care

There is consensus among healthcare providers that oral health affects systemic health. Oral care is a well-established intervention to reduce the risk for secondary infections including ventilator-associated pneumonia

among critically ill patients. Specific studies in neuroscience and aSAH patients are lacking, especially with regard to effects on ICP. Oral care is safe to use in mixed populations in the neurocritical care setting regardless of the duration intensity or type of product used.[62,63] Though oral care is associated with mild increases in ICP and mean arterial pressure (MAP) regardless of duration or intensity, cerebral perfusion pressure (CPP) is not significantly affected. Importantly, the differences in ICP are not clinically significant and do not require intervention.[62,63] Research did not delineate type and frequency of oral care that proved beneficial.

Recommendation
Routine oral care for neurocritically ill patients is recommended (strong recommendation, moderate-quality evidence). This recommendation remains unchanged from the previous 2018 AANN aSAH CPG, as no new studies meeting inclusion criteria were found.

Chest Physiotherapy
Adequate oxygenation and cerebral perfusion are important to minimize secondary ischemia after aSAH. Chest physiotherapy (CPT) is used to promote pulmonary hygiene and improve oxygenation by opening the alveoli and mobilizing respiratory secretions for more effective clearance of mucus. Chest physiotherapy can result in a transient rise in ICP. However, it is considered safe in neurocritical care patients.[64] No recent studies were found that were specific to SAH patients. Two randomized controlled trials (RCTs) specifically addressed CPT in neurocritically injured patients with ICP monitoring. In the first study (N=46), manual CPT was associated with statistically significant transient increases in ICP and hemodynamics vs mechanical CPT (P=.01).[65] In the second RCT (N=60), administration of lidocaine or dexmedetomidine was effective in blunting the rise of ICP in response to CPT followed by tracheal suction without adverse effects on MAP or CPP. Of note, none of the patients in the study had elevated ICP at the time of administration, which limits applicability in patients with existing intracranial hypertension.[66]

Recommendation
Chest physiotherapy is recommended when clinically indicated (strong recommendation, moderate-quality evidence). This recommendation remains unchanged from the previous 2018 AANN aSAH CPG, as few additional studies were found.

Endotracheal Suctioning
Endotracheal suctioning (ETS) is a common and necessary nursing intervention for mechanically ventilated patients. Historically, reports have described the effect of ETS on ICP as transient and the extent of ICP elevation as dependent on the duration of the procedure. There is a paucity of recent studies examining the effect of ETS on ICP in neurocritical care and specifically in SAH. The same two studies on CPT also assessed the effects of ETS following CPT. The observational study of 28 neurocritically ill patients reported no significant effects of hygiene measures, which included ETS, on ICP changes. Patients with a baseline ICP greater than 15 mmHg were at highest risk for secondary ICP insults.[62] In an RCT of traumatic brain injury (TBI) patients, administration of lidocaine was effective in blunting the rise of ICP in response to ETS without adverse effects on MAP or CPP.[66]

Recommendation
ETS is safe to perform in short intervals for patients with aSAH, when clinically indicated (strong recommendation, moderate-quality evidence). This recommendation remains unchanged from the previous 2018 AANN aSAH CPG, as few additional studies were found.

Head of Bed Elevation
Head of bed (HOB) elevation, or head-up position, is frequently used to ameliorate the effects of actual or suspected intracranial hypertension in acute aSAH patients. One systematic review with a meta-analysis reported that an HOB elevation of 30 or 45 degrees is optimal for decreasing ICP after craniotomy.[67] Similarly, a meta-analysis of TBI suggested a 30-degree HOB elevation reduces ICP with concomitant increments in CPP (no P values reported).[68]

Recommendation
Head of bed should be elevated between 30 and 45 degrees (strong recommendation, moderate-quality evidence). This recommendation remains unchanged from the previous 2018 AANN aSAH CPG, as few additional studies were found.

Positioning
Repositioning patients on a regular basis is an important nursing intervention, especially in the neurocritical care setting, to prevent clinical complications such as pneumonia and skin breakdown. There is a dearth of studies examining the effect of positioning on ICP in neurocritical care and specifically in aSAH. In general, only sharp head rotation and prone position were associated with clinically relevant ICP changes.[69] In an observational study of 28 neurocritically ill patients, there was no statistically significant change in ICP during repositions, either from lateral to supine or supine to lateral position. The greatest risk for ICP elevation was observed in patients with baseline ICP greater than 15 mmHg (P=.01).[62]

Recommendations

Patient repositioning is recommended in acute neurocritical care patients in the absence of increased ICP (strong recommendation, low-to-moderate–quality evidence). This recommendation remains unchanged from the previous 2018 AANN aSAH CPG, as only one additional study was found.

Patients with existing or suspected increased ICP should be monitored closely following repositioning for signs of increasing intracranial hypertension (weak recommendation, low-quality evidence).

Early Ambulation

Past aSAH CPGs have included early ambulation. This iteration defers early ambulation to the Clinical Practice Guideline: Mobilization of the Patient After Neurological Insult,[70] as it covers ambulation issues in detail.

Circulation

Does assessing fluid status to maintain euvolemia improve outcomes for patients with aSAH?

Triple-H therapy (hypertension, hypervolemia, hemodilution) is no longer recommended to combat vasospasm and DCI after aSAH. Recent aSAH research has shown hypervolemia to be associated with worse patient outcomes.

Four studies investigated hypervolemia as a primary predictor for clinical outcomes. The first retrospective cohort (N=223) reported hypervolemia was positively associated with DCI (day 1 *P*=.005, day 2 *P*=.004), and DCI was positively associated with worse GOS score (*P*=.008) and 6-month mortality (*P*<.001).[71] A second retrospective study (N=237) found hypervolemia was associated with prolonged ventilator use (*P*<.001), DCI (*P*<.001), and poor functional outcomes (*P*<.001).[72] The largest aSAH therapeutic hypervolemia study (N=5,400) evidenced a positive association with mortality (*P*<.001), deterioration in level of consciousness (LOC; *P*=.001), and reintubation (*P*=.002).[73] One randomized pilot (N=20) found no significant hypervolemia correlations with mRS at 6 months, neuropsychological testing, severe vasospasm, or mortality.[74] An order adherence study (N=41) reported an incidental finding that greater positive fluid intake was associated with DCI (*P*=.02).[75]

Fluid balance was investigated in five studies. A 2012 seminal study (N=356) contributed to the change in practice associated with triple-H therapy. Researchers found positive fluid balance was associated with an H&H grade of at least 3 (*P*=.03), worse ICU and hospital discharge GCS score (*P*=.01), increased ICU length of stay (LOS; *P*=.04), increased inpatient LOS (*P*=.02), and increased inpatient new stroke or death (*P*=.02).[76] Another study reported positive fluid balance was associated with an mRS score of at least 3 (N=288; *P*<.001).[77] Additionally, an RCT (N=413) found positive fluid balance was associated with increased ICU LOS (*P*<.001) and negative fluid balance was associated with DCI (*P*=.013).[78] Positive fluid balance and colloid administration were associated with worse NIHSS and mRS scores (*P*=.04, *P*=.02, respectively). Greater positive fluid balance was associated with mortality (N=6,978; days 1-3: *P*<.01, days 4-7: *P*<.01).[79] The last study (N=142) found relationships between positive fluid balance and worse H&H grade (*P*=.03), worse GCS score (*P*<.01), mechanical ventilation (*P*<.01), vasospasm (*P*=.04), increased LOS (*P*=.02), and inpatient death or new stroke (*P*=.02).[80] This study also found greater negative fluid balance was associated with worse GOS scores at 3, 6, and 12 months (*P*<.001).

Three additional volemia-related publications were found: two bundle studies and one dual-effects study. The first bundle study investigated triple-H therapy and found no decrease in DCI, no improvement in GOS score, no good recovery, and no decreased mortality (N=178).[81] The second study (N=208) incorporated euvolemia and found that bundling was associated with lower rates of DCI (*P*<.001) and lower rates of poor outcomes (*P*=.03).[82] The dual-effects study failed to show any benefits from hypervolemia or positive fluid balance (N=60).[83]

Given that hyper- and hypovolemia have been associated with increased morbidity and mortality in aSAH populations, euvolemia is the ideal target to prevent or minimize DCI, other complications, and mortality after aSAH. Euvolemia also is supported by both the AHA/ASA and Neurocritical Care Society (NCS).[1,2] The optimal method to measure and attain euvolemia is indeterminate. In the literature, direct intake and output measurement, rather than proxy physiological measurements (eg, cardiac index, venous pressure), have been used in studies with significant findings.[71-83] Owing to the sensitivity of volemic status on patient outcomes, it is incumbent upon nursing staff to accurately monitor, measure, and regulate fluid intake and balance to ensure euvolemia.

Recommendations

Support euvolemic fluid status to optimize cerebral perfusion and minimize secondary insults or complications and mortality (strong recommendation, moderate-quality evidence).

Avoid prophylactic hypervolemic therapy (strong recommendation, high-quality evidence).

Venous Thromboembolism Prophylaxis

What is the comparative effectiveness of pharmacological prophylaxis vs mechanical venous thromboembolism prophylaxis for patients with aSAH?

As a result of decreased mobility following aSAH, 4% to 12% of patients develop venous thromboembolism (VTE)[1] including deep vein thrombosis (DVT) and pulmonary embolism (PE). A large study of 15,968 aSAH patients showed overall incidences of VTE (4.4%), DVT (3.5%), and PE (1.2%).[84] VTE complications resulted in longer hospital stays (P=.018) and increased morbidity and mortality rates.[85] In one review, mean LOS was doubled for aSAH patients with VTE,[86] and in another (N=2,188), neurocritical care patients with VTE had higher mortality (P=.019) and longer ICU (P<.001) and hospital (P<.001) LOS.[87]

No studies comparing the effectiveness of pharmacological vs mechanical prophylaxis in aSAH were found. A Cochrane review of surgical and trauma patients found combining intermittent pneumatic compression (IPC) and pharmacological prophylaxis reduced the incidence of PE (P=.0005).[88] Two studies related to VTE prophylaxis in the aSAH population were found. The first (N=556) reported patients who received low-dose intravenous heparin (LDIVH) infusion were a nonsignificant 2.2 times less likely to have DVT, and implementing an LDIVH protocol was deemed noninferior to subcutaneous heparin and efficacious for DVT prophylaxis.[89] The second study (N=196) found aSAH patients to be at high risk for VTE and that they may benefit from both mechanical and chemoprophylaxis.[90]

The AHA/ASA guideline recommends chemoprophylaxis or mechanical VTE prophylaxis in aSAH patients after aneurysm securement.[1] A European guideline recommended the use of IPC in all patients before securing the aneurysm and starting low molecular weight heparin immediately after endovascular coiling (coiling) and more than 12 hours after surgical clipping (clipping).[91]

Recommendations

Low-dose intravenous heparin or subcutaneous heparin are appropriate VTE chemoprophylaxis in aSAH patients (strong recommendation, low-quality evidence).

Initiating both mechanical and pharmacological VTE prophylaxis may be considered in aSAH patients at high risk for VTE (strong recommendation, moderate-quality evidence).

Chemoprophylaxis or mechanical VTE prophylaxis is recommended in aSAH patients after securing the aneurysm (strong recommendation, moderate-quality evidence).

Seizure Prophylaxis

Does seizure prophylaxis improve outcomes for patients with aSAH?

Convulsive and nonconvulsive seizures may occur at hemorrhage onset or anytime during acute hospitalization.[1,92] Reported rates range from 6.4% to 26%.[1,92,93] However, improved electroencephalogram (EEG) technology suggests the incidence of seizures is likely between 7.8% and 15.2%.[1,94-96] These acute statistics were iterated in a longitudinal (N=875) study where a 12% incidence of epilepsy was noted at 5 years.[97]

Risk factors associated with the development of seizures in aSAH patients include surgical management of the aneurysm, higher clinical grade, middle (MCA) and anterior (ACA) cerebral artery aneurysms, and hydrocephalus.[1,31,93,98-102] One prospective cohort study of 288 aSAH patients found significantly higher mean hemorrhage volume in patients with seizure (P=.01).[103] Age as a seizure risk factor yielded conflicting evidence. One retrospective study of 984 aSAH patients reported an overall seizure incidence of 9.5%, with patients younger than 51 years at significantly higher risk (P<.001)[98] Conversely, data on 1,500 patients from the SAH Outcomes Project demonstrated a higher seizure incidence in older patients (64 vs 53 years, P=.001).[104] Lastly, one retrospective study of 69 patients found age, severity of hemorrhage, and hydrocephalus were not significantly associated with seizure.[102]

Uncontrolled seizures may increase the risk of rupture in patients with unsecured aneurysms, causing devastating downstream physiologic effects.[105] Multiple studies found that seizures are associated with higher morbidity and mortality as well as increased ICU and acute hospital LOS.[31,93,101,106] However, one study found that seizures may not be predictive of morbidity and mortality.[107] The optimal type and duration of seizure monitoring in the aSAH population is unestablished.[105] In the absence of clinical seizures, a fluctuating neurological exam should raise suspicion for nonconvulsant seizure, and continuous EEG monitoring for up to 48 hours was found to be reasonable and reliable in detecting subclinical seizures.[108]

Historically, acute aSAH patients received seizure prophylaxis upon admission and continued on antiepileptic drugs (AED) or antiseizure medication (ASM) therapy for months or years. Evidence is lacking regarding the optimal choice of AED for seizure treatment or prophylaxis.[96,109,110] Phenytoin and levetiracetam are the most commonly used.[109-111] Levetiracetam is associated with a lower incidence of adverse effects, and phenytoin, although similar in efficacy, is less used due to reported adverse cognitive effects and poor outcomes.[96,98] Both phenytoin and levetiracetam were well tolerated when limited to the immediate post-hemorrhage period.[112] The use of perampanel was associated with a reduction in the

incidence of DCI (N=121; *P* not reported),[113] and valproic acid was associated with a reduced risk of acute respiratory failure in patients with aSAH (N=16,228; *P*=.014).[114]

While there is no clear consensus regarding the initiation of prophylactic seizure treatment, recommendations increasingly advise against prophylaxis.[115] These changes in recommendations are driven by an increasing number of studies evidencing no significant difference in seizure occurrence between patients treated prophylactically with AEDs vs those who are not.[31,94,95,100,116-118] A study of 259 aSAH patients found prophylactic AED medications had no effect on the occurrence of delayed seizures, DCI, or poor functional outcomes.[119] One meta-analysis found that AED treatment duration beyond 3 to 7 days led to poor clinical outcomes (*P*=.045).[94] In aSAH patients who were awake and following commands post aneurysm securement, discontinuation of AEDs was reported to be safe, feasible, and associated with lower mortality (*P*=.0028) and discharge to home (*P*=.002).[117] Discontinuing AED prophylaxis immediately after aneurysm coiling was not associated with increased risk of seizures.[112,117] A 2017 review of 37 seizure studies determined that seizure prophylaxis was not warranted given the adverse events associated with AEDs.[96]

Recommendations

Seizure prophylaxis may be considered in aSAH patients prior to aneurysm treatment and for those at high risk for the development of seizures. Duration of antiseizure medications should be routinely evaluated to avert long-term adverse effects (weak recommendation, moderate-quality evidence).

Routine neurologic checks should include an assessment for nonconvulsive and other seizure activity (weak recommendation, low-quality evidence).

Endovascular Therapy

What are the pre- and postoperative nursing care considerations regarding endovascular therapy for patients with aSAH?

Endovascular therapy (EVT) for aSAH employs minimally invasive techniques including embolization, coiling, stenting, and flow diversion and is associated with lower risks of cerebral ischemia, vasospasm, infection, pneumonia, and mortality than open craniotomy.[25,120-123] One meta-analysis (N=2,780) found coiling reduced poor outcomes (mRS>2) at 1 year (*P*<.00011) and 3 to 5 years (*P*=.02). Morbidity and mortality were significantly lower following coiling despite concurrent aneurysmal occlusion rates being less than 100% at 1 year (*P*<.00001).[12]

Postprocedure care should target prevention and minimization of secondary complications due to rebleeding,[49] labile systolic blood pressure (SBP),[124,125] higher or labile heart rate,[124] DCI,[49,126] and symptomatic cerebral vasospasm,[125-127] which are associated with unfavorable outcomes and extended LOS. It is important for the bedside nurse to be cognizant of these risk factors and postprocedural care standards in efforts to mitigate complications.

Neurological Assessment

There were no studies that evaluated specific neurological assessments after EVT. Peer-reviewed publications reported that most secondary complications, such as DCI, vasospasm, and rebleeding,[11,122,123,128,129] could be identified early on through routine and frequent neurological assessment.[11,130-132] Signs of DCI and vasospasm include new or worsening neurological focal deficits and decreased mental status.[1,11,25,132,133] Bedside nurses should use and document standardized neurologic exams and severity scales for early identification of neurologic deterioration and to implement early therapeutic interventions.[11,134] The assessments are typically every 15 minutes for 1 hour, every 30 minutes for 1 hour, then every hour for 4 hours until otherwise ordered.[1,134] ICU frequency is typically every 1 to 2 hours. During any postprocedural nursing handoff, a neurological exam should be performed to detect any occurrence of neurological decline.[132,134]

Vital Signs

Research is lacking regarding the optimal interval for frequency of postprocedural vital sign checks, and current assessment parameters are derived from consensus based on historical practice of neuroscience and other specialties. During the immediate recovery phase, vital signs are traditionally monitored every 15 minutes for 1 hour, then every 30 minutes for 1 hour,[134] and are often ordered with the same frequency as neurologic assessment. Similar to neurologic assessment, ICU vital sign monitoring frequency is typically every 1 to 2 hours.

Nurses should monitor heart rate (HR), blood pressure (BP), respiratory rate, pulse oximetry, and temperature[11,132,134] and know the target clinical parameters so as to avoid hemodynamic fluctuations, which can result in complications and worse outcomes.[1,2,11,128] Post-EVT, it is reasonable to target SBP at less than 160 to 180 mmHg to optimize CPP (70-95 mmHg) to prevent and treat vasospasm and DCI.[1,2]

Postprocedural Care

Patients typically receive antiplatelets after stenting and flow diversion due to a higher thrombosis risk after coiling and embolization.[1] Anesthesia or sedation may affect the neurologic exam. Therefore, short half-life sedation is preferred to titrate for neurologic exams except in the setting of hemodynamic instability or status epilepticus. Nurses should monitor for complications after EVT including access-site hematoma, limb ischemia, retroperi-

toneal hemorrhage, vascular complications (eg, dissection, perforation, vasospasm), and stroke.[131,134] Post-EVT assessment should include site assessment; distal circulation checks (perfusion and pulses); pain, bleeding, and neurologic exams; and hemodynamic monitoring.[134]

Recommendation

Standard post-EVT protocols or order sets should include frequent neurologic assessment, vital sign monitoring, and postprocedural assessment including site check, distal pulse, and distal circulation checks (strong recommendation, very low-quality evidence).

Enteral Nutrition

For aSAH requiring enteral nutrition, which tube feeding strategies and protocols are most effective in ensuring optimal nutritional intake?

The most effective strategy for ensuring optimal nutritional intake in aSAH is an understudied topic. The American Society for Parenteral and Enteral Nutrition (ASPEN) Guidelines[135] and the combined guidelines of the Society of Critical Care Medicine (SCCM) and ASPEN[136] recommend enteral nutrition (EN) strategies for critically ill patients. These recommendations include targeting 12 to 25 kcal/kg on days 1 through 10 of ICU stay[135]; identifying patients with high nutritional risk[136]; initiating EN within 24 to 48 hours of ICU admission[136]; avoiding unnecessary interruptions in EN delivery[136]; if using gastric residual volumes (GRV), only holding EN for GRV less than 500 mL except in the setting of gastrointestinal (GI) intolerance[136]; using EN protocols, including volume-based feeding protocols, to ensure optimal calorie delivery[136]; and continuing EN in the setting of diarrhea until the cause can be identified.[136] It is important to note: these guidelines make no recommendations for the neuroscience population, nor aSAH specifically.

There is a paucity of evidence regarding strategies to improve the delivery of EN specific to the aSAH population; studies generally focused on mixed neurocritical care populations with few aSAH participants. Mechanically ventilated aSAH patients are at higher risk for EN interruptions and receiving less than 60% of nutritional goals, with procedures cited as the most common cause of interuption.[137] Of the three studies found, one was a meta-analysis of 23 articles including 1,816 ischemic and hemorrhagic stroke patients (aSAH number not reported), which found probiotics combined with EN improved nutritional status (P<.05) and reduced hospital LOS (P<.05), time spent on bedrest (P<.05), and GI symptoms such as esophageal reflux, bloating, constipation, diarrhea, gastric retention, and GI bleeding (P<.05).[138] An RCT (N=46) of neurocritical care patients found the enteral formula plus prebiotics group had fewer EN complications than the enteral formula without prebiotics group (13% vs 56.5 %, respectively; P=.002).[139] A retrospective study of 1,495 neurocritical care patients (aSAH number not reported) from 353 ICUs found EN adequacy to be better when delivered through gastric feeding tubes vs small bowel feeding tubes in unadjusted analysis (P=.001) but not in bivariate analysis (P=.428). Additionally, in bivariate analysis, interruptions in EN delivery were five times more frequent in the gastric feeding group compared to the small bowel feeding group (P=.015).[140] Ultimately, the literature did not yield a consensus on the optimal nutritional constitution nor optimal feeding methodology and regimen.

Recommendations

Nurses should assess for signs of GI symptoms and intolerance including esophageal reflux, bloating, constipation, diarrhea, gastric retention, and GI bleeding (good practice statement).

The addition of probiotics or prebiotics to EN may be considered to decrease the risk of GI symptoms (weak recommendation, low-quality evidence).

To achieve target nutrient and calorie goals, nurses should avoid unnecessary interruptions in EN delivery (good practice statement).

Small bowel feeding tubes may be preferred over gastric feeding tubes to decrease interruptions in EN delivery caused by GI symptoms (weak recommendation, low-quality evidence).

Pain Management

Which pharmacological and alternative pain management therapies are safe and effective for patients with aSAH (eg, neck pain, headache, postprocedural pain)?

Pain Characterization

Headache (H/A) was the most specified pain site with a reported incidence range of 50% to 90%.[141-146] Other reported pain sites were the back, neck, limbs, and eyes.[146] Additional symptoms included nausea, vomiting, seizure, and meningeal irritation symptoms (ie, nuchal rigidity, neck pain, photophobia, painful neck flexion).[141,143,147,148]

Duration of pain post aSAH varied. Two retrospective studies (N=77; N=106)[144,149] reported pain duration in relation to illness severity and analgesic use, and two simply studied duration. Headache improved upon discharge in 84% of aSAH patients (N=217; P<.001).[150] A second study (N=864) found 63.1% of aSAH patients had H/A for more than 3 months, 50% had H/A at 1 year, and 28% had H/A at 10 years, and the median time for H/A resolution was 149 months (12.4 years).[142] Findings indicate while some patients have H/A resolution by discharge,[149] some

patients may have persistent H/A for an extended period of time.[142,145,149-151]

Pain following aSAH was reported to be the most severe at the time of onset and hospitalization,[141,143,144,149] and severity was associated with illness severity.[144,152] One retrospective study of 217 patients found that 99% of patients whose pain improved on discharge also reported less pain after 1 year.[150] In general, the mean pain severity declined throughout hospitalization and over time.[142,149,150]

Pharmacotherapy

Six studies investigated the effects of pharmacotherapy on aSAH pain management, with no studies reporting absolute optimal medication or dosage. The most frequently reported medications used were acetaminophen or opioids.[144-146,152-154] One study of healthcare providers (N=516) indicated acetaminophen (90%), opioids (66%), corticosteroids (28%), and antiseizure medications (28%) were the most prescribed medications to treat aSAH headache, with opioids perceived as the most effective medications followed by corticosteroids.[153] The most commonly prescribed opioid was oxycodone. Other opioids used included tramadol, hydromorphone, fentanyl, morphine, and hydrocodone and acetaminophen.[144-146,152] In three studies, higher opioid use was associated with higher pain severity (day 1: P=.009, P=.01, P<.01).[151,152,154] These findings were not consistent throughout the literature where reports of highest pain scores were on admission but not in accord with the timing of highest analgesic use, possibly owing to minimization of opioids during workup and pretreatment, confounding assessment concerns.

Reported opioid alternatives included dexamethasone[144,146,153]; gabapentin[146,153]; pregabalin[153]; magnesium[152,153]; ketorolac[152]; and acetaminophen, butalbital, and caffeine.[144,152,154] None of these significantly decreased patient-reported pain.[152] Guidelines recommended the first-line treatment of aSAH-related H/A be nonopioid analgesics, such as acetaminophen, with opioids added as needed.[148,155]

Nonpharmacological Therapies

High-quality data supporting the use of specific nonpharmacological interventions, especially for critically ill people, are limited, and outcomes data are not available. Only one study[156] exclusively researched nonpharmacological interventions on pain control in the aSAH population. Other studies denoted nonpharmacological interventions, but not as primary interventions. The Korean Society of Critical Care Medicine guidelines recommended positioning and eliminating stimuli as first-line interventions before proceeding with pharmacological treatment.[157] One study suggested that a combination of nonpharmacological and pharmacological therapies may optimize the quality of pain management.[158]

In general and critical care research, nonpharmacological pain interventions included CSF diversion for hydrocephalus,[146,153] nerve blocks,[153] acupuncture,[153,159] herbal medicine,[153] osteopathic treatment,[156] massage,[153,155,157-160] Reiki,[153] music,[153,155,158-162] cold therapy,[157,158] relaxation,[157-159] and cognitive behavioral (distraction) therapies.[160] Identifying and then removing and mitigating the primary negative stimulus causing pain is essential, and nurses should consider physiological dysfunction, body alignment, and environmental (eg, noise, light, smell, pressure, temperature) causes.

Nursing Considerations

Given the lack of studies on this topic, the writing group derived nursing considerations for aSAH pain management based on best practices from general pain research, other professional guidelines, and peer-reviewed publications.

A reliable assessment is essential to identify early signs of neurologic deterioration; therefore, it is imperative to consider that analgesics may mask true neurologic status. Short-acting medications with rapid onset and easy dose titration are better choices to allow quicker awakening for accurate neurologic assessment.[157,158] The SCCM guidelines suggest sedation interruption can be useful in providing proper assessment.[155] Depressive side effects of opioids should be monitored and considered, as they coincide with the neurologic exam.[148,157]

Pain assessment, a quality indicator, is also central to nursing care, and the results of established pain assessment tools for both verbal and nonverbal patients must be documented. In nonverbal situations, pain scales such as the Critical Care Pain Observation Tool or Behavioral Pain Scale relay pain level through assessment of facial expression, body movements, muscle tension, and vital signs and can guide pain management.[155,157,158] Owing to the critical and often tenuous nature of aSAH, nursing must consider both assessment and treatment effects of pain management.

Recommendations

A standard pain scale tool should be used to assess pain in all patients, both with and without altered mental status including with and without verbal impairment (good practice statement).

Acetaminophen is the first-line choice for mild pain. Opioids are preferred agents for moderate to severe pain (weak recommendation, low-quality evidence).

Opioid selection should consider short-acting agents, effectiveness, and side effects that could mask the neurological exam findings (good practice statement).

Nonpharmacological treatment, such as nerve blocks, acupuncture, herbal medicine, massage therapy, Reiki therapy, music therapy, and osteopathic treatment, may

be considered as alternative treatments (weak recommendation, low-quality evidence).

Thermodynamics

Do normothermia protocols improve outcomes for patients with aSAH, and which cooling devices are most effective?

Fever refractory to pharmacotherapy is common within 48 hours of having an aSAH. Studies have shown fever is associated with poor outcomes including increased DCI, increased cerebral metabolic distress, worse functional outcomes, and mortality.[30,163-165] Though the effects of therapeutic hypothermia on cerebral salvage status post cardiac arrest are well established, the effects on status post aSAH are not comparably researched. The role of body temperature on aSAH patient outcomes is not clearly defined. Accordingly, the effects of maintaining normothermia on patient outcomes remain inconclusive.[1]

The most effective protocol and cooling device for maintaining normothermia in aSAH patients is unestablished,[1] and published research during the search decade is scant. One study (N=122) found VTE events (eg, PE [*P*=.039], mortality [*P*<.001]) were more likely when endovascular cooling device catheters were used for temperature management compared to central venous lines (*P*<.001).[166] Another study (N=32) found there was a trend in taking longer to achieve normothermia when using an esophageal temperature modulating device (*P*=.07).[167] A sensitivity analysis of a normothermia in severe cerebrovascular disease RCT (N=47) found that lower cooling bath temperatures were positively associated with favorable outcomes (mRS 0-2) at 180 days (*P*=.038).[168] The AHA/ASA guideline reported the effectiveness of therapeutic temperature management was limited but recommended evidence-based protocols and order sets to standardize care.[1] Additionally, the Canadian Stroke Best Practice Recommendations[169] endorsed monitoring temperature and treating fever per protocols.

Recommendations

Measures to promote normothermia after aSAH are recommended (weak recommendation, low-quality evidence). This recommendation remains unchanged from the previous 2018 AANN aSAH CPG, as few additional studies were found.

Implementing protocols and order sets are recommended (good practice statement).

Summary

This guideline is an update of the 2018 AANN aSAH CPG. Though there was broad topical coverage in aSAH research literature, there remain numerous areas lacking sufficient evidence regarding care. Research opportunities are documented in each respective PICO section and can serve as starting points for both seasoned and novice researchers. Publication of standard nursing quality improvement initiatives should not be overlooked in their value to the broader neuroscience nursing application.

References

1. Hoh BL, Ko NU, Amin-Hanjani S, et al. 2023 guideline for the management of patients with aneurysmal subarachnoid hemorrhage: a guideline from the American Heart Association/American Stroke Association. *Stroke*. 2023;54(7):e314-e370. doi:10.1161/STR.0000000000000436.
2. Treggiari MM, Rabinstein AA, Busl KM, et al. Guidelines for the neurocritical care management of aneurysmal subarachnoid hemorrhage. *Neurocrit Care*. 2023;39(1):1-28. doi:10.1007/s12028-023-01713-5.
3. Censullo JL, Tran DS, Starkweather A. Methods for developing neuroscience nursing clinical practice guidelines. *J Neurosci Nurs*. 2024;56(6):236-238. doi:10.1097/JNN.0000000000000795.
4. Global health estimates: life expectancy and leading causes of death and disability. Geneva, Switzerland: World Health Organization; 2024. Licence: CC BY-NC-SA 3.0 IGO.
5. Johnson CO, Nguyen M, Roth GA, et al. Global, regional, and national burden of stroke, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet Neurol*. 2019;18(5):439-458. doi:10.1016/S1474-4422(19)30034-1.
6. Renedo D, Acosta JN, Leasure AC, et al. Burden of ischemic and hemorrhagic stroke across the US from 1990 to 2019. *JAMA Neurol*. 2024;81(4):394-404. doi:10.1001/jamaneurol.2024.0190.
7. Benjamin EJ, Blaha MJ, Chiuve SE, et al. Heart disease and stroke statistics—2017 update: a report from the American Heart Association [published corrections appear in *Circulation*. 2017;135(10):e646. doi:10.1161/CIR.0000000000000491 and *Circulation*. 2017;136(10):e196. doi:10.1161/CIR.0000000000000530]. *Circulation*. 2017;135(10):e146-e603. doi:10.1161/CIR.0000000000000485.
8. Tsao CW, Aday AW, Almarzooq ZI, et al. Heart disease and stroke statistics—2023 update: a report from the American Heart Association [published corrections appear in *Circulation*. 2023;147(8):e622. doi:10.1161/CIR.0000000000001137 and *Circulation*. 2023;148(4):e4. doi:10.1161/CIR.0000000000001167]. *Circulation*. 2023;147(8):e93-e621. doi:10.1161/CIR.0000000000001123.
9. Wettervik TS, Lewén A, Enblad P. Fine tuning of neurointensive care in aneurysmal subarachnoid hemorrhage: from one-size-fits-all towards individualized care. *World Neurosurg X*. 2023;18:100160. doi:10.1016/j.wnsx.2023.100160.
10. Boerboom W, Heijenbrok-Kal MH, Khajeh L, van Kooten F, Ribbers GM. Long-term functioning of patients with aneurysmal subarachnoid hemorrhage: a 4-yr follow-up study. *Am J Phys Med Rehabil*. 2016;95(2):112-120. doi:10.1097/PHM.0000000000000353.
11. Francoeur CL, Mayer SA. Management of delayed cerebral ischemia after subarachnoid hemorrhage. *Crit Care.* 2016; 20(1):277. doi:10.1186/s13054-016-1447-6.
12. Crago EA, Price TJ, Bender CM, Ren D, Poloyac SM, Sherwood PR. Impaired work productivity after aneurysmal subarachnoid hemorrhage. *J Neurosci Nurs*. 2016;48(5):260-268. doi:10.1097/JNN.0000000000000209.
13. Boling B, Groves TR. Management of subarachnoid hemorrhage. *Crit Care Nurse*. 2019;39(5):58-67. doi:10.4037/ccn2019882.
14. Moyer M, Young B, Wilensky EM, et al. Implementation of an early mobility pathway in neurointensive care unit patients with external ventricular devices. *J Neurosci Nurs*. 2017;49(2):102-107. doi:10.1097/JNN.0000000000000258.
15. Al-Mufti F, Mayer SA, Kaur G, et al. Neurocritical care management of poor-grade subarachnoid hemorrhage: unjustified nihilism to reasonable optimism. *Neuroradiol J*. 2021;34(6):542-551. doi:10.1177/19714009211024633.
16. McLaughlin DC, Margretta MM, Freeman WD. Aneurysmal subarachnoid hemorrhage mortality after implementation of nocturnist advanced practice provider coverage. *J Neurosci Nurs*. 2018;50(2):102-104. doi:10.1097/JNN.0000000000000352.
17. Wuchner SS, Bakas T, Adams G, Buelow J, Cohn J. Nursing interventions and assessments for aneurysmal subarachnoid hemorrhage patients: a mixed methods study involving practicing nurses. *J Neurosci Nurs*. 2012;44(4):177-187. doi:10.1097/JNN.0b013e318252763f.
18. Lantigua H, Ortega-Gutierrez S, Schmidt JM, et al. Subarachnoid hemorrhage: who dies, and why? *Crit Care*. 2015;19(1):309. doi:10.1186/s13054-015-1036-0.
19. Roquer J, Cuadrado-Godia E, Guimaraens L, et al. Short- and long-term outcome of patients with aneurysmal subarachnoid hemorrhage. *Neurology*. 2020;95(13):e1819-e1829. doi:10.1212/WNL.0000000000010618.
20. Helbok R, Kurtz P, Vibbert M, et al. Early neurological deterioration after subarachnoid haemorrhage: risk factors and impact on outcome. *J Neurol Neurosurg Psychiatry*. 2013;84(3):266-270. doi:10.1136/jnnp-2012-302804.
21. Autio AH, Paavola J, Tervonen J, et al. Clinical condition of 120 patients alive at 3 years after poor-grade aneurysmal subarachnoid hemorrhage. *Acta Neurochir (Wien)*. 2021;163(4):1153-1166. doi:10.1007/s00701-021-04725-2.
22. Chalouhi N, Tjoumakaris S, Thakkar V, et al. Endovascular management of cerebral vasospasm following aneurysm rupture: outcomes and predictors in 116 patients. *Clin Neurol Neurosurg*. 2014;118:26-31. doi:10.1016/j.clineuro.2013.12.012.

23. Wang L, Zhang Q, Zhang G, et al. Risk factors and predictive models of poor prognosis and delayed cerebral ischemia in aneurysmal subarachnoid hemorrhage complicated with hydrocephalus. *Front Neurol*. 2022;13:1014501. doi:10.3389/fneur.2022.1014501.

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

25. Gross BA, Lai PMR, Frerichs KU, Du R. Treatment modality and vasospasm after aneurysmal subarachnoid hemorrhage. *World Neurosurg*. 2014;82(6):e725-e730. doi:10.1016/j.wneu.2013.08.017.

26. Alfotih GT, Li F, Xu X, Zhang S. Risk factors for re-bleeding of aneurysmal subarachnoid hemorrhage: meta-analysis of observational studies. *Neurol Neurochir Pol*. 2014;48(5):346-355. doi:10.1016/j.pjnns.2014.08.002.

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

28. Rass V, Ianosi BA, Lindlbauer M, et al. Factors associated with prolonged mechanical ventilation in patients with subarachnoid hemorrhage—the RAISE score. *Crit Care Med*. 2022;50(1):103-113. doi:10.1097/CCM.0000000000005189.

29. Wojak JF, Ditz C, Abusamha A, et al. The impact of extubation failure in patients with good-grade subarachnoid hemorrhage. *World Neurosurg*. 2018;117:e335-e340. doi:10.1016/j.wneu.2018.06.027.

30. Suehiro E, Sadahiro H, Goto H, et al. Importance of early postoperative body temperature management for treatment of subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis*. 2016;25(6):1482-1488. doi:10.1016/j.jstrokecerebrovasdis.2016.01.053.

31. Smith AM, Clark PR, Winter KA, et al. The effect of prophylactic antiepileptic medications in aneurysmal subarachnoid hemorrhage patients: a retrospective review. *Clin Neurol Neurosurg*. 2021;205:106633. doi:10.1016/j.clineuro.2021.106633.

32. Wang Z, Zhou J, Liang F, et al. Prognostic models for neurological functional outcomes in aneurysmal subarachnoid hemorrhage patients with intracranial hematoma. *Clin Neurol Neurosurg*. 2020;191:105691. doi:10.1016/j.clineuro.2020.105691.

33. Fang Y, Lu J, Zheng J, et al. Comparison of aneurysmal subarachnoid hemorrhage grading scores in patients with aneurysm clipping and coiling. *Sci Rep*. 2020;10(1):9199. doi:10.1038/s41598-020-66160-0.

34. Magee CA, Bastin MLT, Graves K, et al. Fever burden in patients with subarachnoid hemorrhage and the increased use of antibiotics. *J Stroke Cerebrovasc Dis*. 2019;28(11):104313. doi:10.1016/j.jstrokecerebrovasdis.2019.104313.

35. Zhang LM, Li R, Zhao XC, Wang ML. Decreased tidal volume with increased height, but not colloid transfusion, is associated with worse outcomes and postoperative pneumonia after coil embolization of aneurysmal subarachnoid hemorrhage: a retrospective study. *Shock*. 2018;50(4):421-426. doi:10.1097/SHK.0000000000001095.

36. Duan G, Yang P, Li Q, et al. Prognosis predicting score for endovascular treatment of aneurysmal subarachnoid hemorrhage: a risk modeling study for individual elderly patients. *Medicine (Baltimore)*. 2016;95(7):e2686. doi:10.1097/MD.0000000000002686.

37. Lindner A, Brunelli L, Rass V, et al. Long-term clinical trajectory of patients with subarachnoid hemorrhage: linking acute care and neurorehabilitation. *Neurocrit Care*. 2023;38(1):138-148. doi:10.1007/s12028-022-01572-6.

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

39. Wang X, Han C, Xing D, Wang C, Ding X. Early management of poor-grade aneurysmal subarachnoid hemorrhage: a prognostic analysis of 104 patients. *Clin Neurol Neurosurg*. 2019;179:4-8. doi:10.1016/j.clineuro.2019.02.003.

40. Zheng K, Zhao B, Tan XX, et al. Comparison of aggressive surgical treatment and palliative treatment in elderly patients with poor-grade intracranial aneurysmal subarachnoid hemorrhage. *Biomed Res Int*. 2018;2018:5818937. doi:10.1155/2018/5818937.

41. de Souza ML, Vieira AC, Andrade G, Quinino S, de Fátima Leal Griz M, Azevedo-Filho HR. Fisher grading scale associated with language disorders in patients with anterior circulation aneurysmal subarachnoid hemorrhage. *World Neurosurg*. 2015;84(2):308-313. doi:10.1016/j.wneu.2015.03.017.

42. Zhao B, Tan X, Zhao Y, et al. Variation in patient characteristics and outcomes between early and delayed surgery in poor-grade aneurysmal subarachnoid hemorrhage. *Neurosurgery*. 2016;78(2):224-231. doi:10.1227/NEU.0000000000001038.

43. Kim D, Pyen J, Whang K, et al. Factors associated with rebleeding after coil embolization in patients with aneurysmal subarachnoid hemorrhage. *J Cerebrovasc Endovasc Neurosurg*. 2022;24(1):36-43. doi:10.7461/jcen.2021.E2021.05.006.

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

45. Hammer A, Erguth F, Hohenhaus M, et al. Neurocritical care complications and interventions influence the outcome in aneurysmal subarachnoid hemorrhage. *BMC Neurol*. 2021;21(1):27. doi:10.1186/s12883-021-02054-6.

46. Ota N, Noda K, Chida D, et al. Emergent subarachnoid clot removal with aneurysm repair for subarachnoid hemorrhage might improves clinical outcome. *World Neurosurg*. 2022;167:e100-e109. doi:10.1016/j.wneu.2022.07.151.

47. Ota N, Noda K, Hatano Y, et al. Preoperative predictors and prognosticators after microsurgical clipping of poor-grade subarachnoid hemorrhage: a retrospective study. *World Neurosurg*. 2019;125:e582-e592. doi:10.1016/j.wneu.2019.01.135.

48. Wong GK, Lam SW, Ngai K, et al. Cognitive domain deficits in patients with aneurysmal subarachnoid haemorrhage at 1 year. *J Neurol Neurosurg Psychiatry*. 2013;84(9):1054-1058. doi:10.1136/jnnp-2012-304517.

49. Galea JP, Dulhanty L, Patel HC; UK and Ireland Subarachnoid Hemorrhage Database Collaborators. Predictors of outcome in aneurysmal subarachnoid hemorrhage patients: observations from a multicenter data set. *Stroke*. 2017;48(11):2958-2963. doi:10.1161/STROKEAHA.117.017777.

50. Ferrell BR, Twaddle ML, Melnick A, Meier DE. National Consensus Project Clinical Practice Guidelines for Quality Palliative Care Guidelines, 4th Edition. *J Palliat Med*. 2018;21(12):1684-1689. doi:10.1089/jpm.2018.0431.

51. Creutzfeldt CJ, Holloway RG, Curtis JR. Palliative care: a core competency for stroke neurologists. *Stroke*. 2015;46(9):2714-2719. doi:10.1161/STROKEAHA.115.008224.

52. Frontera JA, Curtis JR, Nelson JE, et al. 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*. 2015;43(9):1964-1977. doi:10.1097/CCM.0000000000001131.

53. Braun LT, Grady KL, Kutner JS, et al. Palliative care and cardiovascular disease and stroke: a policy statement from the American Heart Association/American Stroke Association. *Circulation*. 2016;134(11):e198-e225. doi:10.1161/CIR.0000000000000438.

54. Creutzfeldt CJ, Hanna MG, Cheever CS, et al. Palliative care needs assessment in the neuro-ICU: effect on family. *Neurocrit Care*. 2017;27(2):163-172. doi:10.1007/s12028-017-0426-3.

55. Markovitz N, Morgenstern LB, Shafie-Khorassani F, et al. Family perceptions of quality of end-of-life care in stroke. *Palliat Med Rep*. 2020;1(1):129-134. doi:10.1089/pmr.2020.0041.

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

57. Tran D, Supin E, Young A, Ricke D, Censullo J. *Evidence-Based Clinical Review: Intracranial Monitoring.* American Association of Neuroscience Nurses. 2023. Accessed. https://aann.org/uploads/Publications/CPGs/AANN23_ICP_EBCR_FINAL.pdf.

58. Censullo J. External ventricular drain (EVD) monitoring [quick guide]. American Association of Neuroscience Nurses. 2023. Accessed October 24, 2024. https://aann.org/uploads/Neuroscience_Resources/EVD.pdf.

59. Censullo J. Intraparenchymal monitoring (IPM) [quick guide]. Association of Neuroscience Nurses. (2023). Accessed October 24, 2024. https://aann.org/uploads/Neuroscience_Resources/IPM.pdf.

60. Censullo J. Brain tissue oxygenation ($PbtO_2$) monitoring [quick guide]. Association of Neuroscience Nurses. 2023. Accessed October 24, 2024. https://aann.org/uploads/Neuroscience_Resources/Pbt02.pdf.

61. Olson DM, Parcon C, Santos A, Santos G, Delabar R, Stutzman SE. A novel approach to explore how nursing care affects intracranial pressure. *Am J Crit Care*. 2017;26(2):136-139. doi:10.4037/ajcc2017410 0.

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

63. Szabo CM, Grap MJ, Munro CL, Starkweather A, Merchant RE. The effect of oral care on intracranial pressure in critically ill adults. *J Neurosci Nurs*. 2014;46(6):321-329. doi:10.1097/JNN.0000000000000092.

64. Wen J, Chen J, Chang J, Wei J. Pulmonary complications and respiratory management in neurocritical care: a narrative review. *Chin Med J (Engl)*. 2022;135(7):779-789. doi:10.1097/CM9.0000000000001930.

65. Tomar GS, Singh GP, Bithal P, Upadhyay AD, Chaturvedi A. Comparison of effects of manual and mechanical airway clearance techniques on intracranial pressure in patients with severe traumatic brain injury on a ventilator: randomized, crossover trial. *Phys Ther*. 2019;99(4):388-395. doi:10.1093/ptj/pzy141.

66. Singh S, Chouhan RS, Bindra A, Radhakrishna N. Comparison of effect of dexmedetomidine and lidocaine on intracranial and systemic hemodynamic response to chest physiotherapy and tracheal suctioning in patients with severe traumatic brain injury. *J Anesth*. 2018;32(4):518-523. doi:10.1007/s00540-018-2505-9.

67. Jiang Y, Ye Zp, You C, et al. Systematic review of decreased intracranial pressure with optimal head elevation in postcraniotomy patients: a meta-analysis. *J Adv Nurs*. 2015;71(10):2237-2246. doi:10.1111/jan.12679.

68. Alarcon JD, Rubiano AM, Okonkwo DO, et al. Elevation of the head during intensive care management in people with severe traumatic brain injury. *Cochrane Database Syst Rev*. 2017;12(12):CD009986. doi:10.1002/14651858.CD009986.pub2.

69. Mitchell PH, Kirkness C, Blissitt PA. Chapter 5 cerebral perfusion pressure and intracranial pressure in traumatic brain injury. *Annu Rev Nurs Res*. 2015;33:111-183. doi:10.1891/0739-6686.33.111.

70. Young B, Schmidt M, Moyer M. *Clinical Practice Guideline: Mobilization of the Patient after Neurological Insult.* American Association of Neuroscience Nurses. 2021. Accessed October 24, 2024. https://aann.org/uploads/Publications/CPGs/AANN21_Mobilization_CPG_v6.pdf.

71. Vergouw LJM, Egal M, Bergmans B, et al. High early fluid input after aneurysmal subarachnoid hemorrhage: combined report of association with delayed cerebral ischemia and feasibility of cardiac output-guided fluid restriction. *J Intensive Care Med*. 2020;35(2):161-169. doi:10.1177/0885066617732747.

72. Rass V, Gaasch M, Kofler M, et al. Fluid intake but not fluid balance is associated with poor outcome in nontraumatic subarachnoid hemorrhage patients. *Crit Care Med*. 2019;47(7):e555-e562. doi:10.1097/CCM.0000000000003775.

73. Kuwabara K, Fushimi K, Matsuda S, Ishikawa KB, Horiguchi H, Fujimori K. Association of early post-procedure hemodynamic management with the outcomes of subarachnoid hemorrhage patients. *J Neurol*. 2013;260(3):820-831. doi:10.1007/s00415-012-6710-4.

74. Togashi K, Joffe AM, Sekhar L, et al. Randomized pilot trial of intensive management of blood pressure or volume expansion in subarachnoid hemorrhage (IMPROVES). *Neurosurgery*. 2015;76(2):125-135. doi:10.1227/NEU.0000000000000592.

75. Szmuda T, Waszak PM, Rydz C, et al. The challenges of hypervolemic therapy in patients after subarachnoid haemorrhage. *Neurol Neurochir Pol*. 2014;48(5):328-336. doi:10.1016/j.pjnns.2014.09.001.

76. Martini RP, Deem S, Brown M, et al. The association between fluid balance and outcomes after subarachnoid hemorrhage. *Neurocrit Care*. 2012;17(2):191-198. doi:10.1007/s12028-011-9573-0.

77. Kissoon NR, Mandrekar JN, Fugate JE, Lanzino G, Wijdicks EF, Rabinstein AA. Positive fluid balance is associated with poor outcomes in subarachnoid hemorrhage. *J Stroke Cerebrovasc Dis*. 2015;24(10):2245-2251. doi:10.1016/j.jstrokecerebrovasdis.2015.05.027.

78. Ibrahim GM, Macdonald RL. The effects of fluid balance and colloid administration on outcomes in patients with aneurysmal subarachnoid hemorrhage: a propensity score-matched analysis. *Neurocrit Care*. 2013;19(2):140-149. doi:10.1007/s12028-013-9860-z.

79. Wu CL, Pai KC, Wong LT, Wang MS, Chao WC. Impact of early fluid balance on long-term mortality in critically ill surgical patients: a retrospective cohort study in central Taiwan. *J Clin Med*. 2021;10(21):4873. doi:10.3390/jcm10214873.

80. Sakr Y, Dünisch P, Santos C, et al. Poor outcome is associated with less negative fluid balance in patients with aneurysmal subarachnoid hemorrhage treated with prophylactic vasopressor-induced hypertension. *Ann Intensive Care*. 2016;6(1):25. doi:10.1186/s13613-016-0128-6.

81. Tagami T, Kuwamoto K, Watanabe A, et al. Effect of triple-h prophylaxis on global end-diastolic volume and clinical outcomes in patients with aneurysmal subarachnoid hemorrhage. *Neurocrit Care*. 2014;21(3):462-469. doi:10.1007/s12028-014-9973-z.

82. Duangthongphon P, Souwong B, Munkong W, Kitkhuandee A. Results of a preventive rebleeding protocol in patients with ruptured cerebral aneurysm: a retrospective cohort study. *Asian J Neurosurg*. 2019;14(3):748-753. doi:10.4103/ajns.AJNS_32_19.

83. Rass V, Bogossian EG, Ianosi BA, et al. The effect of the volemic and cardiac status on brain oxygenation in patients with subarachnoid hemorrhage: a bi-center cohort study. *Ann Intensive Care*. 2021;11(1):176. doi:10.1186/s13613-021-00960-z.

84. Kshettry VR, Rosenbaum BP, Seicean A, Kelly ML, Schiltz NK, Weil RJ. Incidence and risk factors associated with in-hospital venous thromboembolism after aneurysmal subarachnoid hemorrhage. *J Clin Neurosci*. 2014;21(2):282-286. doi:10.1016/j.jocn.2013.07.003.

85. Geraldini F, De Cassai A, Correale C, et al. Predictors of deep-vein thrombosis in subarachnoid hemorrhage: a retrospective analysis. *Acta Neurochir (Wien)*. 2020;162(9):2295-2301. doi:10.1007/s00701-020-04455-x.

86. Dizon MAM, De Leon JM. Effectiveness of initiating deep vein thrombosis prophylaxis in patients with stroke: an integrative review. *J Neurosci Nurs*. 2018;50(5):308-312. doi:10.1097/JNN.0000000000000385.

87. Viarasilpa T, Panyavachiraporn N, Jordan J, et al. Venous thromboembolism in neurocritical care patients. *J Intensive Care Med*. 2020;35(11):1226-1234. doi:10.1177/0885066619841547.

88. Kakkos S, Kirkilesis G, Caprini JA, et al. Combined intermittent pneumatic leg compression and pharmacological prophylaxis for prevention of venous thromboembolism. *Cochrane Database Syst Rev*. 2022;1(1):CD005258. doi:10.1002/14651858.CD005258.pub4.

89. Kole MJ, Wessell AP, Ugiliweneza B, et al. Low-dose intravenous heparin infusion after aneurysmal subarachnoid hemorrhage is associated with decreased risk of delayed neurological deficit and cerebral infarction. *Neurosurgery*. 2021;88(3):523-530. doi:10.1093/neuros/nyaa473.

90. Serrone JC, Wash EM, Hartings JA, Andaluz N, Zuccarello M. Venous thromboembolism in subarachnoid hemorrhage. *World Neurosurg*. 2013;80(6):859-863. doi:10.1016/j.wneu.2013.01.012.

91. Steiner T, Juvela S, Unterberg A, et al. European Stroke Organization guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. *Cerebrovasc Dis*. 2013;35(2):93-112. doi:10.1159/000346087.

92. Kikuta Y, Kubota Y, Nakamoto H, Chernov M, Kawamata T. Nonconvulsive status epilepticus after surgery for ruptured intracranial aneurysms: incidence, associated factors, and impact on the outcome. *Clin Neurol Neurosurg*. 2021;200:106298. doi:10.1016/j.clineuro.2020.106298.

93. Allen BB, Forgacs PB, Fakhar MA, et al. Association of seizure occurrence with aneurysm treatment modality in aneurysmal subarachnoid hemorrhage patients. *Neurocrit Care*. 2018;29(1):62-68. doi:10.1007/s12028-018-0506-z.

94. Chen Y, Xia F, Cai C, et al. Duration and choices of prophylactic anticonvulsants in subarachnoid hemorrhage: a systematic review and meta-analysis. *Neurosurg Rev*. 2021;44(5):2459-2467. doi:10.1007/s10143-020-01466-1.

95. Yoon S, Yoon JC, Winkler E, Liu C, Lawton MT. Nationwide analysis of cost variation for treatment of aneurysmal subarachnoid hemorrhage. *Stroke*. 2019;50(1):199-203. doi:10.1161/STROKEAHA.118.023079.

96. Mahmoud SH, Buxton J. Seizures and choice of antiepileptic drugs following subarachnoid hemorrhage: a review. *Can J Neurol Sci*. 2017;44(6):643-653. doi:10.1017/cjn.2017.206.

97. Huttunen JM, Kurki MI, von und zu Fraunberg M, et al. Epilepsy after aneurysmal subarachnoid hemorrhage: a population-based, long-term follow-up study. *Neurology*. 2015;85(22):1997.

98. Oppong MD, Bastias MJ, Pierscianek D, et al. Seizures at the onset of aneurysmal SAH: epiphenomenon or valuable predictor? *J Neurol*. 2021;268(2):493-501. doi:10.1007/s00415-020-10173-2.

99. García-Ballestas E, Florez-Perdomo WA, Starke RM, et al. Risk of seizures after endovascular management of ruptured intracranial aneurysms: a systematic review and meta-analysis. *J Epilepsy Res*. 2020;10(2):55-61. doi:10.14581/jer.20009.

100. Raper DM, Starke RM, Komotar RJ, Allan R, Connolly Jr ES. Seizures after aneurysmal subarachnoid hemorrhage: a systematic review of outcomes. *World Neurosurg*. 2013;79(5-6):682-690. doi:10.1016/j.wneu.2012.08.006.

101. Rush B, Wiskar K, Fruhstorfer C, Hertz P. Association between seizures and mortality in patients with aneurysmal subarachnoid hemorrhage: a nationwide retrospective cohort analysis. *Seizure*. 2016;41:66-69. doi:10.1016/j.seizure.2016.07.008.

102. O'Connor KL, Westover MB, Phillips MT, et al. High risk for seizures following subarachnoid hemorrhage regardless of referral bias. *Neurocrit Care*. 2014;21(3):476-482. doi:10.1007/s12028-014-9974-y.

103. Daou BJ, Khalsa SSS, Anand SK, et al. Volumetric quantification of aneurysmal subarachnoid hemorrhage independently predicts hydrocephalus and seizures. *J Neurosurg*. 2021;135(4):1155-1163. doi:10.3171/2020.8.JNS201273.

104. Jaja BNR, Schweizer TA, Claassen J, et al. The SAFARI score to assess the risk of convulsive seizure during admission for aneurysmal subarachnoid hemorrhage. *Neurosurgery*. 2018;82(6):887-893. doi:10.1093/neuros/nyx334.

105. Freeman WD. The double-edged sword of seizures and nonconvulsive status epilepticus on aneurysmal subarachnoid hemorrhage outcomes. *Neurocrit Care*. 2022;36(3):699-701. doi:10.1007/s12028-022-01490-7.

106. Samuels OB, Sadan O, Feng C, et al. Aneurysmal subarachnoid hemorrhage: trends, outcomes, and predictions from a 15-year perspective of a single neurocritical care unit. *Neurosurgery*. 2021;88(3):574-583. doi:10.1093/neuros/nyaa465.

107. Wittstock M, Kurtieiev K, Grossmann A, Storch A, Walter U. Epileptic seizures and outcome in different subtypes of subarachnoid haemorrhage—results of a single-center retrospective analysis. *J Clin Neurosci*. 2019;70:123-126. doi:10.1016/j.jocn.2019.08.055.

108. Smith M, Citerio G. What's new in subarachnoid hemorrhage. *Intensive Care Med*. 2015;41(1):123-126. doi:10.1007/s00134-014-3548-5.

109. Dewan MC, Mocco J. Current practice regarding seizure prophylaxis in aneurysmal subarachnoid hemorrhage across academic centers. *J Neurointerv Surg*. 2015;7(2):146-149. doi:10.1136/neurintsurg-2013-011075.

110. Gigliotti MJ, Srikanth S, Cockroft KM. Patterns of prophylactic anticonvulsant use in spontaneous intracerebral and subarachnoid hemorrhage: results of a practitioner survey. *Neurol Sci*. 2022;43(3):1873-1877. doi:10.1007/s10072-021-05588-2.

111. Carnegie V, Schweikert S, Anstey M, et al. A multicentre observational study of the use of antiseizure medication in patients with aneurysmal subarachnoid haemorrhage in the PROMOTE-SAH study. *J Clin Neurosci*. 2022;103:20-25. doi:10.1016/j.jocn.2022.06.022.

112. Kodankandath TV, Farooq S, Wazni W, et al. Seizure prophylaxis in the immediate post-hemorrhagic period in patients with aneurysmal subarachnoid hemorrhage. *J Vasc Interv Neurol*. 2017;9(6):1-4.

113. Suzuki H, Kawakita F, Asada R. Neuroelectric mechanisms of delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage. *Int J Mol Sci*. 2022;23(6):3102. doi:10.3390/ijms23063102.

114. Liao WI, Chien WC, Chung CH, et al. Valproic acid attenuates the risk of acute respiratory failure in patients with subarachnoid hemorrhage. *QJM*. 2018;111(2):89-96. doi:10.1093/qjmed/hcx199.

115. Holtkamp M, Beghi E, Benninger F, et al. European Stroke Organisation guidelines for the management of post-stroke seizures and epilepsy. *Eur Stroke J*. 2017;2(2):103-115. doi:10.1177/2396987317705536.

116. Panczykowski D, Pease M, Zhao Y, et al. Prophylactic antiepileptics and seizure incidence following subarachnoid hemorrhage: a propensity score-matched analysis. *Stroke*. 2016;47(7):1754-1760. doi:10.1161/STROKEAHA.116.013766.

117. Chou SH, Latorre JGS, Alpargu G, Ogilvy CS, Sorond FA, Rordorf G. Outcomes after early anticonvulsant discontinuation in aneurysmal subarachnoid hemorrhage. *J Vasc Med Surg*. 2015;3(1):1000173. doi:10.4172/2329-6925.1000173.

118. Fang T, Valdes E, Frontera JA. Levetiracetam for seizure prophylaxis in neurocritical care: a systematic review and meta-analysis. *Neurocrit Care*. 2022;36(1):248-258. doi:10.1007/s12028-021-01296-z.

119. Karamchandani RR, Fletcher JJ, Pandey AS, Rajajee V. Incidence of delayed seizures, delayed cerebral ischemia and poor outcome with the use of levetiracetam versus phenytoin after aneurysmal subarachnoid hemorrhage. *J Clin Neurosci*. 2014;21(9):1507-1513. doi:10.1016/j.jocn.2014.03.009.

120. Yuan K, Li R, Zhao Y, et al. Pre-operative predictors for post-operative pneumonia in aneurysmal subarachnoid hemorrhage after surgical clipping and endovascular coiling: a single-center retrospective study. *Front Neurol*. 2022;13:893516. doi:10.3389/fneur.2022.893516.

121. Luo M, Yang S, Ding G, Xiao Q. Endovascular coiling versus surgical clipping for aneurysmal subarachnoid hemorrhage: a meta-analysis of randomized controlled trials. *J Res Med Sci*. 2019;24:88. doi:10.4103/jrms.JRMS_414_18.

122. Abecassis IJ, Zeeshan Q, Ghodke BV, Levitt MR, Ellenbogen RG, Sekhar LN. Surgical versus endovascular management of ruptured and unruptured intracranial aneurysms: emergent issues and future directions. *World Neurosurg*. 2020;136:17-27. doi:10.1016/j.wneu.2019.12.127.

123. Belavadi R, Gudigopuram SVR, Raguthu CC, et al. Surgical clipping versus endovascular coiling in the management of intracranial aneurysms. *Cureus*. 2021;13(12):e20478. doi:10.7759/cureus.20478.

124. Cai K, Ni Y, Zhang Y, Shen L, Ji Q, Cao M. Heart rate variability after endovascular coiling is associated with short-term outcomes in patients with subarachnoid hemorrhage. *Neurol Res*. 2018;40(10):856-861. doi:10.1080/01616412.2018.1493973.

125. Ge XB, Yang QF, Liu ZB, Zhang T, Liang C. Increased blood pressure variability predicts poor outcomes from endovascular treatment for aneurysmal subarachnoid hemorrhage. *Arq Neuropsiquiatr*. 2021;79(9):759-765. doi:10.1590/0004-282X-ANP-2020-0167.

126. Zhao B, Yang H, Zheng K, et al. Preoperative and postoperative predictors of long-term outcome after endovascular treatment of poor-grade aneurysmal subarachnoid hemorrhage. *J Neurosurg*. 2017;126(6):1764-1771. doi:10.3171/2016.4.JNS152587.

127. Alaraj A, Hussein AE, Esfahani DR, Amin-Hanjani S, Aletich VA, Charbel FT. Reducing length of stay in aneurysmal subarachnoid hemorrhage: a three year institutional experience. *J Clin Neurosci*. 2017;42:66-70. doi:10.1016/j.jocn.2017.03.049.

128. Thilak S, Brown P, Whitehouse T, et al. Diagnosis and management of subarachnoid haemorrhage. *Nat Commun*. 2024;15(1):1850. doi:10.1038/s41467-024-46015-2.

129. McDonald JS, McDonald RJ, Fan J, Kallmes DF, Lanzino G, Cloft HJ. Comparative effectiveness of unruptured cerebral aneurysm therapies: propensity score analysis of clipping versus coiling. *Stroke*. 2013;44(4):988-994. doi:10.1161/STROKEAHA.111.000196.

130. Hill M, Glenn BA, Reese BJ, Morrow B. Recommendations for endovascular care of stroke patients. *Interv Neurol*. 2018;7(1-2):65-90. doi:10.1159/000481541.

131. Jadhav AP, Molyneaux BJ, Hill MD, Jovin TG. Care of the post-thrombectomy patient. *Stroke*. 2018;49(11):2801-2807. doi:10.1161/STROKEAHA.118.021640.

132. Wang M, Strayer A, Harris O, Rosenberg C, Mummaneni P, eds. *Handbook of Neurosurgery, Neurology, and Spinal Medicine for Nurses and Advanced Practice Health Professionals*. First edition. Taylor and Francis; 2017.

133. Jabbarli R, Pierscianek D, Rölz R, et al. Endovascular treatment of cerebral vasospasm after subarachnoid hemorrhage: more is more. *Neurology*. 2019;93(5):e458-e466. doi:10.1212/WNL.0000000000007862.

134. Hill M, Baumann JJ, Newcommon N. Nursing care of the acute ischemic stroke endovascular thrombectomy patient [published correction appears in *Stroke*. 2022;53(9):e441. doi:10.1161/STR.0000000000000414]. *Stroke*. 2022;53(9):2958-2966. doi:10.1161/STROKEAHA.122.034536.

135. Compher C, Bingham AL, McCall M, et al. Guidelines for the provision of nutrition support therapy in the adult critically ill patient: the American Society for Parenteral and Enteral Nutrition [published correction appears in *JPEN J Parenter Enteral Nutr*. 2022;46(6):1458-1459. doi:10.1002/jpen.2419]. *JPEN J Parenter Enteral Nutr*. 2022;46(1):12-41. doi:10.1002/jpen.2267.

136. McClave SA, Taylor BE, Martindale RG, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) [published correction appears in *JPEN J Parenter Enteral Nutr*. 2016;40(8):1200. doi:10.1177/0148607116670155]. *JPEN J Parenter Enteral Nutr*. 2016;40(2):159-211. doi:10.1177/0148607115621863.

137. Badjatia N, Ryan A, Choi HA, et al. Relationship Between Nutrition Intake and Outcome After Subarachnoid Hemorrhage: Results From the International Nutritional Survey. *J Intensive Care Med*. 2021;36(10):1141-1148. doi:10.1177/0885066620966957.

138. Zhong DY, Li L, Ma RM, Deng YH. The effect of probiotics in stroke treatment. *Evid Based Complement Alternat Med*. 2021;2021:4877311. doi:10.1155/2021/4877311.

139. Tuncay P, Arpaci F, Doganay M, et al. Use of standard enteral formula versus enteric formula with prebiotic content in nutrition therapy: a randomized controlled study among neuro-critical care patients. *Clin Nutr ESPEN*. 2018;25:26-36. doi:10.1016/j.clnesp.2018.03.123.

140. Saran D, Brody RA, Stankorb SM, Parrott SJ, Heyland DK. Gastric vs small bowel feeding in critically ill neurologically injured patients: results of a multicenter observational study. *JPEN J Parenter Enteral Nutr*. 2015;39(8):910-916. doi:10.1177/0148607114540003.

141. Mac Grory B, Vu L, Cutting S, Marcolini E, Gottschalk C, Greer D. Distinguishing characteristics of headache in nontraumatic subarachnoid hemorrhage. *Headache*. 2018;58(3):364-370. doi:10.1111/head.13218.

142. Gaastra B, Carmichael H, Galea I, Bulters D. Duration and characteristics of persistent headache following aneurysmal subarachnoid hemorrhage. *Headache*. 2022;62(10):1376-1382. doi:10.1111/head.14418.

143. Ljubisavljevic S, Milosevic V, Stojanov A, Ljubisavljevic M, Dunjic O, Zivkovic M. Identification of clinical and paraclinical findings predictive for headache occurrence during spontaneous subarachnoid hemorrhage. *Clin Neurol Neurosurg*. 2017;158:40-45. doi:10.1016/j.clineuro.2017.04.017.

144. Glisic EK, Gardiner L, Josti L, et al. Inadequacy of headache management after subarachnoid hemorrhage. *Am J Crit Care*. 2016;25(2):136-143. doi:10.4037/ajcc2016486.

145. Klavansky D, Wanchoo S, Lin A, Temes RE, Rebeiz T. Predictors of opiate utilization in the treatment of headache and impact on three-month outcomes following subarachnoid hemorrhage. *Cureus*. 2021;13(12):e20773. doi:10.7759/cureus.20773.

146. Morad AH, Tamargo RJ, Gottschalk A. The longitudinal course of pain and analgesic therapy following aneurysmal subarachnoid hemorrhage: a cohort study. *Headache*. 2016;56(10):1617-1625. doi:10.1111/head.12908.

147. Lin CM, Wang AY, Chen CC, et al. Warning headache correlates survival rate in aneurysmal subarachnoid hemorrhage. *Biomed J*. 2019;42(5):352-357. doi:10.1016/j.bj.2019.04.006.

148. Subarachnoid Haemorrhage Caused by a Ruptured Aneurysm: Diagnosis and Management. London: National Institute for Health and Care Excellence. November 23, 2022. Accessed October 24, 2024. https://www.nice.org.uk/guidance/ng228/resources/subarachnoid-haemorrhage-caused-by-a-ruptured-aneurysm-diagnosis-and-management-pdf-66143842385605.

149. Čomić H, Rinkel GJE, Vergouwen MDI. The initial time-course of headache in patients with spontaneous subarachnoid hemorrhage. *J Neurol Sci*. 2017;379:55-57. doi:10.1016/j.jns.2017.05.050.

150. Hong CK, Joo JY, Kim YB, et al. The course of headache in patients with moderate-to-severe headache due to aneurysmal subarachnoid hemorrhage: a retrospective cross-sectional study. *Headache*. 2015;55(7):992-999. doi:10.1111/head.12612.

151. Huckhagel T, Klinger R, Schmidt NO, Regelsberger J, Westphal M, Czorlich P. The burden of headache following aneurysmal subarachnoid hemorrhage: a prospective single-center cross-sectional analysis. *Acta Neurochir (Wien)*. 2020;162(4):893-903. doi:10.1007/s00701-020-04235-7.

152. Langley T, Hampton D, Wiggins A, Fraser J. Evaluation of headache intensity and treatment associated with subarachnoid hemorrhage. *JNP*. 2021;17(8):995-998. doi:10.1016/j.nurpra.2021.05.006.

153. Maciel CB, Barlow B, Lucke-Wold B, et al. Acute headache management for patients with subarachnoid hemorrhage: an international survey of health care providers. *Neurocrit Care*. 2023;38(2):395-406. doi:10.1007/s12028-022-01571-7.

154. Eisinger RS, Sorrentino ZA, Lucke-Wold B, et al. Severe headache trajectory following aneurysmal subarachnoid hemorrhage: the association with lower sodium levels. *Brain Inj*. 2022;36(4):579-585. doi:10.1080/02699052.2022.2055146.

155. Devlin JW, Skrobik Y, Gélinas C, et al. Clinical practice guidelines for the prevention and management of pain, agitation/sedation, delirium, immobility, and sleep disruption in adult patients in the ICU. *Crit Care Med*. 2018;46(9):e825-e873. doi:10.1097/CCM.0000000000003299.

156. Barnes PL, Haas H, Beck B. Decreasing headache pain secondary to a subarachnoid hemorrhage with the use of osteopathic manipulative medicine. *AAO Journal*. 2021;31(4):34-38. doi:10.53702/2375-5717-31.4.34.

157. Seo Y, Lee HJ, Ha EJ, Ha TS. 2021 KSCCM clinical practice guidelines for pain, agitation, delirium, immobility, and sleep disturbance in the intensive care unit. *Acute Crit Care*. 2023;38(1):149. doi:10.4266/acc.2022.00094.e1.

158. Nordness MF, Hayhurst CJ, Pandharipande P. Current perspectives on the assessment and management of pain in the intensive care unit. *J Pain Res*. 2021;14:1733-1744. doi:10.2147/JPR.S256406.

159. Sandvik RK, Olsen BF, Rygh LJ, Moi AL. Pain relief from nonpharmacological interventions in the intensive care unit: a scoping review. *J Clin Nurs*. 2020;29(9-10):1488-1498. doi:10.1111/jocn.15194.

160. Gélinas C, Arbour C, Michaud C, Robar L, Côté J. Patients and ICU nurses' perspectives of non-pharmacological interventions for pain management. *Nurs Crit Care*. 2013;18(6):307-318. doi:10.1111/j.1478-5153.2012.00531.x.

161. Ganesan P, Manjini KJ, Vedagiri SCB. Effect of music on pain, anxiety and physiological parameters among postoperative sternotomy patients: a randomized controlled trial. *J Caring Sci*. 2022;11(3):139-147. doi:10.34172/jcs.2022.18.

162. Golino AJ, Leone R, Gollenberg A, et al. Impact of an Active Music Therapy Intervention on Intensive Care Patients. *Am J Crit Care*. 2019;28(1):48-55. doi:10.4037/ajcc2019792.

163. Wartenberg KE, Schmidt JM, Claassen J, et al. Impact of medical complications on outcome after subarachnoid hemorrhage. *Crit Care Med*. 2006;34(3):617-624. doi:10.1097/01.ccm.0000201903.46435.35.

164. Douds GL, Tadzong B, Agarwal AD, Krishnamurthy S, Lehman EB, Cockroft KM. 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;2012:479865. doi:10.1155/2012/479865.

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

166. Müller A, Lorenz A, Seifert B, Keller E. Risk of thromboembolic events with endovascular cooling catheters in patients with subarachnoid hemorrhage. *Neurocrit Care*. 2014;21(2):207-210. doi:10.1007/s12028-014-0001-0.

167. Khan I, Haymore J, Barnaba B, et al. Esophageal cooling device versus other temperature modulation devices for therapeutic normothermia in subarachnoid and intracranial hemorrhage. *Ther Hypothermia Temp Manag*. 2018;8(1):53-58. doi:10.1089/ther.2017.0033.

168. Fischer M, Lackner P, Beer R, et al. Cooling activity is associated with neurological outcome in patients with severe cerebrovascular disease undergoing endovascular temperature control. *Neurocrit Care*. 2015;23(2):205-209. doi:10.1007/s12028-015-0122-0.

169. Heran M, Lindsay P, Gubitz G, et al. Canadian Stroke Best Practice recommendations: acute stroke management, 7th edition practice guidelines update, 2022. *Can J Neurol Sci*. 2024;51(1):1-31. doi:10.1017/cjn.2022.344.

Appendices

Appendix I: MeSH Search Terms

Morbidity and Mortality

Stroke, stroke scale, treatment outcome, prognosis, outcome, predictive, prognostication, prediction, predictability, predictor, disability, functional outcome, recovery, functional recovery, survival, modified Rankin Scale, mRS, indicator, factor, determinant, treatment, intervention, therapy, management, rehabilitation, efficacy, effect, effectiveness, decision making, subarachnoid hemorrhage scale, subarachnoid haemorrhage scale, Hunt and Hess, World Federation of Neurosurgeons scale, modified Fisher scale, Fisher scale, National Institutes of Health Stroke Scale, NIHSS, NIH stroke scale, aneurysm scale, aneurysm score, bleed scale, bleed score, haemorrhagic scale, hemorrhage grade, modified Fisher score, stroke severity, WFNS, subarachnoid hemorrhage scale, subarachnoid haemorrhage scale, SAH scale, stroke scale, hemorrhagic stroke scale

Palliative Care

Palliative care, terminal care, end of life care, palliative consult, subarachnoid hemorrhage, subarachnoid haemorrhage, SAH, aneurysmal subarachnoid hemorrhage, intracranial hemorrhage, cerebral hemorrhage, palliative care nursing, palliative care nurses, palliative medicine, palliative, stroke, aSAH, spontaneous hemorrhage, hemorrhagic stroke, cerebral hemorrhage, goals-of-care, advance care planning, end of life, referral, palliative care consultation, end of life decision, palliative supportive care, supportive care, palliative therapy, palliative treatment, cerebral aneurysm

Nursing Interventions

Subarachnoid hemorrhage, intracranial hemorrhage, hemorrhagic stroke, environment, stimulus, noise, lighting, lights, critical care unit, ICU environment, critical care, hospital, hospitalization, hospitalisation, hospitalizing, hospitalizing, intracranial, lightness, cerebral ischemia, cerebral perfusion, cerebral blood flow, cerebral hemodynamics, cerebral circulation, intracranial hypertension, increased intracranial pressure, intracranial pressure, perfusion, cerebral, cerebrovascular circulation, hemodynamics, haemodynamics, oral care, mouth care, oral hygiene, hygiene bundle, oral hygiene care, oral hygiene regime, toothbrushing, vasospasm, chest percussion, positioning, suction, head of bead, environment

Circulation

Subarachnoid hemorrhage, subarachnoid, hemorrhage, subarachnoid space, volemic, fluid, volume, voluming, subarachnoid haemorrhage, manage, management, balance, normovolemia, euvolemia, organization and administration, organization, administration, disease management

Venous Thromboembolism Prophylaxis

Venous thromboembolism, aneurysmal subarachnoid hemorrhage, aSAH, prophylaxis, pharm, mechanic, compression therapy, sequential compression device, hemorrhagic stroke, intracranial hemorrhage, haemorrhagic stroke, pulmonary embolism, venous thrombosis, deep vein thrombosis, anticoagulants, heparin, enoxaparin, neurologic, critical care, neuro critical care, prevent

Seizure Prophylaxis

Subarachnoid hemorrhage, intracranial hemorrhage, hemorrhagic stroke, seizure, seizure prophylaxis, antiepileptic, subarachnoid hemorrhage, subarachnoid haemorrhage, intracranial hemorrhage, intracranial haemorrhage, hemorrhagic stroke, haemorrhagic stroke, anticonvulsants, antiepileptics, prevention and control, seizuring, seizural, outcomes

Endovascular Therapy

SAH, aSAH, aneurysmal subarachnoid hemorrhage, subarachnoid hemorrhage, endovascular, surgery, IR, nursing care, cursing role, preoperative, postoperative

Enteral Nutrition

Enteral nutrition, outcomes, aneurysmal subarachnoid hemorrhage, aneurysmal subarachnoid haemorrhage , stroke, intracerebral hemorrhage, intracerebral haemorrhage, neurocritical care, tube feeding, gastric feeding, subarachnoid hemorrhage, haemorrhage, continuous enteral nutrition, bolus, management, volume based, administration, strategies, interruptions, nutrition, EN, aSAH, subarachnoid hemorrhage, subarachnoid haemorrhage

Pain Management

SAH, aSAH, aneurysmal subarachnoid hemorrhage, subarachnoid hemorrhage, pain, headache

Thermodynamics

Hemorrhagic stroke, intracranial hemorrhage, haemorrhagic stroke, neuro critical care, cooling devices, surface cooling, invasive cooling, esophageal cooling, temperature management, body temperature, targeted temperature management

Appendix II: Tables of Evidence

Morbidity and Mortality

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Lantigua H, Ortega-Gutierrez S, Schmidt J, 2015	Prospective	1,200 enrolled; 218 died during hospitalization	aSAH	To identify factors that predict hospital death after SAH	In-hospital mortality related to H&H grade: • H&H grade 1: mortality rate 3.5% • H&H grade 2: mortality rate 3.2% • H&H grade 3: mortality rate 9.4% • H&H grade 4: mortality rate 23.6% • H&H grade 5: mortality rate 70.5% mFisher scale grade on admissions predicted in-hospital death after SAH (OR 1.25, 95% CI 1.0-1.5, *P*=.03).
Roquer J, Cuadrado-Godia E, Guimaraens L, et al, 2020	Prospective observational	476	aSAH	To describe the short- and long-term clinical course of aSAH patients and associated death and poor outcomes	Findings at 3-month follow-up: • Association between death and H&H scores 4-5 (*P*<.0001) • Association between death and Fisher scores >4 (*P*<.0001)
Helbok R, Kurtz P, Vibbert M, et al, 2013	Prospective cohort	609	aSAH with H&H grades less than 5 on admission with a worsened H&H grade within 24 hours	To determine factors predicting worsening of H&H grade in the first 24 hours and the impact on mRS at 12 months	For aSAH presenting with H&H <5 and worsening in 24 hours: • Admission H&H grade as a predictor of death or severe disability (mRS 4-6) at 12 months post SAH (OR 2.6, 95% CI 1.9-3.4, *P*<.001) • Admission H&H grade as a predictor of death at 12 months post SAH (OR 2.3, 95% CI 1.6-3.3, *P*<.001)
Autio AH, Paavola J, Tervonen J, et al, 2021	Prospective cohort	269 (grade 4=145; grade 5=124)	aSAH grades 4 and 5	To determine the 3-year clinical condition of patients with grades 4 and 5 aSAH	Alive 12 months after SAH: • H&H grade 4: 63% (n=91) • H&H grade 5: 27% (n=34) Alive 3 years after SAH: • H&H grade 4: 61% (n=88) • H&H grade 5: 26% (n=32)
Chalouhi N, Stravropoula T, Thakkar V, et al, 2014	Retrospective review of a prospectively maintained database	116	aSAH	To compare effectiveness and safety of treatments for vasospasm and identify outcome predictors	Higher H&H grade was a predictor of the need for endovascular retreatment of vasospasm (OR 2.3, 95% CI 1.1-5.1, *P*=.02) in multivariate analysis. Higher H&H grade was predictive of poor functional outcomes (*P*=.01) in univariate analysis. Higher Fisher grade was predictive of poor functional outcomes (*P*=.05) in univariate analysis. Higher H&H grade (OR 4.3, 95% CI 1.6-11.1, *P*=.003) was a predictor of poor outcomes in multivariate analysis.
Wang L, Zhang Q, Zhang G, et al, 2022	Retrospective cohort	227 DCI (n=74) and non-DCI (n=153) groups	aSAH patients with hydrocephalus on admission	To determine the ability of biomarkers and assessment scales to predict DCI after intervention and functional outcomes in aSAH patients with hydrocephalus on admission	H&H grade was an independent risk factor of postoperative DCI in patients with hydrocephalus on admission (OR 1.900, 95% CI 1.359–2.657, *P*=.000). High H&H grade was an independent risk factor of mRS > 2 (poor prognosis) at 6 months in patients with hydrocephalus on admission (OR 2.538, 95% CI 1.532–4.208, *P*<0.05).

Morbidity and Mortality (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Dengler N, Sommerfeld D, Vajkoczy P, et al, 2017	Retrospective	423	aSAH	To compare aSAH grading methods (clinical, radiographic, combined)	Higher mFisher grade predicted cerebral infarction ($P \leq .001$). Higher mFisher grade increased the risk of unfavorable outcomes (OR 1.556, 95% CI 1.301-1.862, $P \leq .001$). H&H predicted cerebral infarction (OR 1.514, 95% CI 1.307-1.753, $P \leq .001$) and unfavorable outcomes (OR 1.891, 95% CI 1.628-2.197, $P \leq .001$). WFNS score predicted cerebral infarction (OR 1.457, 95% CI 1.286-1.651, $P \leq .001$) and unfavorable outcomes (OR 1.703, 95% CI 1.501-1.931, $P < .001$). Clinical and combined scores were shown to be superior to radiographic grading in predicting poor outcomes.
Gross BA, Rosalind Lai PM, Frerichs KU, et al, 2014	Retrospective cohort	255 (clipping=203; coiling=52)	aSAH	To determine the effect of the method of treatment on vasospasm, DCI, and clinical deterioration	H&H grade on admission correlated with the risk of radiographic infarction (OR 1.52, 95% CI 1.00-2.30, $P = .048$).
Alfotih G, Li F, Xu X, et al, 2014	Meta-analysis	7 retrospective studies (2,470 patients; 283 rebleeds)	aSAH patients	To assess common risk factors for rebleeding after aSAH	Rebleed risk factors: • Hunt & Hess: grade 4-5 vs 1-3 (OR 4.94, 95% CI 2.29-10.68, $P < .0001$) • Fisher grade ≥ 3 vs < 3 (OR 2.29, 95% CI 1.45-3.61, $P = .0004$)
Yu H, Zhan R, Wen L, et al, 2014	Retrospective	202 (40 shunt dependent)	aSAH	To determine predictors of shunt insertion after aSAH	Higher H&H grades were associated with shunt-dependent hydrocephalus ($P < .001$). Higher mFisher grades were associated with shunt-dependent hydrocephalus ($P < .001$). Higher H&H grades were associated with worse outcomes ([GOS] $P = 0.048$).
Rass V, Ianosi B, Lindbauer M, et al, 2021	Prospective cohort with retrospective data analysis	297	aSAH	To identify risks for prolonged mechanical ventilation (MV) after SAH and create a predictive scoring tool	Likelihood of MV > 48 vs MV ≤ 48 (Univariate) • Admission H&H of 4 vs 2 (OR 1.55, 95% CI 0.93-2.68, $P < .001$) • mFisher scale of 4 vs 3 ($P < .001$) likelihood of MV > 7 days vs MV ≤ 7 days (Univariate) • Admission H&H of 5 vs 2 (OR 2.71, 95% CI 1.83-4.22, $P < .001$) • mFisher scale of 4 vs 3 ($P < .001$)
Wojak J, Ditz C, Abusamha A, et al, 2018	Retrospective cohort	107	aSAH H&H grade 1-3	To determine the effect of extubation failure (EF) in SAH patients with H&H of 1-3	Predictors of EF vs success (ES) • H&H grade 3 vs 1-2 (46.2% vs 23.4%; $P = .005$)
Suehiro E, Sadahiro H, Goto H, et al, 2016	Retrospective cohort	62	aSAH	To assess body temperature in relation to the type of securement, acuity, and patient outcomes	H&H ≥ 4 was predictive of poor outcomes (OR 19.8, 95% CI 1.5-255.5, $P = .022$).
Smith AM, Clark P, Winter K, et al, 2021	Retrospective cohort	348: 120 AED group vs 228 non-AED group	aSAH	To compare the incidence of seizures in aSAH patients treated with prophylactic AEDs vs no prophylactic AEDs and to identify factors associated with poor outcome (mRS>2)	H&H grade > 2 was significantly associated with poor outcome (mRS>2) in patients (OR 4.2, $P < .0001$).
Wang Z, Zhou J, Liang F, et al, 2020	Retrospective	121	aSAH with ICH	To identify predictive factors of clinical features to create preoperative and postoperative models to predict 6-month outcomes	Preoperative factors associated with poor outcome (mRS 3-6) during the 6-month follow-up period: • WFNS ($z = -2.347$; $P = .019$) • H&H ($z = -3.300$; $P = .001$) • Fisher grade ($z = -2.779$; $P = .005$)

Morbidity and Mortality (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Fang Y, Lu J, Zheng J, et al, 2020	Retrospective	669 349 clipping, 320 coiling	aSAH	To compare various aSAH grading scores in predicting outcomes in clipping and coiling patients	Predictors of mRS > 2 at 3 months (AUC>0.750 considered favorable predictive accuracy) Clipping: • WFNS (OR 2.289, 95% CI 1.898-2.762, AUC 0.785, 95% CI 0.738-0.827) • H&H (OR 4.103, 95% CI 2.946-5.714, AUC 0.773, 95% CI, 0.725-0.815) Coiling: • WFNS (OR 3.135, 95% CI 2.395-4.105, AUC 0.865, 95% CI 0.823-0.901) • H&H (OR 3.832, 95% CI 2.695-5.448, AUC 0.818, 95% CI 0.771-0.858) • Clinical scores (WFNS and H&H) appear superior to radiologic score (mFS) in predicting poor outcomes in both coiling and clipping.
Magee C, Thompson Bastin ML, Graves K, et al, 2019	Retrospective observational cohort	194	aSAH	To investigate fever-related use of antibiotics and the relationship between fever, fever burden, and outcomes in aSAH patients	Fisher grades of 3-4 were associated with fever burden (AUC) (coefficient: 30.41; 95% CI, 3.71-57.12; *P*>.026); multivariate. H&H of 3-5 were associated with a poor outcome defined as mRS of 4-6 (OR 3.23, 95% CI 1.64-6.36, *P*=.001).
Zhang LM, Li R, Zhao XC, et al, 2017	Retrospective	926	aSAH patients who underwent coiling	To identify whether tidal volume, height, and colloid transfusion were related to worse outcomes	H&H grade was associated with worse outcomes defined as mRS ≥ 3 (OR 2.045, 95% CI 1.132-4.293, *P*=.000). Fisher grade was associated with worse outcomes defined as mRS ≥ 3 (OR 2.275, 95% CI 1.366-4.187, *P*=.000).
Duan G, Yang P, Li Q, et al, 2016	Prospective observational longitudinal	520	aSAH 60 years and older who underwent EVT	To create a predictive scoring tool for older aSAH thrombectomy patients	1 year after coiling (multivariate): • H&H grades 4-5 were associated with poor outcomes defined as mRS ≥ 3 (OR 1.758, 95% CI 1.133-2.729, *P*=.012). • Fisher grades 3-4 were associated with poor outcomes defined as mRS ≥ 3 (OR 3.229, 95% CI 2.427-4.295, *P*=.000).
Lindner A, Brunelli L, Verena R, et al, 2016	Prospective	298 250 survivors	aSAH patients transferred to neurorehabilitation center	To assess long-term outcomes for SAH	Factors associated with improved mRS in patients receiving specialized neurorehabilitation during the 12 months follow-up: • Lower ICU discharge mRS (OR 0.65, 95% CI 0.52–0.80, *P*<.001} • Lower ICU admission H&H (OR 0.75, 95% CI 0.61–0.92, *P*<.005)
Solanki C, Pandey P, Rao KV, 2016	Prospective and retrospective	99 with rebleed vs 100 control	aSAH with rebleed before securement	To determine predictors of rebleed before securement	Fisher grades 3-4 were independent predictors of rebleed before securement (OR 0.137, 95% CI 0.052-0.365, *P*<.0001); multivariate
Wang X, Han C, Xing D, et al, 2019	Retrospective	104 (WFNS 4=58; WFNS 5=46)	aSAH WFNS grades 4 and 5	To study the effects of early management of aSAH presenting with WFNS grades 4 and 5 and to determine prognostic factors	CT Fisher grades 1-2 were associated with mRS ≤ 2 at 6 months (OR 12.102, 95% CI 2.101-69.712, *P*=.005) multivariate. WFNS grade 4 vs 5 was associated with mRS ≤ 2 at 6 months (OR 3.852, 95% CI 1.094-13.562, *P*=.036) multivariate. Favorable outcome (mRS≤2) prognostic factors: • WFNS grade 4 vs 5 (OR 10.824, 95% CI 3.735-31.367, *P*<.001) • CT Fisher grades 1-2 vs 3-4 (OR 32.000, 95% CI 6.830-149.922, *P*<.001)
Zheng K, Kuang Zhao B, Tan XX, et al, 2018	Prospective, multicenter cohort	104: 49 coiling, 34 clipping, 21 palliative treatment	aSAH > 60 years with H&H 4-5	To compare treatment strategies and determine predictive factors for outcomes in WFNS grade 4-5 aSAH patients > 60 years of age	Findings (multivariate): • Prognoses of CT Fisher grades 1-2 were better than grades 3-5 (*P*=.025). • Prognoses of WFNS grade 4 were better than grade 5 (*P*=.05).

Morbidity and Mortality (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
de Souza ML, Vieira AC, Andrade G, et al, 2015	Cross-sectional retrospective analysis	248 (aSAH=185; control=63)	Anterior circulation aSAH H&H ≤ 3	To determine the relationship between presecurement Fisher grade and language decline in anterior circulation aSAH	Findings ±8 days after onset: • Fisher 1 and 2 were more likely to have deficits than the control in written comprehension (*P*<.001), oral reading (*P*=.028), semantic fluency (animals and fruits *P*<.001), and phonologic fluency (*P*=.001). • Fisher 3 and 4 were more likely to have deficits than the control in oral comprehension (*P*<.001), naming (*P*=.004), written comprehension (*P*<.001), oral reading (*P*<.001), semantic fluency (animals and fruits *P*<.001), and phonological fluency (*P*=.001). • Fisher 3 and 4 were more likely to have deficits than Fisher 1 and 2 in oral comprehension (*P*=.006), repetition (*P*=.031), naming (*P*=.033), semantic fluency (animals *P*=.003; fruits *P*=.007), and phonological fluency (*P*=.010).
Zhao B, Tan X, Zhao Y, et al, 2015	Retrospective cohort	118	aSAH 4-5	To compare the variations of characteristics and outcomes between patients with surgery ≤ 72 hours and > 72 hours after SAH	Likelihood of surgery ≤ 72 hours post SAH • WFNS grade 5 after emergency resuscitation (*P*<.01) • Lower Fisher grade (*P*=.04) Predictors of excellent outcome (mRS 0-1) • WFNS grade 4 after resuscitation (*P*<.01)
Kim D, Pyen J, Whang K, et al, 2021	Retrospective	166	aSAH	To identify risk factors for rebleeding after endovascular coiling post aSAH	Preoperative mFisher grade was an independent risk factor for rebleeding following coil embolization (OR 2.037, 95% CI 1.077-3.853, *P*<.001).
Pegoli M, Mandrekar J, Rabinstein A, et al, 2015	Retrospective cohort	373 out of 381 aSAH cases	aSAH	To identify predictors associated with mRS of 0-1 at last follow-up within 1 year of aSAH	At follow-up within 1 year post SAH: Factors associated with excellent outcomes (mRS 0-1): • WFNS grade I-III after neurologic resuscitation (OR 15.9, 95% CI 7.17-5.44, *P*<.0001) multivariate • mFisher grade (M=2.8 ± 1.0, OR 0.49, 95% CI 0.38-0.65, *P*<.01) univariate Factors associated with outcomes besides excellent (mRS>1): • mFisher grade (M=3.4 ± 0.8, OR 0.49, 95% CI 0.38-0.65, *P*<.01) univariate
Hammer A, Erguth F, Hohenhaus M, et al, 2018	Retrospective observational	203	aSAH	To assess the effect of complications and interventions on outcomes.	WFNS grade as a predictor of poor outcome (mRS>2) after 1 year (OR 3.86, 95% CI 2.23–6.68, *P*<.0001) WFNS grade as a predictor of mortality after 1 year (OR 4.67, 95% CI 2.49–8.75, *P*<0.0001)
Ota N, Noda K, Chida D, et al, 2022	Retrospective	260	aSAH	To determine the effect of the removal of cisternal SAH clots at time of aneurysm securement	Higher WFNS grades were more likely to have a bad outcome defined as mRS > 2 at 6 months (adj OR 2.18, 95% CI 1.63-2.92, *P*<.001).
Ota N, Noda K, Hatano Y, et al, 2019	Retrospective	186	aSAH	To ascertain outcome predictors in poor-grade SAH	WFNS grade 5 was predictive of poor outcome (mRS 3-6) at 1 year after SAH (OR 15.35, 95% CI 4.01-58.78, *P*<.001).
Wong GK, Lam SW, Ngai K, et al, 2013	Prospective observational	168	aSAH	To ascertain whether cognitive domain deficits impacted function in aSAH patients at 1 year	mRS 3–5 at 1 year was associated with the number of cognitive domain deficits (OR 2.3, 95% CI 1.4-3.8, *P*=.002). WFNS grades 4 and 5 were associated with death or inability to follow commands at 1 year post aSAH (*P*<.001).
Galea J, Dulhanty L, Patel H, 2017	Prospective	3,341	aSAH	To ascertain predictors of poor outcomes in aSAH	Worse grade WFNS was predictive of negative outcomes defined as GOS 1-3 (OR 2.06, 95% CI 1.91-2.22, *P*<.001).

Palliative Care

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Creutzfeldt CJ, Hanna MG, Cheever CS, et al, 2017	Prospective, longitudinal cohort	91 (stroke=30)	Family members of patients discharged from the neuro-ICU	To examine the effect of daily use of a palliative care needs checklist on family perspectives of ICU care, long-term outcomes among family members and patients	Family satisfaction with care: • Relatively high with mean subscale and total scores between 81 and 87 on a scale from 0 to 100 Family satisfaction scores for different levels of mRS: • Distribution of family satisfaction scores for patients who did not die in the hospital suggested a linear relationship with lower satisfaction scores as the mRS increased. Patients who died in the hospital without palliative care screen vs with palliative care screen: • Family satisfaction: Decision 84.5 vs 80.5, *P*=0.35; Care score 87 vs 85.3, *P*=.72 • Decision: 84.1 vs 80.5, *P*=.43
Markovitz N, Morgenstern LB, Shafie-Korassani F, et al, 2020	Cross-sectional survey	145 (patients=66; surrogates=79)	Ischemic stroke or intracerebral hemorrhage patients and surrogates/family	To study the withdrawal of care among SAH patients	Overall quality of end-of-life care was generally high (median 8.3, quartiles 6.1, 9.6).
Blacquiere D, Bhimji K, Meggison H, et al, 2013	Qualitative	15	Families of deceased stroke patients	To assess families' perceptions of palliative care after stroke	Family member perceptions of palliative care after stroke (Likert 1-10 scores): • Overall satisfaction: 9.1 • Family emotional support: 8.7 • Ensured dignified death: 9.1 • Adequate symptom control: 8.7 • Respectful medical care: 9.1 • Communication with patient: 9.2

Nursing Interventions

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Nyholm L, Howells T, Enblad P, 2017	Prospective observational	28	TBI patients > 16 years, mechanical ventilation, ICP monitoring	To determine the occurrence of secondary increased ICP associated with nursing interventions	Patients with ICP > 15 mmHg have a higher risk than those with ICP < 15 mmHg of developing secondary brain insult/injury (OR 4.7, *P*=.01).
Szabo C, Grap M, Munro C, et al, 2014	Observational	23 (SAH=7)	TBI, aSAH, ICH, IVH, brain tumor, craniectomy	To evaluate ICP and CPP 5 minutes before, during, and 5 minutes after oral care	CPP: • Difference before, during, and after oral care (*P*=.3529) ICP increase: • From before to during oral care (*P*=.0551) • From during oral care to after oral care (*P*=.4859) • From before oral care to after oral care (*P*=.0026) Relationship between ICP change and oral care: • Duration (*P*=.5687) • Oral care intensity (*P*=.9154)
Tomar G, Singh G, Bithal P, et al, 2019	RCT—crossover	46	Adult severe TBI on mechanical ventilation with ICP monitoring	To compare CPT vs mechanical CPT applied 10 minutes alternately, separated by 4-hour intervals	Manual CPT group compared to mechanical technique: • Rise in ICP greater in manual CPT (*P*=.01) • Peak mean ICP, HR, and MAP higher in manual CPT group (*P*<.001)
Singh S, Chouhan RS, Bindra A, et al, 2018	Prospective, randomized	60	Severe adult TBI on mechanical ventilation with parenchymal ICP monitoring	To compare the effect of IV dexmedetomidine and lidocaine on ICP and systemic hemodynamic response to CPT and ETS	Both dexmedetomidine and lidocaine blunted rise in ICP in response to CPT and ETS. After CPT and ETS, dexmedetomidine group vs lidocaine group (mean ± SD) Dexmedetomidine group: • CPP decreased (68±12, *P*=.0001) • MAP decreased (79±13, *P*=.0001) • ICP (11±4, *P*=.13) Lidocaine group**:** • CPP decreased (84±14, *P*=.19) • MAP decreased (97±13, *P*=.07) • ICP (13±6, *P*=.52) Intergroup comparison: • CPP decreased (*P*=.0001) • MAP decreased (*P*=.00), ICP (*P*=.14)
Jiang Y, Ye Z, You C, et al, 2015	Systematic review with meta-analysis	10 studies; 237 patients	Craniotomy patients including TBI, SAH, ICH, tumor	To determine optimal head elevation degree to decrease ICP in post craniotomy patients ICP at HOB elevations of 0, 10, 15, 30, and 45 degrees	ICP at 30 and 45 degrees was significantly lower than at 10 and 15 degrees ICP at 30 degrees was not significantly different than at 45 degrees
Alarcon J, Rubiano A, Okonkwo D, et al, 2017	Systematic review	3 RCTs; 20 patients	Severe TBI with different HOB elevations or backrest positions	To evaluate the effects of HOB elevation on clinical outcomes	HOB 30 degrees reduced ICP with concomitant increments in CPP

Circulation

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Vergouw LJM, Egal M, Bergmans B, et al, 2020	Retrospective cohort	223	aSAH	To assess if high, early fluid input or positive fluid balance is associated with DCI To assess if fluid input can be safely decreased using transpulmonary thermodilution	Absence of DCI vs presence of DCI • Admit GCS: 14 (13-15) vs 13 (6-15), *P*=.001 • Day 1 fluid input (L): 4.4 ± 0.13 vs 4.9 ± 0.19, *P*=.005 • Day 2 fluid input (L): 4.2 ± 0.12 vs 5.0 ± 0.21, *P*=.004 • Day 1 MAP: 93.7 ± 1.1 vs 99.1 ± 1.4, *P*=.002 • Day 2 MAP: 97.2 ± 1.2 vs 101.0 ± 1.8, *P*=.072 • Follow-up GOS (months): 3.5 ± 0.1 vs 2.8 ± 0.2, *P*=.008 • 6-month mortality: 9% vs 38%, *P*<.001
Rass V, Gaasch M, Kofler, M, et al, 2019	Retrospective cohort	237	Nontraumatic SAH	To assess the effect of daily fluid intake and balance on complications and functional outcomes	Daily fluid intake: • Prolonged mechanical ventilation: Wald 20.08, *df*=1, *P*<.001 • DCI: OR 1.31, 1.14-1.51, *P*<.001 • Poor 3-month outcomes: OR 1.25, 1.10-1.41, *P*<.001
Kuwabara K, Fushimi K, Matsuda S, et al, 2013	Retrospective	5,400	SAH	To determine if hypervolemia and hemodynamic augmentation during the pre-DCI period is associated with decreased complications and mortality	Mortality (OR, 95% CI, *P*) • Normalized fluid volume (mL/kg/day) pre-DCI: 1.02, 1.01-1.03, *P*<.001 • Normalized fluid volume (mL/kg/day) during DCI: 0.95, 0.94-0.96, *P*<.001 Consciousness deterioration (OR, 95% CI, *P*) • Normalized fluid volume (mL/kg/day) pre-DCI: 1.01, 1.01-1.02, *P*=.001 • Normalized fluid volume (mL/kg/day) during DCI: 0.95, 0.95-0.96, *P*<.001 Complications (OR, 95% CI, *P)* • Normalized fluid volume (mL/kg/day) pre-DCI: 0.99, 0.98-0.99, *P*=.004 • Normalized fluid volume (mL/kg/day) during DCI: 1.01, 1.01-1.02, *P*<.001 Reintubation (OR, 95% CI, *P)* • Normalized fluid volume (mL/kg/day) pre-DCI: 1.01, 1.00-1.02, *P*=.002
Togashi K, Joffe AM, Sekhar L, et al, 2015	Randomized pilot	20	aSAH	To assess feasibility, adherence and retention of volume expansion, and BP management on DCI	Normo-volemia (NV) vs hypervolemia (HV): • Mean days on vasopressors: 5.5 ± 3.9 vs 6.4 ± 4.2, *P*=.53 • 6-month mRS: 1.7 ± 0.67 vs 1.6 ± 1.84, *P*=.87 • Neuropsychiatric exam: 75 ± 26 vs 68 ± 30, *P*=.64 • Severe vasospasm: 10% vs 20%, *P*=.53 • Mortality: 0 vs 10%, *P*=.07 CBP(10) vs ABP(10) vs NV(10) vs HV(10) • MI: 0 vs 0 vs 0 vs 0 • CHF: 0 vs 0 vs 0 vs 0 • PE: 1 vs 2 vs 1 vs 2 • Mortality: 0 vs 1 vs 0 vs 1
Szmuda T, Waszak PM, Rydz C, et al, 2014	Prospective	41	SAH	To assess errors in fluid administration, fluid monitoring and charting completeness during triple-H therapy	Presence of DCI vs absence of DCI • Fluid intake: *P*=.02 • Fluid output: not significant • Fluid balance: not significant 25% errors in intake calculations (omissions of IV fluid documentation) were noted and subsequent errors in next intake orders were based on incorrect previous day miscalculations.

Circulation (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Martini RP, Deem S, Brown M, et al, 2012	Retrospective cohort	356	Adult SAH	To assess the association between early fluid balance and outcomes of SAH patients	3-day positive balance vs 3-day negative balance • H&H ≥ 3: 43% vs 55%, *P*=0.03 • GCS: 10.0 ± 4.5 vs 8.7 ± 4.5 *P*<0.01 • ICU ALOS: 10 ± 5 vs 12 ± 6, *P*=0.04 • ICU discharge (d/c) GCS: 12 ± 4 vs 11 ± 4, *P*=0.01 • ALOS: 17 ± 9 vs 20 ± 14, *P*=0.02 Mortality or new stroke: 25% vs 38%, *P*=0.02
Kissoon NR, Mandrekar JN, Fugate JE, et al, 2015	Retrospective	288	SAH > 18 years	To assess if positive fluid balance adversely affects clinical outcomes	mRS 0-2 vs mRS 3-6 Net fluid balance (L): -0.02 ± 5.3 vs 3.52 ± 5.51, *P*<.001

Ibrahim GM , Macdonald RL, 2013	Randomized	413	SAH, sensitivity analysis of CONSCIOUS 1 subset	To assess delayed ischemic neurological deficit (DIND), delayed ischemia, and outcomes among propensity matched cohorts (+/- colloid treatment)
				
				To assess the effects of positive fluid balance on DIND, DI, and outcomes
					Positive fluid balance vs negative fluid balance (L/day)
					• ICU ALOS days: 17 vs 12, *P*<0.001
					• Negative fluid balance associated with DI: OR 0.13, 95% CI 0.02-0.57, *P*=.013
					• Positive fluid balance worse than moderate disability mRS: OR 2.14, 95% CI 1.11-4.19, *P*=.02
					• Colloids and positive fluid balance
					  • NIHSS: *P*=.04
					  • mRS: *P*=.02

Wu CL, Pai KC, Wong LT, et al, 2021	Retrospective cohort	6,978	All surgical ICU admits (49.6% NICU, 20.4% CV)	To assess the impact of day 1-3 and day 4-7 fluid balance on long-term mortality in critically ill patients	Mortality vs nonmortality • Days 1-3 fluid balance (mL): 269.5 ± 2,300.3 vs 145.4 ± 1,526.2, *P*<.01 • Days 4-7 fluid balance (mL): 269.5 ± 2,300.3 vs 145.4 ± 1,526.27, *P*<.01

Circulation (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Sakr Y, Dünisch P, Santos C, et al, 2016	Retrospective	142	SAH > 18 years	To assess the impact of fluid balance on neurological outcomes, adjusting for confounders	Cumulative ICU days fluid balance (L): -8.7 (-14.0 to -2.2) 3 months GOS 4-5, cumulative fluid balance, *P*<.001 3 months GOS 4-5, first 7 days fluid balance, *P*<.001 3 months GOS 4-5, cumulative ICU days fluid balance, *P* not significant 6 months GOS 4-5, cumulative fluid balance, *P*<.001 6 months GOS 4-5, first 7 days fluid balance, *P*<.001 6 months GOS 4-5, cumulative ICU days fluid balance, *P*=.05-.01 2 months GOS 4-5, cumulative fluid balance, *P*<.001 12 months GOS 4-5, first 7 days fluid balance, *P*<.001 12 months GOS 4-5, cumulative ICU days fluid balance, *P*=.05-.01 Fluid balance with GOS ≤ 3 after 3 months: OR 1.24, 1.08-1.42, *P*=.002 Fluid balance with GOS ≤ 3 after 6 months: OR 1.21, 1.06-1.39, *P*=.006 Fluid balance with GOS ≤ 3 after 12 months: OR 1.19, 1.04-1.36, *P*=.011 WFNS (per point) with GOS ≤ 3 after 3 months: *P* value not documented WFNS with GOS ≤ 3 after 6 months: OR 1.59, 0.97-2.61, *P*=.064 WFNS with GOS ≤ 3 after 12 months: OR 1.84, 1.15-2.93, *P*=.011
Tagami T, Kuwamoto K, Watanabe A, et al, 2014	Prospective	178	SAH	To assess the effects of triple-H therapy on global end diastolic volume index	Triple-H therapy vs no triple H therapy (control) • DCI %: 24.2% vs 17.2%, *P*=.27 • 28-day GOS recovery: *P*=.47 • Good recovery: 26.7% vs 23.3%, *P* not documented • Mortality: 6.7% vs 6.0%, *P* not documented • Cohorts were different: clip vs coil, *P*=.001; fasudil use, *P*=.01

Circulation (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Duangthongphon P, Souwong B, Munkong W, et al, 2019	Prospective cohort	N=208 (pregroup=104; postgroup=104)	aSAH	To assess if a preventative rebleed protocol (REST—**R**est, pain control, minimize stimuli, laxatives; **E**uvolemia; **S**BP control < 160 mmHg; **T**reatment: earliest possible and IV tranexamic acid for treatment delay > 72 hrs) improved patient outcomes	In hospital rebleeding • Before protocol: 6.7% (7/104) • After protocol 2.8% (3/104) • OR 0.4, 95% CI, 0.10–1.63, *P*=.20 DCI: after protocol, 7.7% had DCI vs 44.2% in the before protocol (OR 0.10, 95% CI 0.04–0.23, *P*<.001). Pneumonia: 26.9% after protocol vs 36.5% before protocol (OR 0.63, 95% CI 0.35–1.15, *P*=.13). Hospital LOS: median of 8 days after protocol vs 11 days before protocol (*P*=.09) Unfavorable outcomes (mRS 4–6) at 1 year • Before protocol: 33 of 104 (32.7%) • After protocol: 28 of 104 (26.9%) • OR 0.74, 95% CI 0.41–1.35, *P*=.33 WFNS grade 1-3 after protocol • Lower in-hospital rebleeding rates (2.8% vs 6.7%, OR 0.4, 95% CI 0.07–2.16, *P*=.29) • Lower rates of DCI (4.3% vs 40.5%, OR 0.06, 95% CI 0.01–0.22, *P*<.001) • Lower proportion of unfavorable outcomes at 1 year (mRS 4–6, 12.8% vs 27.0%, OR 0.40, 95% CI 0.17–0.95, *P*=.03) WFNS grade 4-5 after protocol • Lower rates of DCI (14.7% vs 53.3%, OR 0.15, 95% CI 0.04–0.49, *P*<.001)
Rass V, Bogossian EG, Ianosi BA, et al, 2021	Prospective, observational cohort	60	Poor-grade (H&H 4-5) SAH	To assess the associated between cardiac index ([CI] as a proxy for euvolemia) and $PbtO_2$ To assess the effect of fluid challenges on CI and $PbtO_2$ Target: CI ≥ 3.0 L/min/m2 Target: $PbtO_2$ < 20 mmHg	Brain tissue hypoxia (BTH) CI target: 3.0–5.0 L/min/m2 • Decreased $PbtO_2$ levels were associated with poor 3-month functional outcome, corrected for age and admission H&H (adj OR 0.99, 95% CI .98–.99, *P*=.011). • Higher CI levels were associated with higher $PbtO_2$ levels (Wald=14.2, *P*<.001). • Higher CI levels were associated with decreased odds of BTH (OR 0.88, 95% CI 0.78–0.995, *P*=.042). • Higher CI was associated higher $PbtO_2$ (Wald=10.5, *P*<.001.) • Normal $PbtO_2$ vs BHT • CI L/min/m2: (4.0±1.0 vs 3.8±0.8, *P*=.042) • Fluid intake and $PbtO_2$: *P*=.94 • Fluid balance and $PbtO_2$: *P*=.85 • Fluid challenge and CI: from 3.5 ± 1.2 to 4.4 ± 1.6 L/min/m2, *P*<.001 • Fluid challenge and $PbtO_2$: from 3.5 ± 1.2 to 4.4 ± 1.6 L/min/m2, *P*=.89

Venous Thromboembolism Prophylaxis

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Kole MJ, Wessell AP, Ugiliweneza B, et al, 2021	Retrospective	556 (subcutaneous heparin=323; low-dose IV heparin=233)	aSAH	To assess the safety and efficacy of LDIVH for DVT prophylaxis	LDIVH vs subcutaneous heparin • Cerebral infarct: 9% vs 19%, *P*<.001, OR 0.40, 95% CI 0.23-0.71 • Delayed neurological deficit: OR 0.53, 95% CI 0.33-.085, *P*=.004 • No radiographic vasospasms: 39% vs 60%, *P*<.001, OR 1.62, 95% CI 0.99-2.66 • LDIVH cohort 2.2 times less likely to have DVT: OR 0.46, 95% CI 0.16-0.91
Serrone JC, Wash EM, Hartings JA, et al, 2013	Retrospective	196	aSAH	To propose a refinement for risk stratification of venous thromboembolism	2% risk of pulmonary embolism despite aggressive chemoprophylaxis aSAH patients may benefit from both mechanical and early chemoprophylaxis (subcutaneous heparin).

Seizure Prophylaxis

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Kikuta Y, Kubota Y, Nakamoto N, et al, 2021	Prospective cohort	66	aSAH patients undergoing continuous EEG (cEEG) monitoring	To assess incidence of nonconvulsive status epilepticus (NCSE), factors associated with NCSE, and NCSE impact on outcomes	All NCSE occurred in microsurgical patients (15%) NCSE—Male (70%, *P*=.041) Factors associated with NCSE • GCS < 13: 90%, *P*=.004 • HH > 2: 90%, *P*=.013 • Hydrocephalus: 70%, *P*=.04 Positive NCSE vs negative NCSE • LOS: 62.5 vs 39.5, *P*=.015. • Favorable disability: 20% vs 54%, *P*=.084
Allen BB, Forgacs PB, Fakhar MA, et al, 2018	Retrospective cohort	282	aSAH	To assess the association between new onset seizures and aneurysm treatment, clinical severity, and outcomes	6.4% seizure incidence Positive seizures • High-grade (H&H 4-5): *P*=.016 • Clipping: *P*=.0089 • Lower GCS at ICU discharge: *P*<.001 • Higher mRS on follow-up: *P*<.001 • Infarct: *P*<.05
Chen Y, Xia F, Cai C, et al, 2021	Meta-analysis	5 studies (poor clinical outcomes = 959; in-hospital seizure incidence = 1,024)	SAH patients	To assess effectiveness of different durations of prophylactic AED. To assess in-hospital seizure incidence	AED use > 3 days is associated with poor clinical outcomes mRS (3-6): OR 1.55, 95% CI 1.10-2.39, *P*=.045. No association between duration of prophylactic AED use and in-hospital seizures: OR 0.62, 95% CI 0.18-2.15, *P*=.447
Yoon SJ, Joo JY, Kim YB, et al, 2015	Retrospective	84	aSAH H&H Grades 1-3	To assess effects of prophylactic AED on clinical outcomes	Positive AED vs no AED • Clinical outcomes at discharge: *P*=.607 • Clinical outcomes after 6 months: *P*=.178 Seizure incidence: 0 vs 0 Variables independently associated with poor outcomes in the AED group: • Hydrocephalus (OR 14.286, 95% CI 1.277 to 166.67, *P*=.031) • Symptomatic vasospasm (OR 9.615, 95% CI 1.088-83.333, *P*=.042)
Huttunen J, Kurki MI, von Und Zu Fraunber M, et al, 2015	Retrospective chart review	875	Saccular intracranial aneurysm SAH (sIA—SAH)	To assess the incidence epilepsy after sIA—SAH and risk factors of epilepsy after sIA-SAH	1-month mortality: 6% 6-month mortality: 9% 1-year mortality: 11% 5-year mortality: 12% Epilepsy incidence at 1 year: 8% Epilepsy incidence at 5 years: 12% Risk factors for epilepsy • ICH > 15 cm^3: *P*=.02 • H&H 3-5: *P*>.001 • Acute seizures: > 0.001
Darkwah Oppong M, Bastias MJ, Pierscianek D, et al, 2021	Retrospective chart review	984	aSAH	To assess predictors of seizures at onset (SAO) of aSAH and the impact of seizures at onset of aSAH	SAO associated with • Younger age (<51 years): *P*<.001 • WFNS grade > 3: *P*<.001 • Aneurysm location—ACA: *P*=.037 • Irregular sac: *P*=.019 • Admit body temperature > 38.3C: *P*=0.008 Complications SAO associated with • Early infarct: *P*=.004 • Decomp craniotomy: *P*=.024

Seizure Prophylaxis (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
García-Ballestas E, Florez-Perdomo WA, Starke RM, et al, 2020	Meta-analysis	5 studies; 3,077 patients	Ruptured aSAH treated endovascularly	To assess seizure risk after endovascular treatment	Seizure risk factors: • Worse clinical severity: OR 1.79, 95% CI 1.37-2.34, *P*<.00001 • Severe vasospasm: OR 2.2, 95% CI 1.67-2.92, *P*<.0001 • Cerebral infarct: OR 5.19, 95% CI 3.23-8.35, *P*<.0001 • Cerebral edema: OR 1.79, 95% CI 1.37-2.34, *P*<.000
Raper DM, Starke RM, Komotar RJ, et al, 2013	Meta-analysis	25 case series and RCT studies; 7,002 patients	aSAH	To assess seizure risk post aSAH and AED prophylaxis effectiveness	Early (after intervention) seizure incidence: 2.3% Late (after discharge) seizure incidence: 5.5% Seizure risk factors • Worse clinical severity: OR 1.79, 95% CI 1.37-2.34, *P*<.00001 • Severe vasospasm: OR 2.2, 95% CI 1.67-2.92, *P*<.00001 • Cerebral infarct: OR 5.19, 95% CI 3.23-8.35, *P*<.00001 • Cerebral edema: OR 1.79, 95% CI 1.37-2.34, *P*<.00001 Positive AED vs no AED • Early seizure: 2.3% vs 3%, *P*>0.99 • Late seizure: 5.9% vs 6.3%, *P*>0.99 • Mean time to late seizure (months): 5.6 vs 6.5 Clip vs coil • Early seizure: 2.4% vs 1.4%, *P*=.16 • Late seizure: 6.5% vs 3.3% *P*<.003
Rush B, Wiskar K, Fruhstorfer C, Hertz P, et al, 2016	Retrospective cohort	12,647	aSAH ≥ 18 years	To assess the association between seizures and hospital mortality	Incidence: 10.6% Positive seizure vs negative seizure: • Unadjusted mortality higher for patients with seizures: 16.2% vs 11.6%, *P*<.01 • Severity adjusted mortality: OR 1.57, 95% CI 1.32-1.87, *P*<.01 • Age: 52.3 years vs 54.8 years, *P*<.01 • Male: 35.6% vs 31%, *P*<.01 • LOS: 18.3 days vs 14.8 days, *P*<.01
Smith AM, Clark PR, Winter KA, et al, 2021	Retrospective chart review	348	SAH	To assess seizure incidence and outcomes between patients on AEDs vs those not on AEDs	With AED vs without AED: • ICH: 10.5% vs 22.5%, *P*=.004 • Seizure incidence: 4.8% vs 8.3%, *P*=.24 Association with poor outcome mRS > 2; OR, *P*-value • Seizure: 8.34, *P*=.0008 • DCI: 3.4, *P*<.0001 • AED: 1.7, *P*=.041 • Age: 1.07, *P*<.001 • H&H > 4: 4.2, *P*=.0002 • Hydrocephalus: 2.76, *P*<.0001 Positive seizure: • Clip vs coil: 3.8, *P*=.012 • DCI: 2.77, *P*=.023

Seizure Prophylaxis (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
O'Connor KL, Westover MB, Phillips MT, et al, 2014	Retrospective	69	High grade (HH 4-5 or Fisher 3), nontraumatic aSAH	To determine seizure predictors, frequency, and impact in patients with high-grade aSAH	Frequency: 11.6% Predictors: • MCA aneurysm location was associated with increased seizure probability: OR 6.7, *P*<.05. • Prophylactic AED was associated with decreased seizure probability: OR 0.23, *P*<.01. Clinical impact: • Poor mRS and H&H 4-5: 52.2%, *P*≤.01 • Poor mRS and clinical suspicion for seizure: 58.3%, *P*≤.01
Daou GJ, Khalsa SS, Anand SK, et al, 2021	Prospective cohort	288	aSAH	To assess the association of aSAH volume with hydrocephalus and seizures.	13.2% developed seizures. Seizure was associated with: • Larger mean hemorrhage volume: mean difference = 17.3 ml, *P*=.01 • Larger hemorrhage volume on CT: OR 2.81, 95% CI 1.03-7.8, *P*=.014 • Hemorrhage volume > 50 ml: OR 2.81, 95% CI 1.03-7.8, *P*=.022 • Hemorrhage volume > 75 ml: OR 3.07, 95% CI 1.46–6.47, *P*=.003 • Younger age: OR 1.04, 95% CI 1.01–1.06, *P*=.003 • H&H ≥ 4: OR 3.46, 95% CI 1.68–7.09, *P*=.001 • WFNS grade ≥ 4: OR 2.96, 95% CI 1.45–6.07, *P*=.003 • Intraparenchymal hemorrhage: OR 2.87, 95% CI 1.42–5.79, *P*=.003 • Rebleeding: OR 4.58, 95% CI 1.41–14.8, *P*=.011
Jaja BNR, Schweizer TA, Claassen J, et al, 2018	Prospective validation study	1,500 and 852 (validation)	SAH patients	To develop and validate a risk score for convulsive seizure during acute SAH.	Population characterization: Seizure vs no seizure: 64 years vs 53 years, *P*=.001
Samuels OB, Sadan O, Feng C, 2021	Retrospective	3,970	SAH	To describe trends in care and outcomes in SAH patients	Poor functional outcome associations (OR, CI, *P*) • Seizures: 1.69, 1.07-2.70, *P*=.025 • Admit H&H: 1.67, 1.45-1.94, *P*=.000 • Coil: 0.35, 0.25-0.48, *P*=.000 Clip vs coil • ICU H&H 1-2: 47.8% vs 39.8%, *P*=.003 • ICU H&H 3: 35.26% vs 42.5%, *P*=.006 • Respiratory failure: 53.8 vs 44.6%, *P*=.001 • Vent > 96 hours: 42.4% vs 27.6%, *P*=.000 • H&H 1-2, discharge mRS ≤ 2: 54.4% vs 77.7%, *P*<.01 • H&H 3, discharge mRS ≤ 2: 22.4% vs 47.6%, *P*<.01 • Mortality, H&H 1-2: 8.5% vs 3.5%, *P*<.01 Mortality, H&H 3: 16.4% vs 9.5%, *P*=.02

Seizure Prophylaxis (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Wittstock M, Kurtieiev K, Grossmann A, et al, 2019	Retrospective cohort	109	aneurysmal and nonaneurysmal SAH	To assess SAH seizure incidence and the impact of seizure on aneurysmal and nonaneurysmal SAH	Incidence: • Perimesencephalic SAH: 22.9% • Nonaneurysmal SAH: 10.1% • aSAH: 67% Peri-mesencephalic vs non-aneurysmal SAH vs aSAH • Seizure incidence: *P*=.232 • Vasospasm: *P*=.028 • Mortality: 12% vs 9.1% vs 17.8%, *P*=.647 • D/C mRS: 1.2 ± 1.8 vs 1.9 ± 2 vs 3.3 ± 2.2, *P*=.000 Good vs poor mRS : OR, 95% CI, P • Acute epileptic seizure: 0.350, 0.018–6.961, *P*=.491 • Remote epileptic seizure: 1.716, 0.146–20.105, *P*=.667 • H&H: 0.820, 0.265–2.535, *P*=.295 • Modified Fisher: 0.925, 0.325–3.635, *P*=.911 • Admit NIHSS: 1.520, 1.055–2.190, *P*=.025
Dewan MC, Mocco J, 2015	Mixed-methods survey	25 institutions	25 geographically diverse US hospitals with > 100 aSAH patients/ year	To determine current seizure prophylaxis practices in aSAH	Seizure prophylaxis practices: • Routine EEG: 8% • Believed seizure prophylaxis was useful: 52% • Believed seizure prophylaxis was NOT useful: 40% • Routine seizure prophylaxis use: 68% • Ordered ASM prophylaxis, despite not believing it was necessary: 16% • Only ordered if positive ICH: 8% Only ordered if Fisher grade was ≥ 3: 8% • Only ordered if H&H ≤ 2 prior to rupture: 12% • Ordered for all clippings, but not for coiling unless ICH was present: 1% • Levetiracetam (LEV) first choice: 94% • Phenytoin (PHT) first choice: 1% (n=1) • ASM admin range: 1 day to 6 weeks, Mean = 13.2 days
Gigliotti MJ, Srikanth S, Cockroft KM, 2022	Qualitative survey	794 (n=103; neurosurgeons = 84%; vascular neurosurgeons = 38%; neurocritical care specialists = 10%)	US and Canadian neurosurgeons, vascular neurosurgeons, neurocritical care specialists	To determine prophylactic ASM use in spontaneous intracerebral hemorrhage and aSAH in North America	Prophylactic ASM use: • No difference in ASM use by MD specialty • aSAH prophylactic ASM: 43% • No routine ASM prophylaxis: 22% • All aSAH routine AED prophylaxis: 82%. • ASM LEV: 99% • Serum ASM level check: 5%

Seizure Prophylaxis (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Carnegie V, Schweikert S, Anstey M, et al, 2022	Prospective observational	357	SAH admitted to ICU	To describe ASM prescription patterns and associations between ASM use and death and disability	Prescribed ASM: 40% Prescribed ASM ≥ 7 days: 66% ASM prescribed: • LEV: 30% • PHT: 2.8% • Valproic acid (VPA): 1.1% • Other ASM: 6.4% Number of ASM concurrently used: • One: 94% • Two: 4.9% • Three: 0.7% Positive ASM vs no ASM • Witnessed prehospital seizure: 29% vs 6.6%, *P*<.001 • Acute Physiology and Chronic Health Evaluation: 50.5 (35, 68) vs 41 (28, 57), *P*=.001 • Admit intubated: 67% vs 46%, *P*<.001 • WFNS grades 4&5: 52% vs 27.9%, *P*<.001 • Fisher 3&4: 94% vs 86%, *P*=.01 • Clipped: 33% vs 22%, *P*=.03 • 6-month poor mRS (≥4): 42% vs 31%, *P*=.18 • ICU mortality: 15% vs 16%, *P*=.86 • Inpatient mortality: 25% vs 21%, *P*=.33 • 6-month mortality: 27% vs 23%, *P*=.34
Kodankandath TV, Farooq S, Wazni W, et al, 2017	Retrospective cohort	49	SAH admitted to NICU	To determine the effects of AED use limited to the presecurement period in aSAH	AED discontinued immediately after securement vs AED discontinued 3-7 days after securement • LEV: 16% vs 26%, no seizure at DC, no seizure at 3 months • PHT: 41% vs 16%, no seizure at DC, no seizure at 3 months
Suzuki H, Miura Y, Yasuda R, et al, 2022	Retrospective	121 (No AED=31; LEV=59; perampanel=31)	SAH ≥ 20 years	To assess if LEV and perampanel delayed neurovascular events s/p SAH	No AED vs LEV vs perampanel • DCI on CT %: 12.9 vs 6.8 vs 9.7, no difference • DCI on diffusion weighted imaging %: 19.4 vs 16.9 vs 3.2, *P* not documented • Good D/C mRS (0-2) %: 51.6 vs 49.2 vs 32.2, *P* not documented • Good 3-month mRS(0-2) %: 67.7 vs 67.8 vs 64.5, *P* not documented • Seizure %: 6.5 vs 18.6 vs 16.1, not significant
Liao W, Chien W, Chung C, et al, 2018	Retrospective	16228	SAH	To assess the association between valproic acid and the risk of acute respiratory failure (ARF) in patients with SAH	Positive VPA vs no VPA • Epilepsy: 0.96% vs 1.54%, *P*=.247 • ARF: 22.46% vs 27.74%, *P*=.014 • Cardiac dysfunction: 1.54% vs 1.15%, *P*=.338 • Renal dysfunction: 3.45% vs 2.59%, *P*=.210 • Neurologic dysfunction: 1.54% vs 2.02%, *P*=.328
Panczykowski D, Pease M, Zhao Y, et al, 2016	Retrospective matched analysis of prospective data	353	aSAH	To determine the effect of prophylactic AED on seizure incidence in SAH	Seizure incidence: • Prophylactic AED group (11%) vs no AED group (8%); *P*=.33 Propensity score matched analysis: • Likelihood of seizure was equivalent between groups; *P*=.49
Chou SH, Latorre JGS, Alpargu G, et al, 2015	Prospective observational	166	SAH > 18 years	To assess the safety and feasibility of early AED discontinuation in SAH	No AED vs positive AED • H&H 4-5: 11% vs 27%, *P*=.01 • Fisher 3-4: 79.2% vs 87.3%, *P*=.19 • Seizure incidence: 0% vs 1.1%, *P* not documented • Mortality: 2.7% vs 24%, *P*=.0028 • ICU ALOS: 10.3 vs 10.3, *P* not documented • ALOS: 16.6 vs 15.7, *P* not documented • Vasospasm: 30% vs 39%, *P*=.76 • D/C to home: 59% vs 29%, *P*=.002

Seizure Prophylaxis (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Fang T, Valdes E, Frontera JA, 2022	Meta-analysis	30 studies: 6 randomized, 9 prospective, 15 retrospective; 7,609 patients, 701 were SAH	TBI, SAH, ICH, supratentorial neurosurgery	To determine the effectiveness, optimal dosing, and adverse events associated with LEV	Most common dosing: 500 mg 2xD (48% of studies) Dosing range: 250 mg-1,500 mg 2xD (26% of studies) Weight based dosing or ≥ 1,000 mg 2xD (26% of studies) LEV vs no ASM: general seizure events per patient per year: 4.5% vs 3.8%, *P*=.23 LEV vs VPA: seizure events per patient per year: 8.1% vs 6.0%, *P*=.90 LEV vs comparator group: adverse events 8% vs 21%, *P* not documented Benefit: OR 0.34, 95% CI 0.20-0.58, *P*≤ .001 (sensitivity analysis excluding serious risk of bias studies)
Karamchandani RR, Fletcher JJ, Pandey AS, et al, 2014	Retrospective	259	aSAH	To compare the risk of poor outcomes and complications associated with LEV vs PHT	mRS > 3 • LEV use ≥ 72: 79%, *P*=.43 • PHT use ≥ 72: 43%, *P*=.33 • DCI: 40%, *P*=.62 • Seizure pre ASM: 13%, *P*=.20 • Seizure post ASM: 0.03%, *P*=.97 DCI • Clipped: 52%, *P*=.09 • LEV use ≥ 72: 86%, *P*=.46 • PHT use ≥ 72: 47%, *P*=.68 • Seizure pre ASM: 14%, *P*=.02 • Seizure post ASM: 2%, *P*=1.00 DCI and one ASM • Clipped: 48%, *P*=.03 • PHT use: 21%, *P*=.47 • Seizure pre ASM: 14%, *P*=.13 • Seizure post ASM: 3%, *P*=.78 Vasospasm • LEV use ≥ 72: 82%, *P*=.48 • PHT use ≥ 72: 49%, *P*=.95 • Seizure pre ASM: 6%, *P*=.15 • Seizure post ASM: 3%, *P*=.67 Delayed infarct • LEV use ≥ 72: 84%, *P*=.99 • PHT use ≥ 72: 50%, *P*=.66 • Seizure pre ASM: 5%, *P*=.01 • Seizure post ASM: 2%, *P*=.62 No stats directly comparing LEV vs PHT were reported.

Endovascular Therapy

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Yuan K, Li R, Zhao Wang K, et al, 2022	Retrospective	843 (clipped=414; coiled=429)	aSAH	To assess pneumonia risk factors in clipped vs coiled aSAH	Clip vs coil • DCI: 32.9% vs 21.0%, *P*<.001 • Intracranial infection: 20.25% vs 2.33%, P<.001 • Anemia: 41.79% vs 19.81%, *P*<.001 • Hypoproteinemia: 46.86% vs 21.91%, *P*<.001 • Post-op pneumonia: 34.54% vs 26.57%, *P*=.015 • DC mRS 3-6: 48.5% vs 34.97%, *P*<.001 • 90-day mRS 3-6: 22.22% vs 14.45%, *P*=.005
Luo M, Yang S, Ding G, et al, 2019	Meta-analysis	2,780	Clipped or coiled aSAH	To compare outcomes between surgical clipping and endovascular coiling	Coil vs clip • 1-year poor outcomes (mRS: 3-6): 23.3% vs 31.4%, OR 0.67, 95% CI 0.57–0.79, *P*<.00001 • 3–5-year poor outcomes: 23.1% vs 27.2%, OR 0.8, 95% CI 0.67–0.96, *P*=.02 • 1-year mortality: 8.13% vs 10.08%, OR 0.79, 95% CI 0.6–1.05, *P*=.10 • 1-year cerebral ischemic events clipped: OR 0.37, 95% CI 0.16–0.86, *P*=.02 • 1-year postprocedural rebleed: OR 1.15, 95% CI 0.75– 1.78, *P*=.52 • Technical failure (<100% occlusion) coil: OR=2.84, 95% CI 1.86–4.34, *P*<.00001
Galea JP, Dulhanty L, Patel HC, 2017	Retrospective	3,341 (clipped=741; coiled=2,600)	Clipped and coiled aSAH from the UK and Ireland SAH audit database	To determine predictors of unfavorable outcomes in aSAH	Favorable (GOS 4-5) vs unfavorable outcomes (GOS 1-3) • Median age: 54 vs 60, *P*<.001 • Mean time to treat (days): 3.2 vs 2.7, *P*<.03 • Pre-op rebleeding: 1.9% vs 10.7%, *P*<.001 • WFNS 1: 63.6% vs 17.5% , *P*<.001 • Hypertension: 32.7% vs 44.1%, *P*<.001 • Endovascular treatment: 79.1% vs 65.5%, *P*<.001 • CSF diversion: 207% vs 64.2%, *P*<.001 • DCI: 17.9% vs 31.2%, *P*<.001
Cai K, Ni Y, Zhang Y, et al, 2018	Retrospective	345	Coiled aSAH	To assess heart rate variability as a predictor of outcomes in SAH patients undergoing endovascular coiling	Unfavorable (GOS 1-3) vs favorable (GOS 4-5) outcomes • Admit H&H: *P*<.001 • Admit GCS: 8.9 ± 4.7 vs 13.7 ± 2.4, *P*<.001 • HR variability successive variation: 10.7 ± 3.9 vs 8.4 ± 3.9, *P*=.0009 • HR range: 30.5 ± 10.1 vs 22.7 ± 9.8, *P*<0.001 • Interval from ictus to coil (hours): 18.5 (8–62) vs 48.8 (22–107), *P*=0.001 • Post-op fever: *P*<.001
Ge XB, Yang QF, Liu ZB, 2020	Prospective	120	aSAH following endovascular treatment	To determine which factors predict poor outcomes after endovascular treatment	Good outcomes (mRS=0-2) vs poor outcomes (mRS=3-6) • 24 hour SD of SBP: 19.3 ± 5.5 vs 14.1 ± 4.8 mmHg, P<.001 • 24 hour SD of DBP: 9.5 ± 2.3 vs 9.9 ± 3.5 mmHg, *P*=.464 • Age ≥ 65 years: 26.7% vs 8.8%, *P*=.032 • H&H 3-4: 27.9% vs 55.9%, *P*=.006 • Fisher 3-4: 27.9% vs 58.8%, *P*=.002 • Intra-op complications: 25.6% vs 50.0, *P*=.01 • Post-op complications : 29.1% vs 52.9%, *P*=.014 • < 3 days p-ictus: 90.7% vs 88.2%, *P*=.688
Zhao B, Yang H, Zheng K, et al, 2017	Prospective observational, multicenter	136	Endovascular-treated aSAH	To develop prognostic models to predict poor outcomes following endovascular treatment	Favorable (mRS 1-3) vs unfavorable outcomes (mRS 4-6) • Age: 51.2 ± 11.3 vs 58.4 ± 11.2, *P*=.001 • Female: 38.9% vs 56.3%, *P*=.044 • Mean pre-op GCS ± SD: 8.3 ± 2.6 vs 6.6 ± 2.4, *P*<.001 • Preop WFNS Grade 5: 20.8% vs 57.8%, *P*<.001 • Mean Fisher ± SD: 2.8 ± 0.9 vs 3.4 ± 0.7, *P*<.001 • Mean aneurysm neck size ± SD: 2.7 ± 0.9 vs 3.2 ± 1.4, *P*=.026 • Cerebral infarct: 5.6% vs 17.2%, *P*=.039 • Symptomatic vasospasm: 5.6% vs 17.2%, *P*=.039

Enteral Nutrition

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Zhong DY, Li L, Ma RM, et al, 2021	Systematic review with meta-analysis	23 articles, 1,816 patients	RCT and case-control trials of stroke patients	To assess effects of probiotics combined with EN in stroke patients	Probiotics combined with EN was associated with: • Reduced hospital LOS (P<.05) • Time spent on bedrest (P<.05) • Improved nutritional status (P<.05) • Gastrointestinal symptoms such as esophageal reflux, bloating, constipation, diarrhea, gastric retention, and GI bleeding (P<.05)
Tuncay P, Arpaci F, Doganay M, et al, 2018	RCT	46 (aSAH=4)	Neurocritical care patients	To compare EN formula vs EN formula plus prebiotic content in neuro ICU patients	Enteral formula plus prebiotics group had less EN complications than the enteral formula without prebiotics (13% vs 56.5%, respectively; P=.002).
Saran D, Brody R, Stankorb S, et al, 2014	Retrospective observational study of data from prospective, observational studies and a cluster randomized control trial	1,495 patients (G-tube=1,407; small bowel tube=88)	Neurological critically ill patients	To investigate gastric vs small bowel tubes' effect on nutritional and clinical outcomes	In unadjusted analysis, EN adequacy was higher for the gastric group vs small bowel (60.2%±21.8% and 52.3%±22.0%, P=.001), but not in bivariate analysis (P=.428). Interruptions in EN delivery due to GI complications (abdominal distension, vomiting, high GRV) were 5 times more frequent in the gastric feeding (19.6%) group vs small bowel feeding group in bivariate analysis (P=.015).

Pain Management

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Mac Grory B, Vu L, Cutting S, et al, 2017	Prospective, observational study	20	SAH	To examine the clinical characteristics of H/A post SAH	Patients experiencing: • H/A peak within 1 second of onset: 65% • H/A in occipital location with neck stiffness: 55% • Stabbing quality: 35% • Presence of prior H/A: 50%, and associated meningismus: 80%
Gaastra B, Carmichael H, Galea I, et al, 2022	Retrospective	4,320 (control=3,456; aSAH=864)	aSAH and control cohort (UK national health databank)	To assess long-term headache frequency and duration, and migrainous H/A prevalence post aSAH	Migrainous H/A: 63.6% Median time for H/A to resolve from aSAH: 149 months (12.4 years) Headache frequency did not correspond to aSAH severity (z=0.249, *P*=.803), nor treatment (z=0.583, *P*=.560) Headache frequency decreased over time: RS=–0.71, *P*=.028 • 50% patients in the first year • 28% patients 10 years later
Ljubisavljevic S, Milosevic V, Stojanov A, et al, 2017	Retrospective	431	Nontraumatic SAH	To determine predictive factors of headache and H/A characteristics	Positive H/A vs no H/A Neck stiffness: OR 1.93, CI: 1.19-3.10, *P*<.007 Neck pain and stiffness: OR 0.34, CI 0.21-0.55, *P*<.001 Nausea and vomiting: 188 vs 115, *P* not documented Photophobia: 101 vs 202, *P* not documented
Glisic EK, Gardiner L, Josti L, et al, 2016	Retrospective	77	Nontraumatic SAH; H&H grades 1-3	To assess H/A post SAH	Severe HA: ≥ 2 days with maximum pain scores ≥ 8 Severe headache: • 73% overall: 73% • H&H 1: 20% • H&H 2: 62% • H&H 3: 18% Hijdra score and positive H/A: • Score 0-10: 27%, *P*=0.02 • Score 11-20: 45%, *P*=0.07 • Score 21-30: 29%, *P*=0.70 Opioid peak administration: days 3-7

Pain Management (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Klavansky D, Wanchoo S, Lin A, et al, 2021	Retrospective	138	SAH	To assess the opioid usage, related length of stay, and opioid consumption post discharge	Opioids prescribed during hospitalization: 90% of the time Most common opioid used: oxycodone, followed by tramadol, hydromorphone, fentanyl, and morphine Mean daily morphine equivalent dosage: 18.74 mg Factors associated with an increase in 14-day opioid use: • Steroid use: *P*=.0001 • Smoker vs nonsmoker: 353 mg vs 184 mg, *P*=.01 • aSAH compared to peri-mesencephalic SAH: 283 mg vs 195 mg, *P*=.004 • Coiling vs clipping: 320 mg vs 186 mg, *P*=.08. Predictors for opioids usage: • Steroid: *P*=.0001 • High H&H scale: *P*=.003 • mFisher grade: = .005 3-month follow-up: • Persistent H/A: 42% (n=48) • Still on opioids: 6% Higher opioid use in the first 14 days was associated with higher postdischarge headache rate: *P*=.002.
Morad AH, Tamargo RJ, Gottschalk A, 2016	Retrospective	46	SAH patients able to report pain	To assess pain characteristics and associated treatment of pain in aSAH	Reporting severe pain (7-10/10): 89% Reporting pain 10/10 during hospitalization: 63% Pain severity mean declined at a rate of 0.06 (0.04, 0.07) units/day (*P*<.001) Pain location: • Head: 76% • Other location: back, neck, limbs, and eyes Pain medications: • Acetaminophen with increasing daily doses: 100% of patients • Opioid administration: all but 3 patients Daily analgesic consumption over time: *P*=.57
Čomić H, Rinkel GJE, Vergouwen MDI, 2017	Retrospective	106	SAH with normal LOC and no focal deficits	To evaluate the course of SAH H/A during hospitalization	Acetaminophen use: 12% Acetaminophen plus opioid: 88% Patients reporting first numeric rating scale (NRS) score < 3: • 7% (n=7) within 12 hours • 14% (n=15) within 24 hours • 21% patients (n=22) with 48 hours after ictus Shortest time lapse between H/A report and NRS score of 0 was 10 hours. Patients reporting first NRS score of 0: • 1% patient (n=1) within 12 hours • 6% patients (n=6) within 24 hours • 8% patients (n=9) within 48 hours after ictus

Pain Management (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Hong CK, Joo JY, Kim YB, et al, 2015	Retrospective	217	aSAH	To assess post SAH headache and predisposing factors	Headache improvement (NRS score≤3) • Headache improved at discharge: 83.9%, *P*<.001 • After 1 month: 94.0% • After 3 months 95.9% • After 6 months: 98.2% • After 12 months: 99.1% Mean NRS scores: • Admit: 6.9 • D/C: 2.0 • 1 month: 1.0 • 3 months: 0.6 • 6 months: 0.5 • 12 months: 0.4 Predisposing factors for improving H/A on DC • No previous stroke (*P*<.001) • No previous H/A treated with medication (*P*=.008) Predisposing factors for improving H/A during follow-up • Endovascular treatment (*P*=.026) or no symptomatic vasospasm
Huckhagel T, Klinger R, Schmidt NO et al, 2020	Prospective with retrospective chart review	93 (burdensome H/A=38; nonburdensome H/A=55)	SAH	To assess long-term headache and health-related quality of life in good grade SAH	Burdensome H/A vs nonburdensome H/A: • Opioid and nonopioid use: 42% vs 7%, *P*<.01 • WFNS scores 1-2: 95% vs 75%, *P*=.03 • Discharge GOS 4-5: 100% vs75%, *P*=.08 • Highest ICP mean: 12.4/4.6 vs 11.0/3.6, *P*=.24 • SF-12 physical composite (quality of life): 40.3 ± 9.9 vs 49.6 ± 8.6, *P*<.01 • Depression Anxiety Stress Scales (DASS; anxiety subscore): 4.0 ± 4.4 vs 2.0 ± 2.6, *P*=.02 • DASS (stress subscore): 8.3 ± 6.1 vs 4.9 ± 4.9, *P*=.01 • Weariness: 65.8% vs 14.5%, *P*<.01 • Chronic H/A: 10.5% vs 12.7%, *P*>.99
Langley T, Hampton D, Wiggins A, et al, 2021	Retrospective	172 (H&H grade 1=49; grade 2=76; grade 3=47)	SAH H&H grades 1-3 able to self-report pain	To maximum pain scores and medication use	Mean daily pain score on day 5 (*P*=.023): • H&H grade 1: 5.57 • H&H grade 2: 7.0 • H&H grade 3: 7.07 Acetaminophen (mg) day 1 (*P*=.01): • H&H grade 1: 729.59 • H&H grade 2: 679.93 • H&H grade 3: 338.82 Acetaminophen (mg) day 10 (*P*=.021): • H&H grade 1: 437.75 • H&H grade 2: 718.42 • H&H grade 3: 912.76 Morphine equivalent received on day 1 (*P*=.009): • H&H grade 1 = 2.69 • H&H grade 2 = 5.52 • H%H grade 3 = 0.86 No statistically significant difference in pain score for patients who received intravenous magnesium, ketorolac, and acetaminophen/butalbital/caffeine.

Pain Management (continued)

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Maciel CB, Barlow B, Lucke-Wold B, et al, 2022	Cross-sectional survey	516	Members of five professional societies	To determine clinician management of SAH headache	Most common analgesics used to treat H/A: • Acetaminophen: 90% • Opioids: 66% • Corticosteroids: 28% • ASM: 28% Opioids were perceived as most effective followed by corticosteroids. Acetaminophen was most commonly prescribed at discharge, followed by opioids and antiseizure medications. Opioid alternatives: gabapentin, pregabalin, and magnesium ≥ 5-day courses of dexamethasone were perceived to be associated with adverse events, such as infection and hyperglycemia, and unfavorable outcomes at discharge. Alternative treatments used: CSF diversion, nerve-blocks, acupuncture, herbal medicine, massage, Reiki, and music therapy
Eisinger R, Sorrentino ZA, Lucke-Wold B, et al, 2022	Retrospective	91	aSAH	To assess the character and progression of aSAH headaches	Mild-moderate H/A vs moderate-severe H/A • Aneurysm size: 2.5 ± 0.3 vs 7.6 ± 0.2, *P*=.91 • Aneurysm location: *P*=.31 • Fisher: 2.9 ± 0.1 vs 2.8 ± 0.1, *P*=.92 • H&H: 2.4 ± 0.7 2.2 ± 0.1, *P*=.38 • Hydrocephalus: *P*=.74 • Ventriculostomy: *P*=1 • Vasospasm: *P*=1 • Mean Na^+: 139.3 ± 0.5 vs 137.5 ± 0.3, *P*<.01 • Opioid use: *P*<.001 • Acetaminophen: *P*=.68
Barnes PL, Haas H, Beck B, 2021	Retrospective pilot study	21	SAH with H/A	To determine the effect of osteopathic manipulative medicine (OMM) on H/A post SAH with no adverse effects	Changes in pain scores following OMM therapy (***P*** not documented): • Decreased by an average of 4 points after first treatment • Decreased by an average of 3 points after second treatment • Decreased by an average of 2.5 points after third treatment
Ganesan P, Manjini KJ, Bathala Vedagiri SC, 2022	RCT	70	Sternotomy patients	To assess the effect on postoperative sternotomy patients	Mean pain score pre-/post-therapy: • Music: 10.94 vs 1.94, *P*=.001 • Routine therapy: 10.23 vs 7.71, *P*=.001 Mean SBP pre-/post-therapy: • Music: 139.7 vs 122.6, *P*=.001 • Routine therapy: 134.8 vs 124.4, *P*=.001 Mean HR (beats/min) pre-/post-therapy: • Music: 100.4 vs 80, *P*=.001 • Routine therapy: 100.2 vs 86.57, *P*=.001 Mean respiration (breathes/min) pre-/post-therapy: • Music: 23.94 vs 16.09, *P*=.001 • Routine therapy: 22.86 vs 16.89, *P*=.001 O_2 saturation pre-/post-therapy: • Music: 95.54% vs 98.86%, *P*=.001 • Routine therapy: 96.1% vs 97.66%, *P*=.001

Thermodynamics

Reference	Study Design	Sample Size	Population	Study Aims	Findings
Suehiro E, Sadahiro H, Goto H, et al, 2016	Single-center observational cohort	62 (44 female)	aSAH with H&H 1-5, clipped or coiled, age > 18 years	To assess the relationships between temperature and treatment method, severity, and outcome	Three variables predicted a poor outcome • HH ≥ 4 (OR 19.8, 95% CI 1.5-255.5) • Coil (vs clip) (OR .05, 95% CI .004-.788) • Mean body temperature (OR 31.6 per 1°C, 95% CI 3.0-337.5)
Müller A, Lorenz A, Seifert B, et al, 2014	Case-controlled retrospective chart review	122 (aSAH=117; non-aSAH=5)	All patients admitted with aSAH to the NICU treated with ECC	To assess the risk for thromboembolic (TEE) events associated with ECC use	Without ECC vs with ECC • Mortality: 8% vs 33%, *P*<.001 • TEE: 5% vs 37%, *P*<.001 • Pulmonary embolism: 3% vs 12%, *P*=.039 • Mean H&H: 2.5 ± 1.2 vs 3.6 ± 1.2, *P*<.001 • Mean age: 59.3 ± 12.9 vs 53.1 ± 9.7 years, *P*=.004 Fever treatment vs hypothermia • Mortality: 29% vs 41%, no *P* value documented, stated as nonsignificant • Thrombosis: 52% vs 23%, *P*=.044
Khan I, Haymore J, Barnaba B, et al, 2018	Prospective matched with retrospective control	32 (ensoETM=8; control=24)	SAH or ICH with refractory fever	To assess shiver burden and pharmacotherapy cost differences between esophageal cooling device and other cooling devices	Control vs ensoETM • Temperature control measure initiation: 38.5 ± 0.5 vs 38.7 ± 0.4, *P*=.4 • Time to target (hours): 2.9 ± 3.2 vs 5.4 ± 3.7, *P*=.07 Fever burden (C·hours), mean ± SD: • >37.5: -0.15 ± 0.28 vs 0.05 ± 0.25, *P*=.09 • >38: -0.53 ± 0.31 vs -0.44 ± 0.25, *P*=.47 Shiver interventions/patient: • Total: 30 vs 14, *P*=.02 • Per day: 5 vs 3, *P*=.03