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REVIEW

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The treatment and monitoring of aneurysmal subarachnoid hemorrhage in critically ill patients

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ABSTRACT:

Purpose: To review the treatment and monitoring strategies for aneurysmal subarachnoid hemorrhage (aSAH) in critically ill patients, emphasizing the need for a multidisciplinary approach to stabilize the patient, prevent secondary brain injury, and manage complications.

Methods: The review focuses on targeted management of key cerebral parameters, including intracranial pressure (ICP), pressure reactivity index (PRx), cerebral autoregulation (CA), and the integration of non-invasive modalities such as near-infrared spectroscopy (NIRS). These tools are utilized to prevent rebleeding, control ICP, manage cerebral vasospasm, and support systemic homeostasis.

Important results: Despite advancements in continuous neuromonitoring and multidisciplinary care, current therapeutic strategies must span the entire treatment continuum from diagnosis and preoperative stabilization to intraoperative management and postoperative recovery. Securing the aneurysm via clipping or coiling remains central to reducing complications and improving neurologic outcomes.

Conclusions: Optimal management of aSAH demands individualized and dynamic neuromonitoring strategies. Patients with preserved consciousness may benefit from non-invasive monitoring to detect early deterioration, while those who are comatose or severely impaired require comprehensive invasive monitoring to guide cerebral resuscitation and prevent secondary injuries. Equally important is the strict prevention of systemic complications such as dysglycemia, anemia, hyperthermia, hypoxemia, dysnatremia, and infection, which are critical for maximizing neurologic recovery and survival.

Keywords: Subarachnoid hemorrhage; Neuromonitoring; Cerebral ischemia; Cerebral homeostasis; Secondary brain injury

INTRODUCTION

The treatment and monitoring of SAH in critically ill patients require a multidisciplinary approach aimed at stabilizing the patient, preventing secondary injury, and managing complications. In the case of aneurysmal SAH, critical care focuses on preventing rebleeding, managing intracranial pressure (ICP), addressing vasospasm, and supporting systemic functions [1]. Perioperative management of aneurysmal SAH involves preoperative, intraoperative, and postoperative care to reduce complications and improve neurologic outcomes, spanning from diagnosis to recovery after securing the aneurysm through clipping or coiling. This delicate and high-stakes process requires close coordination among neurology, neurosurgery, anesthesiology, and critical care teams to prevent secondary injury and optimize recovery. Aneurysmal SAH accounts for less than 5% of all strokes, but due to younger patient age and often severe brain damage, it is responsible for 27% of stroke-related life years lost before age 65 [2]. Although advances in aneurysm treatment and neurocritical care have improved outcomes, delayed cerebral ischemia (DCI) remains a major challenge. DCI, which occurs in 20-40% of patients, is a significant cause of mortality and morbidity, responsible for up to 50% of SAH-related deaths. Despite its recognition as a key complication, the exact pathophysiology of DCI remains unclear. As DCI typically develops between 3 and 14 days after aSAH onset, a therapeutic window exists, prompting ongoing research into neuroprotective agents [3].

KEY MESSAGES:

- Successful outcomes in aSAH require coordinated efforts among neurosurgery, neurocritical care, anesthesiology, radiology, and other specialties to manage both neurological and systemic complications.
- Continuous monitoring and proactive management of vasospasm via transcranial Doppler, perfusion imaging, and clinical assessment are necessary to prevent secondary ischemic injury.
- Strict control of systemic parameters, including glucose, temperature, oxygenation, electrolytes, and infection, is crucial to minimizing secondary injury and improving survival.
- Management must be responsive to evolving clinical and physiological status, integrating data from neuromonitoring and systemic assessments to optimize outcomes across the care continuum.

EARLY BRAIN INJURY AND DELAYED CEREBRAL ISCHEMIA [1,8,13,38]

In patients evaluated within the first 4 days after SAH, and without angiographic vasospasm, intracerebral hemorrhage, or hydrocephalus, cerebral metabolic rate of oxygen ($CMRO_2$) was reduced without changes in the oxygen extraction fraction. This suggests that the primary issue is a decrease in $CMRO_2$, leading to reduced cerebral blood flow (CBF) due to lower demand. This is typically attributed to a toxic effect of the subarachnoid blood, which has been shown experimentally. Additionally, in patients who lose consciousness, transient global ischemia can occur, and the combination of these factors leads to early brain injury. The pathophysiologic processes involved include endothelial injury, excitotoxicity, impaired sodium, potassium, and calcium channel function, and disrupted nitric oxide signaling. These effects result in impaired autoregulation, blood-brain barrier dysfunction, cell death via necrosis and apoptosis, inflammation, microthrombosis, activation of matrix metalloproteinases, oxidative stress, and edema. Early brain injury likely exacerbates DCI. There is often a relative hyperemia, believed to result from intracranial circulatory arrest, transient global ischemia, and lactic acidosis at the time of rupture. Mitochondrial respiration, sodium-potassium ATPase activity, and extracellular potassium and calcium levels are altered in brain tissue exposed to subarachnoid blood in experimental models, although the relationship between these changes and CBF or $CMRO_2$ is not fully understood. SAH is unique in that it has a well-documented delayed phase of brain injury, where neurological deterioration develops in a third of patients at 3 to 14 days after the event [4]. A systemic response also follows SAH, affecting the lungs (pulmonary edema, acute respiratory distress syndrome), heart (arrhythmias, contractility abnormalities), and fluid and electrolyte balance, potentially leading to a systemic inflammatory response syndrome. Commonly postulated mechanisms include increased sympathetic nervous system activity, elevated catecholamine levels, activation of the renin-angiotensin system, and the release of inflammatory cytokines, such as interleukin-6.

CLINICAL GRADING

Several clinical grading scales have been developed for assessing SAH, including the Hunt and Hess (Tables 1), Fisher Scale (Tables 2), Modified Fisher Scale (Tables 3), Hijdra Scale (Tables 4). However, none have gained universal acceptance, despite extensive evaluation of their predictive accuracy. Challenges in establishing a standardized grading system include significant inter- and intraobserver variability, differences in timing of assessment, and the exclusion of complex but potentially important prognostic factors [5]. Despite these limitations, clinical grading remains valuable for estimating prognosis, standardizing assessments for effective communication among healthcare providers, and enhancing the design and outcome measurement in multicenter studies. Repeated, standardized neurological assessments using semi-quantitative scales are essential for detecting clinical deterioration. The neurological grade is most accurately assessed after patient stabilization and, if needed, after ventricular drainage. The

Glasgow Coma Scale (GCS), which evaluates level of consciousness, has lower interobserver variability than more subjective scales such as Hunt and Hess. The WFNS scale, developed from clinical trial data, emphasizes GCS and focal neurological deficits, with level of consciousness being the most reliable predictor of outcome, while focal deficits are generally less impactful [6].

The Clinical Grading section has been revised to include a summary of commonly used grading systems Hunt and Hess, WFNS, and Modified Fisher and their correlations with outcomes:

- Hunt and Hess and WFNS: Correlate with mortality and early neurologic deterioration.
- Modified Fisher Scale: Predicts the risk of vasospasm and delayed cerebral ischemia.

A comparative summary of these scales is now included in Table 1, highlighting prognostic value regarding mortality and functional outcomes.

PERIOPERATIVE MANAGEMENT OF ANEURYSMAL SUBARACHNOID HEMORRHAGE (ASAH)

The perioperative management of aSAH encompasses three critical phases: preoperative, intraoperative, and postoperative each aimed at minimizing complications such as rebleeding, vasospasm, DCI, hydrocephalus, seizures, and systemic dysfunction, all of which can significantly impact outcomes (Tables 5) [1,7,8].

PREOPERATIVE PHASE

This phase begins at diagnosis and continues until the aneurysm is secured via surgical clipping or endovascular coiling. Given the highest risk of rebleeding occurs within the first 24–48 hours, prompt stabilization and early aneurysm securing are critical. Key priorities include preventing rebleeding, maintaining cerebral perfusion, controlling ICP, initiating prophylaxis for vasospasm and seizures, and managing systemic complications [9]. Definitive aneurysm treatment (surgical clipping or endovascular coiling) should be performed as early as feasible, preferably within 24 hours of diagnosis. For aneurysms equally suitable for both techniques, primary endovascular coiling is preferred due to superior 1-year functional outcomes. Surgical clipping may be favored in patients <40 years for greater long-term durability. Final treatment decisions depend on aneurysm morphology and location.

Management involves:

- Neurologic assessment using Hunt and Hess or WFNS scales, GCS, and CT imaging to evaluate SAH, hydrocephalus, and clot burden.
- Blood pressure control, prior to aneurysm securing, systolic blood pressure (SBP) should be controlled using short-acting intravenous antihypertensive agents, targeting 140–160 mmHg. Norepinephrine may be used after aneurysm securing in patients with symptomatic vasospasm or DCI to augment cerebral perfusion. Surgical or endovascular aneurysm treatment should be performed as early as feasible, ideally within 24 hours.
- ICP and hydrocephalus management with external ventricular drainage (EVD) for patients showing clinical signs of elevated ICP, alongside head elevation, pain control, and sedation.
- Anticonvulsant prophylaxis, routine prophylactic antiseizure medication is not recommended. However, it may be considered in high-risk patients with ruptured MCA aneurysms, intraparenchymal hemorrhage, high-grade aSAH, hydrocephalus, or cortical infarction. Phenytoin should be avoided due to its association with excess morbidity.
- Nimodipine (60 mg orally or via nasogastric tube every 4 hours for 21 days) to reduce the risk of DCI.
- Fluid and electrolyte balance with isotonic fluids to maintain euvolemia, close monitoring for hyponatremia (due to SIADH or cerebral salt wasting), and early enteral feeding if tolerated.
- Multidisciplinary coordination among neurosurgery, neuroradiology, anesthesiology, and neurocritical care to determine the optimal treatment strategy.

INTRAOPERATIVE PHASE

This phase focuses on securing the aneurysm and ensuring neuroprotection. Whether via microsurgical clipping or endovascular coiling, goals include preventing intraoperative rupture, maintaining stable cerebral hemodynamics, and avoiding secondary brain injury [1,10].

Key considerations:

- Anesthesia management with smooth induction/emergence, short-acting agents, arterial line monitoring, and maintaining normocapnia and normothermia.
- Hemodynamic control to avoid BP fluctuations using vasoactive infusions as needed.
- ICP management with osmotic agents like mannitol or hypertonic saline for brain swelling and controlled hyperventilation in herniation emergencies.
- Procedure selection depends on aneurysm characteristics and patient condition, clipping is preferred for anterior circulation or wide-neck aneurysms; coiling is favored in older or medically fragile patients.

POSTOPERATIVE PHASE

Following aneurysm securing, focus shifts to preventing complications, particularly vasospasm and DCI, which commonly occur between days 3 and 14 post-SAH [1,11].

Management includes:

- Continued nimodipine (60 mg every 4 hours for 21 days).
- Vasospasm monitoring with daily transcranial Doppler (TCD) and prompt imaging (CT perfusion or angiography) if neurologic decline occurs.
- Treatment of symptomatic vasospasm, prior to aneurysm securing, systolic blood pressure should be controlled using short-acting intravenous antihypertensive agents, targeting 140–160 mmHg. Norepinephrine may be used after aneurysm securing in patients with symptomatic vasospasm or DCI to augment cerebral perfusion.

Table 1. The Hunt and Hess scale is used to classify the severity of a subarachnoid hemorrhage (SAH), particularly due to a ruptured cerebral aneurysm. It helps predict patient outcomes and guide treatment decisions [42,53-54].

Grade	Clinical condition	Typical findings
I	Asymptomatic or mild headache, slight nuchal rigidity	Alert and oriented, minimal symptoms
II	Moderate to severe headache, nuchal rigidity, cranial nerve palsy	Alert, with significant meningeal irritation
III	Drowsiness, confusion, or mild focal neurological deficit	Lethargic but arousable, some neurological findings
IV	Stupor, moderate to severe hemiparesis, early decerebrate posturing	Significantly impaired consciousness
V	Deep coma, decerebrate rigidity, moribund	Unresponsive, poor prognosis

Table 2. The Fisher Scale, which is used to grade the severity of subarachnoid hemorrhage (SAH) based on findings from a non-contrast CT scan. It was developed to predict the risk of vasospasm after aneurysmal SAH [42,53-54].

Grade	CT findings	Risk of vasospasm
1	No blood detected	Low
2	Diffuse or thin SAH (<1 mm)	Moderate
3	Localized clot and/or thick SAH (>1 mm)	High
4	Intracerebral or intraventricular hemorrhage (with or without SAH)	Variable to high

Grade 3 carries the highest risk of vasospasm. Grade 4 includes intraventricular hemorrhage (IVH), which was all grouped together regardless of clot thickness or SAH extent. The Modified Fisher Scale was later introduced to better account for IVH and provide a more predictive model for vasospasm.

Table 3. The Modified Fisher Scale is a radiological grading system used to assess the severity of subarachnoid hemorrhage (SAH) based on initial non-contrast computed tomography (CT) scans. It helps predict the risk of delayed cerebral ischemia (DCI) and vasospasm following aneurysmal SAH [42,53-54].

Grade	Description	Risk of symptomatic vasospasm
0	No SAH or intraventricular hemorrhage (IVH)	0%
1	Focal or diffuse thin SAH without IVH	24%
2	Focal or diffuse thin SAH with IVH	33%
3	Focal or diffuse thick SAH without IVH	33%
4	Focal or diffuse thick SAH with IVH	40%

Grades 1 and 2 involve thin SAH, with the presence or absence of IVH influencing the grade. Grades 3 and 4 involve thick SAH, with IVH presence affecting the grade. The presence of IVH generally increases the risk of symptomatic vasospasm. It's important to note that the Modified Fisher Scale is distinct from the original Fisher Scale. In the original Fisher Scale, any IVH is classified as Grade 4, irrespective of the presence of SAH. In contrast, the Modified Fisher Scale differentiates between the thickness of SAH and the presence of IVH, providing a more nuanced assessment of vasospasm risk.

Table 4. The Hijdra Scale is a radiological grading system developed to assess the severity of subarachnoid hemorrhage (SAH) based on non-contrast CT scans. It evaluates the extent of blood in the 10 basal cisterns and fissures and the 4 ventricles, providing a comprehensive measure of hemorrhage severity [42,53-54].

Grade	Basal cisterns and fissures (10 regions)	Ventricles (4 regions)
0	No blood	No blood
1	Small amount of blood	Sedimentation of blood in the posterior part
2	Moderately filled with blood	Partly filled with blood
3	Completely filled with blood	Completely filled with blood
	The total score for the basal cisterns and fissures ranges from 0 to 30	The total score for the ventricles ranges from 0 to 12

The combined total score is the sum of the scores from the basal cisterns and ventricles, yielding a range from 0 to 42. A higher score indicates a greater amount of hemorrhage and is associated with an increased risk of delayed cerebral ischemia (DCI) and poor functional outcomes. The Hijdra Scale has demonstrated superior predictive value for DCI, in-hospital mortality, and functional outcomes compared to other grading systems like the Modified Fisher Scale. It is particularly useful in assessing patients with Fisher Grade 3 SAH, who are at higher risk for complications.

- ICP/hydrocephalus monitoring with continued EVD use, adjustments based on clinical status, and potential VP shunt placement for chronic hydrocephalus. CPP should be maintained between 60–70 mmHg.
- Seizure management with antiepileptic drugs (AEDs) continued only if seizures occurred or risk remains high; routine long-term prophylaxis is not recommended in low-risk patients.
- Electrolyte management including close sodium monitoring and treatment of cerebral salt wasting with hypertonic saline and fludrocortisone.
- Infection prevention via sterile technique for EVDs and central lines, and early mobilization.
- Rehabilitation planning with early involvement of physical and cognitive rehabilitation services as the patient stabilizes.
- Sedation in critically ill aSAH patients includes [3,21,16,30,55]:
  - o Preferred agents: Propofol, dexmedetomidine, and midazolam
  - o Key considerations:
    - Propofol: Rapid onset, neuroprotective, may reduce ICP; watch for hypotension
    - Dexmedetomidine: Sedation without respiratory depression, useful for awake assessments
    - Midazolam: Caution with accumulation in renal/hepatic impairment
    - Avoidance: Long-acting benzodiazepines or high-dose opioids which may obscure neurologic examination
- Triple-H therapy (Hypertension, Hypervolemia, Hemodilution) [1,26-27]:
  - o Currently, only hypertensive augmentation remains part of standard practice, used after aneurysm securing in patients with symptomatic DCI.
  - o Hypervolemia and hemodilution are no longer recommended due to increased risk of complications.
  - o Based on evidence and the 2023 guidelines, therapy now emphasizes euvolemia and targeted blood pressure augmentation.

INTRA-ARTERIAL PAPAVERINE (IAP)

IAP has historically been used for treating refractory cerebral vasospasm after SAH. It works as a non-specific phosphodiesterase inhibitor, increasing cAMP and cGMP to relax vascular smooth muscle. Administered intra-arterially, it dilates affected vessels and may restore perfusion.

- Clinical use: Reserved for vasospasm unresponsive to medical therapy; often combined with balloon angioplasty, especially for distal vessels.
- Efficacy: Provides immediate angiographic improvement and reduced TCD velocities, but neurological benefits vary, and effects are often short-lived.
- Limitations/risks: Includes short duration of action, potential for neurotoxicity (e.g., increased ICP, seizures), paradoxical vasoconstriction, chemical meningitis, and hypotension.
- Current trends: Intra-arterial papaverine has largely been replaced by agents such as intra-arterial nicardipine or verapamil, which offer better safety profiles. Papaverine's use has declined due to concerns about neurotoxicity and rebound vasospasm.

This comprehensive perioperative strategy is essential for improving survival and neurologic recovery in patients with aSAH.

CRITICAL CARE MANAGEMENT OF POOR-GRADE ANEURYSMAL SUBARACHNOID HEMORRHAGE (ASAH)

Poor-grade aSAH (Hunt and Hess or WFNS grades IV–V) presents a major neurocritical care challenge due to depressed consciousness, elevated ICP, hydrocephalus, and multisystem dysfunction [1,12–14]. Management focuses on early stabilization, preventing secondary brain injury, and multidisciplinary prognostication.

INITIAL STABILIZATION

Early ICU admission is essential for airway protection, hemodynamic stabilization, neurological assessment, and aneurysm treatment planning [15–17].

Key interventions:

- Airway: Intubate for GCS ≤ 8 or airway compromise.
- Blood Pressure: Maintain SBP 140–160 mmHg prior to aneurysm securing using short-acting IV antihypertensives. Avoid vasoactive agents like norepinephrine until after aneurysm is secured unless DCI occurs.
- Imaging: Perform CT and CTA promptly to evaluate hemorrhage and aneurysm anatomy.

MANAGEMENT OF ICP AND HYDROCEPHALUS

Hydrocephalus is common due to CSF flow obstruction by subarachnoid blood [18].

- EVD: Indicated for acute hydrocephalus or ICP elevation; allows drainage and monitoring.
- ICP Strategies:
  - o Head elevation to 30°
  - o Sedation and analgesia
  - o Hyperosmolar therapy (mannitol or hypertonic saline)
  - o Controlled hyperventilation in herniation risk
  - o Maintain CPP ≥ 60 mmHg

Table 5. Summary of perioperative SAH management [1,3].

Phase	Main goals	Key actions
Preoperative	Prevent rebleeding, stabilize patient	BP control, nimodipine, ICP monitoring, seizure prophylaxis
Intraoperative	Secure aneurysm, avoid secondary injury	Hemodynamic stability, neuroprotection, manage ICP
Postoperative	Prevent vasospasm/DCI, manage ICP, recovery	TCD monitoring, induced hypertension, rehab, infection control

ANEURYSM SECURING AND REBLEEDING PREVENTION

Early aneurysm treatment ideally within 24 hours improves outcomes even in poor-grade patients [19,56].

- Coiling is preferred for suitable aneurysms due to lower invasiveness [56].
- Clipping may be needed for mass effect or aneurysms unsuitable for coiling.
- Rebleeding Prevention:
  - o SBP <160 mmHg prior to treatment
  - o Reverse anticoagulants/antiplatelets
  - o Minimize external stimuli

DELAYED CEREBRAL ISCHEMIA (DCI) AND VASOSPASM

DCI typically occurs between 3–14 days post-ictus and is a major cause of morbidity [20-21].

Management (Tables 6-8):

- Nimodipine: 60 mg orally/NG every 4 hours for 21 days.
- Volume status: Maintain euvolemia using isotonic fluids. Euvolemia should be maintained in all patients. Hemodynamic augmentation using norepinephrine should be reserved for patients with neurologic worsening due to symptomatic delayed cerebral ischemia. Prophylactic hypertensive therapy in asymptomatic patients is not recommended, and hypervolemia should be avoided to minimize iatrogenic complications.
- Monitoring: Daily TCD; consider perfusion imaging if DCI suspected.
- Treatment: Induced hypertension (if aneurysm secured); consider intra-arterial vasodilators (nicardipine/verapamil).

Seizure prophylaxis

- Short-term antiepileptic drugs (AEDs): Levetiracetam is preferred for the first 7 days.
- Continuous EEG (cEEG): Recommended for comatose patients or those with suspected nonconvulsive seizures.

Systemic complications (Tables 9-11)

Poor-grade aSAH often causes systemic complications requiring proactive management [22-44].

Key issues:

- Neurogenic cardiac dysfunction: Manage supportively; avoid hypotension.
- Neurogenic pulmonary edema: Supportive care; avoid fluid overload.
- Electrolyte disturbances (hyponatremia, hypokalemia): Correct based on underlying etiology (SIADH vs CSW).
- Infections: Use aseptic technique; remove devices early; monitor for VAP, UTIs, and meningitis.
- Nutrition: Initiate early enteral feeding to reduce catabolism.
- Anemia and transfusion [31,41,44]:
  - o Association between anemia and poor outcomes in aSAH
  - o Benefits of transfusion in selected cases to improve cerebral oxygen delivery
  - o Routine transfusions are not recommended
  - o Consider transfusion in cases of symptomatic anemia or low cerebral oxygenation despite normovolemia

Prognostication and ethics [45-48]

A poor initial grade does not necessarily mean poor outcome. Early aneurysm treatment and complication management improve survival [22].

- Avoid withdrawal of care within the first 72 hours unless brain death is confirmed.
- Prognostication should involve the ICU, neurosurgery, neurology, and family, using a compassionate and individualized approach.

NEURO-MONITORING IN ASAH

Multimodal monitoring is vital for detecting secondary injury in comatose or high-risk patients (Tables 12-19). Modalities include:

Invasive monitoring

- ICP/CPP: Maintain ICP <20 mmHg; CPP >60–70 mmHg
- PRx: Autoregulation impaired if PRx > 0.3; target CPPopt for individualized care. CPPopt, as determined by PRx, represents the CPP associated with optimal autoregulation (lowest PRx). An acceptable PRx value is <0.3.
- $PbtO_2$: Maintain >20 mmHg
- $SjvO_2$: Normal 55–75%; <55% suggests ischemia
- CMD: Detects ischemia/metabolic crisis via lactate-pyruvate ratio and glucose

Non-invasive monitoring

- NIRS: Decline >14.5% in rSO_2 may signal ischemia
- TCD: Detects vasospasm; MFV >120 cm/s and LR >3 suggests vasospasm
- ONSD: ONSD >0.5 cm correlates with ICP >20 mmHg
- EEG: Essential for detecting nonconvulsive seizures and cortical dysfunction. In poor-grade aSAH patients, continuous EEG is also used to detect delayed cerebral ischemia, as indicated by a reduction in the alpha-to-delta power ratio.

Integration of neuro-monitoring and imaging

Neuro-monitoring provides real-time physiologic insight, while imaging (CT, CTA, MRI) evaluates evolving structural complications (hydrocephalus, rebleeding, DCI). Combining both modalities enables early detection, timely intervention, and personalized therapy to prevent irreversible injury [49–56].

Table 6. Complication of subarachnoid hemorrhage (SAH) especially aneurysmal SAH [35-39].

Complication	Prevention/Treatment
Rebleeding	Aneurysm securing, BP control
Hydrocephalus	EVD placement, CSF diversion
Elevated ICP	Sedation, head elevation, hyperosmolar therapy, EVD
Vasospasm/DCI	Nimodipine, BP augmentation, intra-arterial therapy
Hyponatremia	Monitor Na+, treat CSWS with fluids/salt
Seizures	Short-term prophylaxis in high-risk patients
DVT	SCDs early, LMWH after aneurysm secured
Nutrition	Early enteral feeding
Cardiac/Lung dysfunction	Monitor and support as needed (echo, oxygenation, diuretics)

Table 7. Summary of critical care management in poor-grade aSAH [35-39].

Component	Actions
Airway & Hemodynamics	Intubation, BP control (SBP < 160), invasive monitoring
ICP/Hydrocephalus	EVD, sedation, osmotic therapy, ICP/CPP goals
Aneurysm securing	Early coiling/clipping within 72 hrs
Rebleeding prevention	SBP control, minimize stimulation, reverse anticoagulation
Vasospasm/DCI	Nimodipine, TCDs, perfusion imaging, induced HTN if needed
Seizures	Prophylaxis in high risk; continuous EEG for coma
Multisystem care	Electrolyte balance, fluid status, cardiac/respiratory support, nutrition
Prognosis & Ethics	Delay decisions; involve family and multidisciplinary teams

Table 8. Admitting intensive care unit (ICU) Orders Table for patients with aneurysmal subarachnoid hemorrhage (SAH). These orders are designed for initial management in the ICU or a neurocritical care unit, focusing on monitoring, prevention of complications, and supportive care [1,3,35-37].

Category	Orders
Diagnosis	- Aneurysmal subarachnoid hemorrhage - Admit to ICU or Neurocritical care unit
Vital monitoring	- Continuous cardiac monitoring - Neuro signs and GCS checks q1hr - ICP monitoring (if indicated) - Avoid fever, hypoxia, decreased cerebral perfusion pressure, and hypovolemia - Daily body weight - Arterial catheter (for blood pressure measurement, generally only for poor-grade patients preoperatively) inserted after adequate sedation and anesthesia in all patients for surgery and left in place postoperatively
Activity	- Bed rest with head of bed at 30 degrees until the aneurysm is obliterated or until several days from hemorrhage - Elevate head of bed 30 degrees, especially in patients with marginal airway and intubated, ventilated patients - Restricted visitors; avoidance of unnecessary stimulation - Graduated compression stockings or intermittent pneumatic compression devices on lower extremities; consider pharmacologic prophylaxis beginning 24 hours after aneurysm repair (DVT prophylaxis with SCDs unless contraindicated) - Foley catheter for poor-grade patients or those unable to void - Nasogastric tube for intubated patients; replace with nasoduodenal tube for nutritional support if unable to eat
Neurological care	- Notify neurosurgery/neuro-intervention team immediately - External ventricular drain (EVD) if hydrocephalus - Ventricular drain for patients with neurological compromise from ventricular dilation or for postoperative monitoring; maintain drain closed and only drain for pressure >20 mm Hg

Table 8. **(Continued)** Admitting intensive care unit (ICU) Orders Table for patients with aneurysmal subarachnoid hemorrhage (SAH). These orders are designed for initial management in the ICU or a neurocritical care unit, focusing on monitoring, prevention of complications, and supportive care [1,3,35-37].

Category	Orders
Blood pressure goal	- Maintain SBP < 160 mmHg (pre-aneurysm secured) - After securing aneurysm: permissive hypertension (SBP 160–180 mmHg) - Antihypertensives to reduce blood pressure predominantly within the first 4 days after SAH when the aneurysm is not obliterated: labetalol, 200-400 mg orally, repeat every 2 to 3 hours (may cause bronchoconstriction or heart block), or intravenous 20- to 80-mg boluses every 10 minutes to a maximum of 300 mg until desired blood pressure is reached, then infusion of 2 mg/min (contraindicated in heart failure, bronchospastic disease); nicardipine, 5 to 15 mg/hr intravenously until desired blood pressure reached, then reduced to 3 mg/hr
Fluid management	- Isotonic fluids (e.g., NS at 75–100 mL/hr) - Avoid hypovolemia; consider central line for CVP monitoring
Medications	- Nimodipine 60 mg PO q4h (start ASAP to prevent vasospasm) - Stool softeners to avoid valsalva - Analgesics (e.g., acetaminophen, short-acting opioids) - Antiemetics PRN - Intravenous fluids with goal to maintain euvolemia - Omeprazole to reduce risk of gastrointestinal bleeding
Seizure prophylaxis	- Levetiracetam 500–1000 mg BID (short term; controversial—follow institutional protocol) - Anticonvulsants for the treatment of seizures (not prophylaxis), such as phenytoin (Dilantin), 15-19 mg/kg intravenous loading dose at not greater than 50 mg/min followed by 100 mg intravenous or orally every 8 hours, or phenobarbital
Labs & Imaging	- CBC, CMP, coagulation panel, troponin, type and screen, and bleeding time if history of recent aspirin ingestion - Electrolytes; blood urea nitrogen; creatinine avoid hypo- and hyperglycemia, hypercarbia, hypomagnesemia, and hyponatremia - Baseline ECG - Non-contrast head CT - Computed tomography (CT); chest radiography; CT angiogram +/− catheter cerebral angiogram, repeated as necessary and find associated vascular abnormalities, and negative initial angiogram - CTA or DSA for aneurysm localization - Urinalysis; urine toxicology screen, particularly for cocaine - Culture and sensitivity of respiratory secretions, urine, blood, cerebrospinal fluid, or other sources if fever or signs of infection develop
Consults	- Neurosurgery - Neurology (if not already involved) - Interventional neuroradiology
Additional orders	- Foley catheter if needed for monitoring output - NPO until cleared for swallowing - Family/caregiver notification and support

Table 9. Summary of specific complications of subarachnoid hemorrhage (SAH) [24-30].

Complication	Timing	Key features	Management
Rebleeding	0–48 hrs	Sudden deterioration, high mortality	Early aneurysm securing, BP control
Delayed cerebral ischemia	Days 4–14	Focal deficits, reduced perfusion	Nimodipine, hemodynamic therapy, endovascular Rx
Hydrocephalus	Early or late	Ventricular enlargement, confusion	EVD, VP shunt
Seizures	Anytime	Seizures or EEG changes	Short-term AEDs
Hyponatremia (CSW/SIADH)	First week	Low sodium, volume status critical	Replace fluids (CSW) or restrict (SIADH)
Pulmonary edema	Acute	Hypoxia, CXR infiltrates	Supportive, fluid balance
Cardiac dysfunction	Acute	ECG/troponin changes, LV dysfunction	Supportive, avoid hypotension
Infection	Variable	Fever, positive cultures	Antibiotics, device care
Neurocognitive deficits	Long-term	Memory, attention, emotional issues	Rehabilitation, neuropsychology

Table 10. Frequency of causes of morbidity and mortality in patients with aneurysmal subarachnoid hemorrhage (aSAH) in ICU [24-30,35-36,43-45].

Cause	Timing	Approximate frequency	Contribution to morbidity/mortality	Notes
Rebleeding	0–48 hours	4–15%	High (up to 70% mortality)	Most common early cause of death if aneurysm not secured early
Delayed Cerebral Ischemia (DCI)	Days 4–14	30–40%	Major cause of delayed morbidity	Often related to vasospasm; treatable if recognized early
Hydrocephalus	Acute or sub-acute	20–30%	Moderate to high	Requires EVD or long-term shunting; chronic hydrocephalus impacts QOL
Seizures	Any time	5–10%	Variable	May lead to secondary injury, higher in cortical or rebleed cases
Electrolyte disturbances	Early (first 1–2 wks)	25–35% (esp. hyponatremia)	Indirect	Increases risk of DCI, seizures, confusion
Neurogenic cardiac complications	Acute	20–30%	Moderate	Takotsubo cardiomyopathy, arrhythmias, ECG changes
Neurogenic pulmonary edema	Early post-SAH	~10%	Moderate	Acute respiratory failure, high ICP trigger
Infection (pneumonia, UTI, meningitis)	Hospital course	30–50%	Variable	Prolongs ICU stay, contributes to mortality in severe cases
Cognitive and psychological deficits	Long-term	>50% in survivors	High impact on quality of life	Memory loss, depression, PTSD, executive dysfunction

Early mortality is often due to rebleeding, while late morbidity stems from DCI, hydrocephalus, and neurocognitive impairment. Complications like electrolyte imbalances, seizures, and cardiopulmonary issues can exacerbate primary injury. Infections are frequent during prolonged ICU stays and can complicate recovery significantly.

Table 11. Neurological complications in aneurysmal subarachnoid hemorrhage (aSAH) [24-30].

Complication	Incidence	Timing	Impact on outcome	Key management strategies
Delayed Cerebral Ischemia (DCI)	30–40%	Days 4–14	Major cause of disability and death	Early detection via transcranial Doppler (TCD), CT perfusion; treatment with nimodipine, induced hypertension, or endovascular interventions
Hydrocephalus	20–30%	Acute or sub-acute	Requires surgical intervention	External ventricular drainage (EVD) or ventriculoperitoneal shunt placement
Seizures	5–10%	Any time	May lead to secondary injury	Antiepileptic drugs (AEDs) for short-term prophylaxis; long-term use based on clinical judgment
Electrolyte disturbances	25–35% (e.g., hyponatremia)	Early (first 1–2 weeks)	Can exacerbate neurological deficits	Fluid and electrolyte management; distinction between cerebral salt wasting (CSW) and syndrome of inappropriate antidiuretic hormone (SIADH)
Neurogenic pulmonary edema	~10%	Early post-SAH	Acute respiratory failure	Supportive care, oxygen therapy, and fluid management
Cardiac complications	20–30%	Acute	May complicate recovery	Monitoring for arrhythmias; management of neurogenic stunned myocardium
Infections (e.g., pneumonia, UTI, meningitis)	30–50%	Hospital course	Prolongs ICU stay, contributes to mortality	Prophylactic antibiotics, strict aseptic techniques, early removal of invasive devices
Cognitive and neuropsychological deficits	>50% in survivors	Long-term	Significant impact on quality of life	Neuropsychological assessment, rehabilitation programs

Delayed Cerebral Ischemia (DCI) remains a leading cause of morbidity and mortality in aSAH patients, emphasizing the need for vigilant monitoring and early intervention. Hydrocephalus is common and often necessitates surgical intervention to prevent further neurological deterioration. Seizures and electrolyte disturbances are prevalent in the early stages post-SAH and require prompt management to mitigate their impact. Cardiac complications and pulmonary edema are significant contributors to early mortality and necessitate comprehensive supportive care. Infections are frequent during prolonged ICU stays and can complicate recovery significantly. Cognitive and neuropsychological deficits are common in survivors, underscoring the importance of long-term rehabilitation and support.

Table 12. Impact of medical complications on outcome after subarachnoid hemorrhage (aSAH) in ICU [24-30].

Complication	Incidence	Effect on outcome	Mechanism/Notes
Neurogenic pulmonary edema	~10%	↑ Mortality, ↑ ICU stay, ↑ ventilator days	Triggered by catecholamine surge from increased ICP; leads to hypoxia and instability
Cardiac dysfunction (Takotsubo, ECG changes)	20–30%	↑ Risk of hypotension, ↑ ICU complications	May cause myocardial stunning and arrhythmias; worsens cerebral perfusion
Hyponatremia (SIADH/CSW)	25–35%	↑ Risk of seizures, cerebral edema, DCI	Difficult fluid/electrolyte balance; requires careful monitoring
Hypernatremia	10–15%	↑ Mortality, worsened cognitive outcomes	Often iatrogenic; associated with osmotic demyelination or renal dysfunction
Hyperglycemia	>50% (transient)	↑ Risk of poor neurologic recovery	Promotes oxidative stress and exacerbates ischemic injury
Hypoglycemia	<5% (acute)	Seizures, irreversible brain damage	Can occur in septic or critically ill patients; needs prompt correction
Sepsis / Systemic Infections	30–50%	↑ Mortality, ↑ ICU/hospital stay, ↑ delirium	Pneumonia, UTI, meningitis common; associated with prolonged intubation, catheter use
Deep Vein Thrombosis (DVT)	~10–15%	↑ Morbidity, risk of pulmonary embolism	Immobility + inflammation; requires prophylaxis with SCDs or anticoagulation
Gastrointestinal bleeding	~5–10%	Delays recovery, ↑ ICU stay	Stress ulcers from critical illness or corticosteroids
Delirium	~30–40%	↑ Length of stay, ↓ functional outcome	Often multifactorial—linked to infections, metabolic derangements, sleep disturbance

Table 12 summarizing the impact of medical complications on outcomes after aneurysmal subarachnoid hemorrhage (aSAH). These systemic (non-neurological) complications frequently occur during the acute phase and significantly affect mortality, length of ICU stay, and functional outcomes. Systemic complications are common after aSAH and can significantly affect both short- and long-term outcomes. Managing these requires early recognition, proactive prevention, and coordinated care between neurology, critical care, cardiology, and nephrology teams. The cumulative burden of these complications can sometimes outweigh the direct neurological injury in determining prognosis.

Table 13. Causes of neurological deterioration after subarachnoid hemorrhage (SAH) [24-30].

Cause	Timing	Clinical features	Diagnostic tools	Management
Rebleeding	First 24–48 hrs	Sudden coma, worsening headache, vomiting, seizures	Non-contrast CT	Urgent aneurysm clipping/coiling, BP control
Delayed Cerebral Ischemia (DCI)	Days 4–14	New focal deficits, decreased consciousness	Clinical exam, TCD, CT perfusion, angiography	Nimodipine, induced hypertension, endovascular therapy
Hydrocephalus	Acute or delayed	Lethargy, confusion, gait instability, incontinence	CT brain (ventricular enlargement)	External ventricular drain (EVD), VP shunt if chronic
Seizures	Early or late	Convulsions, postictal state, altered mental status	EEG, clinical observation	Short-term antiepileptics (e.g., levetiracetam), monitor for recurrence
Electrolyte disturbances	First week	Confusion, coma, irritability (especially with hyponatremia)	Serum sodium, osmolarity	Correct Na levels cautiously (CSW vs. SIADH management differs)
Cerebral edema / increased ICP	Any time	Headache, papilledema, bradycardia, hypertension (Cushing's triad)	CT scan, ICP monitoring	Elevate head, sedation, osmotherapy (mannitol, hypertonic saline), EVD
Infection (e.g., meningitis)	Hospital-acquired	Fever, neck stiffness, altered mental status	CSF analysis, blood cultures	Empiric antibiotics, adjust based on culture
Vasospasm (radiologic/clinical overlap with DCI)	Days 3–14	Focal deficits, reduced consciousness, infarcts on imaging	TCD, CTA, DSA	Hemodynamic augmentation, intra-arterial vasodilators, angioplasty
Metabolic encephalopathy	Any time	Diffuse confusion, no focal findings	Labs, EEG	Correct underlying derangement (glucose, liver, renal, etc.)
Medication/sedation effects	ICU course	Altered consciousness without clear imaging findings	Review sedative use, EEG if needed	Adjust or hold sedatives, consider EEG to rule out non-convulsive seizures

Table 13 summarizing the causes of neurological deterioration after subarachnoid hemorrhage (SAH) a critical aspect of post-bleed management, as timely identification and intervention can significantly improve outcomes. Deterioration is often multifactorial; more than one cause may be contributing simultaneously. Serial neurologic assessments are essential—small changes can be early signs of major complications. Use a systematic approach: check airway, vitals, neurologic exam, imaging, labs, then medications.

Table 14. Predictor variables in models of prognosis for outcome after SAH [24-30,43,45].

Predictor Variable	Category	Impact on outcome	Rationale/Notes
Age	Demographic	Older age = worse outcome	Decreased brain reserve, comorbidities, and recovery potential
Initial Glasgow Coma Scale (GCS)	Clinical	Lower GCS = worse prognosis	Reflects severity of initial neurological insult
Hunt and Hess / WFNS grade	Clinical	Higher grade = poorer outcome	Standardized clinical grading scales incorporating consciousness and motor response
Fisher or modified fisher Grade	Radiological	Higher grade = ↑ risk of vasospasm/DCI	Reflects amount of subarachnoid blood and IVH
Presence of Intraventricular Hemorrhage (IVH)	Radiological	↑ Risk of hydrocephalus, poor outcome	Often included as separate predictor or in modified Fisher grade
Hydrocephalus on admission	Radiological/ clinical	Associated with delayed recovery	Increases ICP, requires CSF diversion
Delayed Cerebral Ischemia (DCI)	Clinical	Major cause of poor outcome	Often not known at admission, but used in post-hoc models
Rebleeding	Clinical	Strongly predicts mortality	Major early cause of deterioration; often fatal
Aneurysm characteristics	Radiological	Location and size can impact risk	Posterior circulation and large aneurysms associated with worse outcomes
Comorbidities (e.g., hypertension, diabetes)	Clinical	Additive risk factor	May impair recovery or exacerbate complications
Admission serum glucose	Laboratory	Hyperglycemia linked to poor outcome	Marker of stress response; may worsen secondary injury
White blood cell count / CRP	Laboratory	Elevated levels may indicate worse prognosis	Reflects inflammatory response and potential secondary injury
Early infarction on CT/MRI	Radiological	Predicts poor functional outcome	Indicates early ischemic injury
Treatment modality (clipping vs coiling)	Intervention	No consistent effect across studies	Often included to account for potential confounding

A comprehensive table outlining the predictor variables commonly included in prognostic models for outcome after subarachnoid hemorrhage (SAH). These models help estimate the likelihood of survival, functional independence, or poor outcomes by integrating clinical, radiologic, and demographic variables.

Table 15. Risk factors for cerebral vasospasm following subarachnoid hemorrhage (SAH) [24-30].

Risk factor	Category	Explanation/Clinical relevance
High fisher/Modified fisher grade	Radiologic	Greater amount of subarachnoid blood increases risk of irritation and vasospasm
Intraventricular Hemorrhage (IVH)	Radiologic	Associated with worse outcomes and higher risk of vasospasm
Younger age (<50 years)	Demographic	Younger patients have more reactive vasculature and greater risk of vasospasm
Female sex	Demographic	Slightly higher risk reported; hormonal influences may play a role
Smoking	Lifestyle	Chronic endothelial dysfunction contributes to susceptibility
Hypertension	Clinical history	Chronic vascular changes may predispose to vasospasm
Early hydrocephalus	Clinical/Radiologic	Linked to increased intracranial pressure and blood burden
High initial neurological grade (e.g., Hunt and Hess >3)	Clinical	Reflects more severe hemorrhage and higher risk of complications
Elevated inflammatory markers (e.g., CRP, leukocytosis)	Laboratory	Systemic inflammation contributes to vascular spasm and microthrombosis
Delayed aneurysm treatment (>72 hours)	Procedural delay	Prolongs exposure of vessels to blood products
Poor cisternal clearance of blood on CT	Radiologic	Persistent blood products increase vasospastic stimuli
Genetic factors (e.g., endothelin-1 polymorphisms)	Genetic/Research	May increase individual susceptibility (under investigation)

A table of risk factors for cerebral vasospasm following subarachnoid hemorrhage (SAH). Vasospasm is a significant cause of delayed cerebral ischemia (DCI) and neurological deterioration in SAH patients, particularly between days 4–14 after the bleed. The volume and distribution of blood on initial CT (Fisher grade) is the strongest predictor of vasospasm. Modifiable risk factors like smoking and delayed treatment should be addressed early. Monitoring high-risk patients closely with transcranial Doppler (TCD) and neurologic exams is essential for early detection.

Table 16. The diagnosis of cerebral vasospasm following subarachnoid hemorrhage (SAH), including both clinical and radiologic approaches [24-29].

Diagnostic method	Type	Utility	Advantages	Limitations
Clinical examination	Bedside / Clinical	Detects new focal deficits, confusion, decreased consciousness	Non-invasive, immediate, low-cost	Not sensitive in sedated/comatose patients
Transcranial Doppler (TCD) ultrasound	Non-invasive Imaging	Monitors mean flow velocities in cerebral arteries	Bedside, serial monitoring, good for trend detection	Operator-dependent, limited acoustic window, indirect measurement
CT Angiography (CTA)	Imaging	Visualizes arterial narrowing	Widely available, non-invasive	May miss distal or moderate vasospasm, radiation exposure
CT Perfusion (CTP)	Imaging	Assesses brain perfusion deficits	Functional insight into ischemia	Requires contrast, limited spatial resolution
Digital Subtraction Angiography (DSA)	Gold standard	Directly visualizes vasospasm	High spatial resolution; allows for concurrent intervention	Invasive, risk of stroke, requires contrast and expertise
MRI/MRA	Imaging	Can detect delayed ischemia and vessel narrowing	Non-invasive, better soft tissue resolution	Less accessible, longer scan time, limited in unstable patients
EEG (for comatose patients)	Functional monitoring	Can detect ischemia through focal slowing	Useful in sedated patients when neuro exam is limited	Indirect; needs correlation with imaging

Initial suspicion often arises from clinical deterioration (new deficits, confusion). In alert patients, clinical exam + TCD is often sufficient for screening. In non-responsive or high-risk patients, combine CTA + CTP, or proceed directly to DSA if intervention is likely.

Table 17. Summarizing the key strategies for prevention of vasospasm and cerebral protection in patients with subarachnoid hemorrhage (SAH) [24-30,41,45].

Strategy/Intervention	Purpose	Mechanism/Rationale	Clinical notes
Nimodipine	Prevent vasospasm & improve outcomes	Calcium channel blocker; improves microvascular perfusion	Standard of care: 60 mg PO q4h for 21 days
Maintenance of euvolemia	Prevent DCI	Ensures adequate cerebral perfusion without fluid overload	Use isotonic fluids (e.g., NS); avoid dehydration or hypotonic fluids
Blood pressure management	Prevent DCI or rebleeding	Post-aneurysm repair: allow permissive hypertension	SBP 160–180 mmHg unless contraindicated
Early aneurysm securing	Prevent rebleeding & vasospasm	Reduces blood burden in subarachnoid space	Ideally within 24–72 hours
Avoidance of hypoxia & hypercapnia	Cerebral protection	Prevents secondary brain injury and increased ICP	Maintain normal oxygenation and ventilation
Magnesium sulfate (adjunct)	Neuroprotective (controversial)	Vasodilatory and anti-excitotoxic properties	Some centers use empirically; trial data inconclusive
Statins (e.g., simvastatin)	Anti-inflammatory, vasoprotective	May stabilize endothelium and reduce vasospasm risk	Mixed evidence; not routinely recommended
Endovascular prophylaxis (e.g., balloon angioplasty)	High-risk patients	Mechanical dilation of spastic vessels	Reserved for documented vasospasm unresponsive to medical therapy
Avoid hyperglycemia	Prevent secondary injury	Hyperglycemia linked to worse neurologic outcomes	Maintain glucose <180 mg/dL
Temperature control	Prevent hyperthermia	Fever exacerbates metabolic demands and neuronal injury	Use antipyretics, cooling if needed
Electrolyte management (Na, Mg)	Cerebral homeostasis	Low Na or Mg associated with worse outcomes, vasospasm risk	Monitor and correct imbalances

Nimodipine and euvolemia are cornerstones of vasospasm prevention. Aggressive monitoring and early treatment of risk factors such as electrolyte imbalance and hypoxia are essential for cerebral protection. Strategies should be individualized based on the patient's neurologic status, comorbidities, and aneurysm repair timing.

Table 18. The management of cerebral vasospasm following subarachnoid hemorrhage (SAH) [24-30,41,45].

Management strategy	Type	Indication	Mechanism/Rationale	Notes
Nimodipine	Pharmacologic (Preventive)	All patients with SAH	Calcium channel blocker; improves outcomes, reduces DCI incidence	60 mg PO/NG q4h x 21 days; not titrated based on vasospasm severity
Induced hypertension ("Triple-H" modified)	Hemodynamic	Symptomatic vasospasm with clinical decline	Increases cerebral perfusion pressure to ischemic areas	Target SBP 160–220 mmHg if aneurysm secured; avoid in unsecured aneurysms
Euvolemia/Volume expansion	Supportive	All SAH patients; especially high risk for vasospasm	Ensures optimal perfusion and prevents hypovolemia-induced ischemia	Use isotonic fluids; avoid hypovolemia or overhydration
Endovascular balloon angioplasty	Interventional	Focal large-vessel vasospasm unresponsive to medical therapy	Mechanical dilation of spastic arteries	More effective in proximal vasospasm; durable response
Intra-arterial vasodilator therapy (e.g., nicardipine, milrinone, verapamil)	Interventional	Diffuse or distal vasospasm with clinical deficits	Temporarily dilates cerebral vessels, improves perfusion	May require repeat dosing; shorter duration effect than angioplasty
Transcranial doppler (TCD) monitoring	Diagnostic / Monitoring	All patients with moderate-to-severe SAH	Detects changes in cerebral artery flow velocities	Used daily for early vasospasm detection; MCA mean velocity >120 cm/s
CT perfusion / CTA / DSA	Imaging	Suspected vasospasm or clinical worsening	Confirms vasospasm and guides endovascular therapy	DSA is gold standard for diagnosis and intervention
Magnesium sulfate (adjunctive)	Pharmacologic (Adjunct)	Used in some centers as preventive or supportive	Mild vasodilator and neuroprotectant properties	Evidence mixed; not standard of care
Sedation/Analgesia optimization	Supportive	All critically ill SAH patients	Reduces metabolic demand and prevents agitation-induced BP surges	Important in patients at high risk for vasospasm

Early recognition and intervention are critical to prevent infarction from vasospasm. Once symptomatic vasospasm occurs, induced hypertension and endovascular rescue therapies are the primary treatments. Daily monitoring with TCD and neurologic exams is essential in the vasospasm risk window (Days 3–14 post-SAH).

Table 19. Neuro-monitoring in aneurysmal SAH (aSAH) [1,3,32-37].

Modality	Target/Threshold	Clinical use/Interpretation
Invasive monitoring		
ICP monitor	ICP < 20 mmHg	Maintain to reduce risk of secondary brain injury
Cerebral Perfusion Pressure (CPP)	CPP > 90 mmHg	Ensures adequate brain perfusion; calculated as MAP - ICP
Pressure Reactivity Index (PRx)	PRx < 0	Indicates intact cerebrovascular autoregulation
Optimal CPP (CPPOPT)	CPPOPT guided by autoregulation (lowest PRx)	Tailored CPP based on patient's best autoregulatory status
Brain tissue oxygenation ($PbtO_2$)	$PbtO_2$ > 20 mmHg	Reflects adequate oxygen delivery to brain tissue
Jugular venous oxygen saturation ($SjvO_2$)	55–75%	Assesses global cerebral oxygenation and extraction
Cerebral microdialysis	L/P ratio < 25 Lactate < 4 mmol/L Glucose > 0.8 mmol/L	Monitors cellular metabolism; detects ischemia or hypoglycemia
Non-Invasive monitoring		
Near-Infrared Spectroscopy (NIRS)	↓ rSO_2 > 14.5% from baseline	Detects regional cerebral desaturation; useful trend in perfusion changes
Transcranial Doppler (TCD)	MFV > 120 cm/s and LR > 3 (suggestive) MFV > 200 cm/s and LR > 6 (strongly suggestive)	Diagnoses cerebral vasospasm, especially in MCA
Optic Nerve Sheath Diameter (ONSD)	> 0.5 cm	Suggests elevated ICP (>20 mmHg); fast bedside tool
Electroencephalography (EEG)	-	Detects seizures (clinical and subclinical) and monitors cerebral function

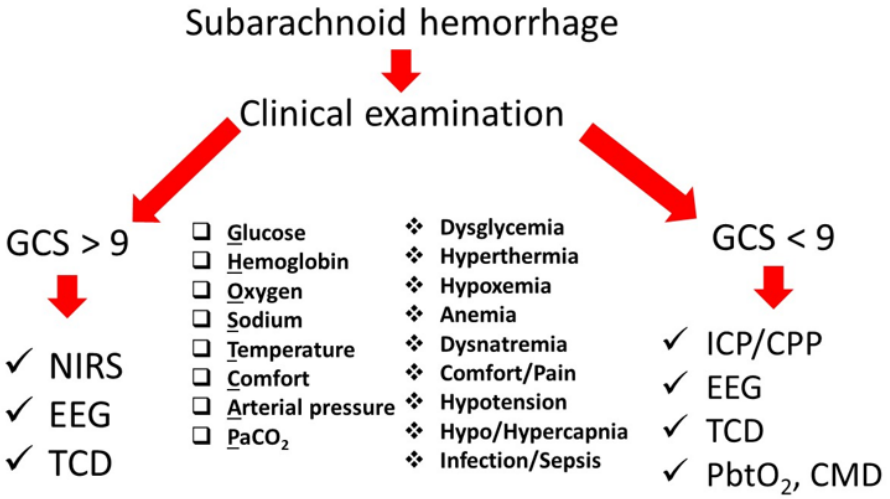

Figure 1. Clinical guidance on monitoring based on Glasgow Coma Scale (GCS) in patients with subarachnoid hemorrhage (SAH), emphasizing the appropriate use of technologies and avoidance of secondary insults.

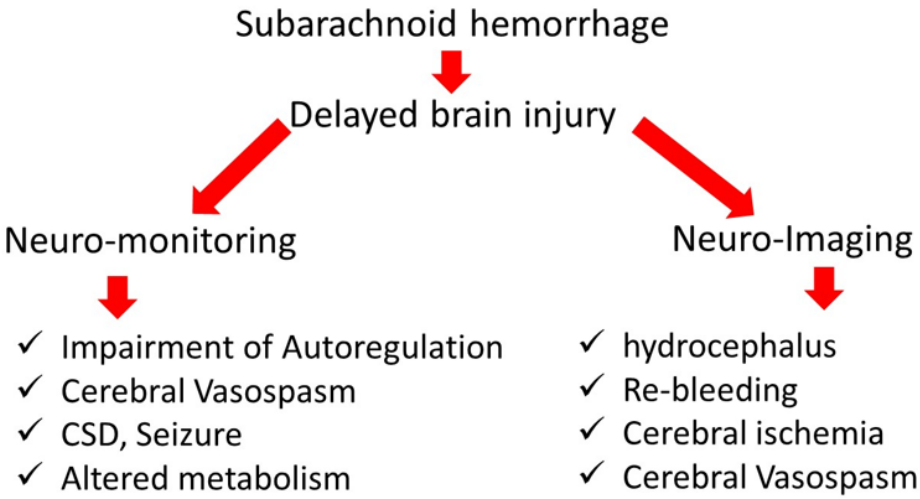

Figure 2. Comprehensive neuro-monitoring and neuroimaging plays a critical role in the early detection and management of these complications.

CONCLUSION

Comprehensive neuro-monitoring in aSAH integrates multimodal invasive and non-invasive tools to detect and respond to early signs of cerebral ischemia, elevated ICP, metabolic failure, and seizures. By continuously assessing cerebral perfusion, oxygenation, autoregulation, and metabolism, clinicians can tailor management in real time mitigating secondary injury, guiding therapeutic targets, and ultimately improving neurologic outcomes. In high-risk or critically ill patients, aggressive monitoring is essential during the peak window for complications, particularly between Days 3 to 14 post-ictus, when vasospasm and delayed cerebral ischemia are most likely to occur.

Effective management of aSAH requires dynamic and individualized monitoring strategies. Patients with preserved consciousness benefit from non-invasive monitoring tools to detect early deterioration. In contrast, comatose or severely impaired patients require comprehensive invasive neuromonitoring to guide cerebral resuscitation and prevent secondary injury. Strict avoidance of systemic complications including dysglycemia, anemia, hyperthermia, hypoxemia, dysnatremia, and infection is critical to optimizing neurologic recovery and survival.

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