THE NeuroICU BOOK

SECOND EDITION

Kiwon Lee

Mc Graw Hill Education

THE NeuroICU BOOK

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With love, this book is again dedicated to my father, Duckhee Lee, who has shown me diligence and love; to my mother, Younghee Lee, who has taught me how to become a leader with legacy; to my one and only sister, Katelyn Jongmee Lee; to my dearest daughters, Sophia Koen Lee and Estelle Charin Lee, who constantly bring me happiness even when I am under stress; and to my lovely wife, Kyongsook Lee, without whose support I certainly would not be at where I am today. I am also grateful to all my colleagues, trainees, and medical students as we continue our journey as a team of healers and teachers for our patients and their families.

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Contents

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Foreword

Three converging realities provide the raisons d'etre for this new edition of *The NeuroICU Book*. First, management of neurological and neurosurgical patients is becoming increasingly complex and fluid as diagnostic and therapeutic options increase. There are few "standards of care" that have not substantially changed over the past few years. Second, as our population increases, engages in risky behavior, and ages, the burden of trauma and neurological disease threatens to overwhelm our hospitals. The number of physicians and other staff who need training to care for these patients is truly exploding. Finally, evidence-based protocol-driven care improves quality metrics, value and patient centered outcomes. Every hospital and healthcare system now accepts and is adapting to this new reality of medical practice. Nowhere are these themes more evident than in the NeuroICU, hence the need for a comprehensive, authoritative, and easily negotiated reference for medical staff managing patients in this environment.

This second edition of *The NeuroICU Book* builds on the huge success of the first edition published in 2012. The interactive, case-driven format of the first edition is retained, with practical recommendations that consider the myriad possible responses of patients to our interventions. Little is predictable in the NeuroICU, and this book prepares the reader for the unexpected. The second edition follows the general organization of the first, with expanded coverage of encephalopathy, spine trauma, pediatric neurosurgery, and newer cardiovascular interventions such as extracorporeal membrane oxygenation.

Dr Lee has assembled an all-star lineup of authors; they are interdisciplinary leaders in their fields. Rarely will one find a more comprehensive and authoritative compilation within the front and back covers of any textbook. The chapters are all edited for consistent style, so that each topic is not only covered with the same degree of expertise and depth, but once familiar with the approach, the reader can easily find within each chapter the coverage they are seeking.

Finally, while this text will be most useful to those who work full-time in the NeuroICU, it is a welcome addition to the bookshelf of any clinician taking care of hospitalized neurological and neurosurgical patients. The new reality of neurological care is that a large percentage of hospitalized patients will spend some time in the NeuroICU, or require ICU-level care while in the ED, endovascular suite, or other non-ICU setting. Hence patients are frequently co-managed by clinicians who will care for the patient before they transition into, or after they move out of,

the NeuroICU. As a vascular neurologist, I frequently dive into the "non-stroke" chapters to help me understand what Dr Lee and his staff are doing or would be doing with my patients!

James C. Grotta, MD
June 26, 2016

Preface

A lot has happened since the first edition of this book and my departure from New York City to arrive in Houston, Texas. The field of neurology continued to evolve, especially in the area of therapeutics. Multiple class I-type of evidence emerged from intraarterial thrombectomy studies showing its undeniable, outcome-improving intervention, when combined with intravenous rt-PA compared to intravenous thrombolysis alone in 2015. This ground-breaking study result, along with other therapeutic advances in other neurological subspecialties, further transformed the field of neurology from merely admiring phenomenology to more time-sensitive interventional field. The field of neurocritical care is at the forefront of therapeutic efforts, as our daily practice focuses on resuscitating and reversing multiple organ failures.

The idea of neurologic critical care is to provide acute medical therapies and appropriate interventions promptly by constantly monitoring the patients in one area by specially trained physicians and nurses. Injured brains do not wait for consults to show up and have a low threshold for irreversible damages. "Time is brain" in many situations, and it is of paramount importance that neurocritical care team must physically staff the unit 24/7 and act quickly in the event of neuro-emergencies. Patients with acute, severe brain injuries are often accompanied by other organ failures at the time of initial presentation or during the ICU stay. Therefore, neurointensivists must be trained to handle not only the brain, but also rest of the body. This textbook is written for that very reason. Readers will once again find this textbook being not just about the brain and spine. It is about all organ insufficiencies and failures along with neurologic illnesses. After the success and popularity of the first edition, the second edition again emphasizes and focuses on one principle: practicality. This book is easy to read and full of real cases that one may encounter in a neurocritical care unit. The flow of content is in the form of dialogue. You will feel like you are making rounds with me. By discussing and focusing on multiple challenging problems, and by paying attention to all organs rather just the brain, it is my hope that you would find this textbook extremely helpful for both daily practices as well as for board examinations.

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SECTION 1

Neurocritical Care Diseases

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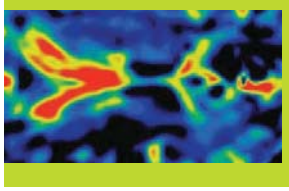
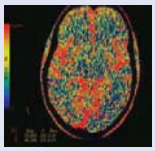


CHAPTER 1

Subarachnoid Hemorrhage

Kiwon Lee, MD, FACP, FAHA, FCCM



A 49-year-old man with history of hypertension and hyperlipidemia presents with a sudden onset of severe bifrontal headache followed by nausea. The headache, the worst headache he had ever experienced, came on suddenly. The patient vomited on his way to the nearby emergency department (ED) and became obtunded in the ambulance. On arrival to the ED, he was intubated for airway protection as his mental status continued to worsen. About 30 minutes after the onset of the initial symptoms, he progressed to stuporous mental status. He was able to flex his elbows bilaterally to painful stimulation. Brainstem reflexes were all intact. Stat head computed tomography (CT) (Figure 1-1) revealed acute subarachnoid hemorrhage (SAH) filling the basal cistern, bilateral sylvian fissures with thick hemorrhages along with early radiographic evidence for hydrocephalus, and intraventricular hemorrhage (IVH) mainly in the fourth ventricle. The local ED physicians decided to transfer the patient immediately to the nearest tertiary medical center. During the emergent transfer, patient stopped responding to any painful stimuli and had only intact brainstem reflexes.

On arrival at the neuroscience intensive care unit (NeuroICU), the following is the clinical observation: Patient is intubated with endotracheal tube, is in coma, decerebrate posturing to painful stimulation, has intact corneal reflexes, pupils 5 mm in diameter briskly constricting to 3 mm bilaterally to light, intact oculocephalic reflexes, and positive bilateral Babinski signs.

Vital signs on arrival to the NeuroICU: heart rate, 110 bpm in sinus tachycardia; respiration rate, 20 breaths per minute on the set rate of 14 breaths per minute on assist control–volume control mechanical ventilation; temperature, 99.3°F; and blood pressure (BP), 190/100 mm Hg by cuff pressure.

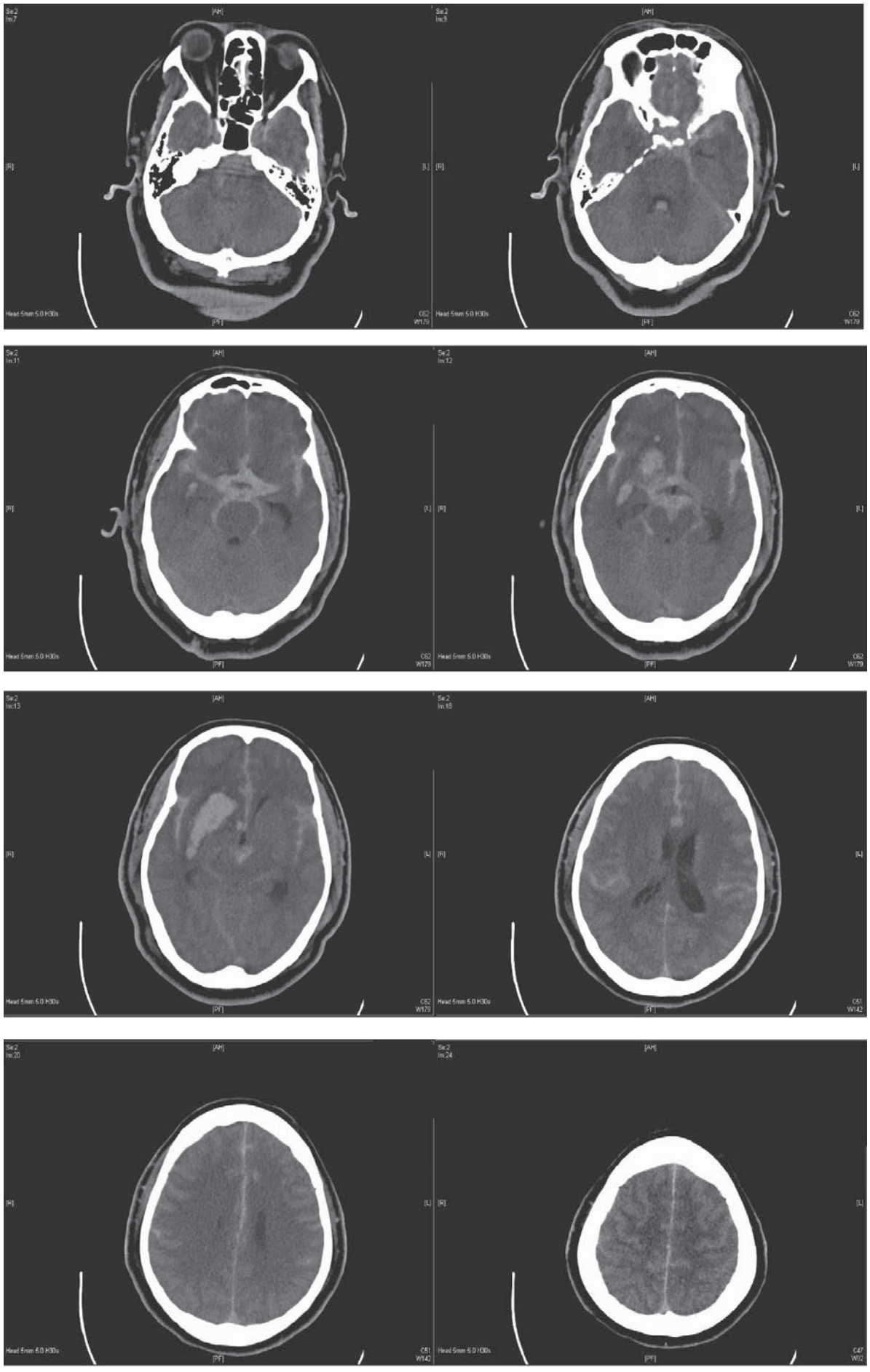


Figure 1-1. Axial CT images of the brain without contrast.

What are the initial steps for resuscitating acute aneurysmal SAH in this case?

ABC and EVD. You must optimize cerebral perfusion pressure (CPP) for all poor-grade SAH

The clinical and radiographic presentation of this case is consistent with poor grade (initially Hunt and Hess [HH] grade IV, which quickly progressed to grade V while in transit to the tertiary care center) acute aneurysmal SAH. Airway, breathing, and circulation (ABC) have all been addressed, although the BP is high at this time. The very first step in managing this patient is ventricular drain, the second step is ventricular drain, and the third step is ensuring that the ventricular drain you have just placed is working (ie, draining bloody cerebrospinal fluid [CSF] adequately when the drain is open, and maintaining good waveforms when the drain is clamped). After ABC, placing external ventricular drain (EVD) is the most crucial, lifesaving, important early step for managing patients with *high-grade* acute SAH with poor mental status and IVH. The presence of IVH complicates the natural course of both intracerebral hemorrhage (ICH) as well as SAH cases. IVH is often associated with development of an acute obstructive hydrocephalus, which may lead to vertical eye movement impairment and depressed level of arousal by its mass effect on the thalamus and midbrain. IVH is also associated with elevated intracranial pressure (ICP), which lowers the CPP (by the principle of the equation, CPP = MAP – ICP) if the mean arterial pressure (MAP) remains constant. Moreover, IVH has been reported to be an independent risk factor for increased risk of developing symptomatic vasospasm. The mass effect and cerebral edema may rapidly progress to herniation syndrome and death. As such, the presence of IVH has been recognized as a significant risk factor for poor outcome for both ICH and SAH.[1-3] Placing an EVD provides two benefits: (1) reliable (as long as the catheter tip is in the right location to provide appropriate ICP waveforms without obstructing the ventricular catheter by any blood clot) measurements of the ICP and (2) therapeutic drainage of the CSF in order to alleviate the intracranial hypertension (Figure 1-2). Placement of an EVD may not be necessary in a low-grade ([HH] grade 1-2) SAH patient if IVH is not severe and hydrocephalus is not present. In a high-grade patient, however, even if hydrocephalus is not seen in the first CT scan, if the patient has bled significantly (ie, a thick basal cistern SAH clot with classic Fisher group 3 and a modified Fisher grade 3 or 4; more on this below), placement of an EVD is often required.

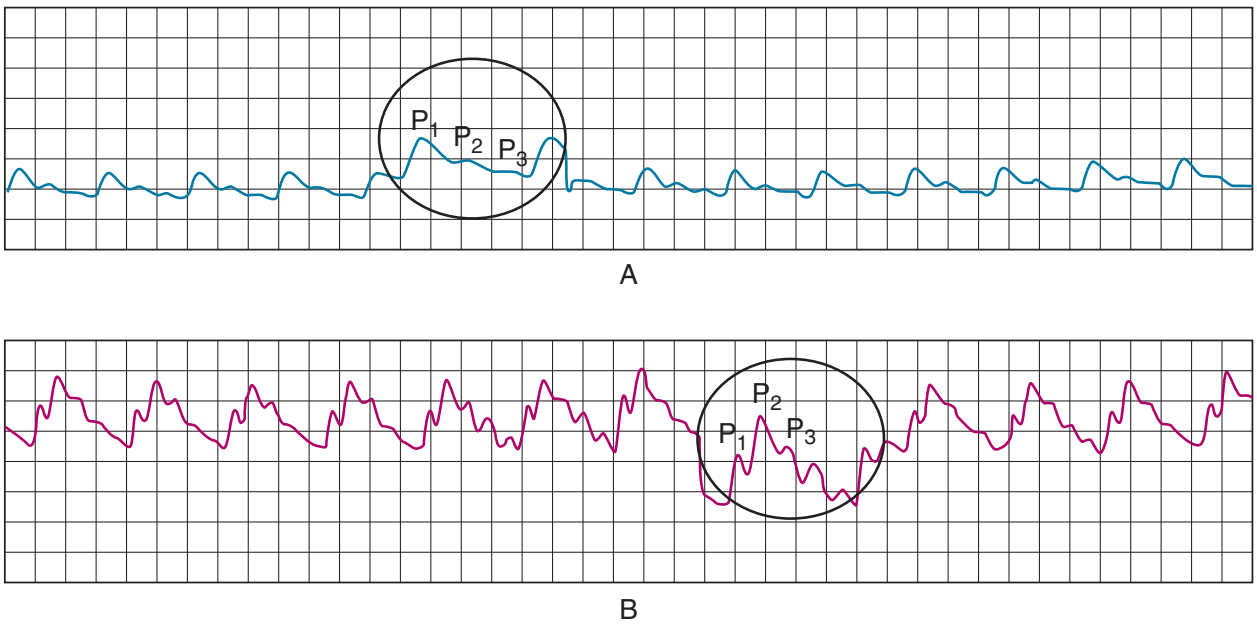


Figure 1-2. ICP waveforms and compliance. **A.** ICP waveform with normal compliance. **B.** ICP waveform with poor compliance.

It is important to note that the presence of IVH does not necessarily mean the ICP is abnormally elevated, and the placement of an EVD alone may not always lead to an improved outcome even if the high ICP responds favorably to opening and lowering of the drain.[4] In the past, there were concerns regarding the potential harmful effect of EVD placement in treating the acute hydrocephalus in SAH patients. These concerns were mainly focused on the theoretical impact of suddenly lowering the ICP by EVD placement and eliminating the tamponade effect on a ruptured aneurysmal wall, leading to an increased risk of rebleeding in the acute phase. However, clinical studies have failed to prove such a hypothesis, and there is not sufficient evidence to believe that the CSF diversion by an EVD in treating acute hydrocephalus after SAH results in a higher incidence of rerupturing of the unsecured aneurysms.[5,6] It is wise, however, to avoid aggressively lowering the drain level immediately after placement. For example, leaving the EVD open at about 15 cm above the level of the external auditory meatus is reasonable before securing the aneurysm. In managing SAH cases, whether or not to place an EVD is occasionally debatable. For instance, a patient with low-grade (eg, HH I or II) SAH who is awake, follows commands with normal strength, and has no IVH, no acute hydrocephalus, and either absent or minimal volume of SAH (eg, classic Fisher groups 1 to 2) is *not* a candidate for EVD placement. On the other hand, a patient with a high HH grade and Fisher group 3 SAH, plus the radiographic evidence of severe IVH and acute hydrocephalus, who exhibits a progressively worsening level of arousal needs *emergent* placement of an EVD (classic indication for ABC and EVD). These are extreme ends of the clinical spectrum of SAH, and the timing and indication for EVD could be debated for the cases that are somewhere between these two extreme case scenarios. Acute hydrocephalus with IVH and clinical signs and symptoms of intracranial hypertension are all good indications for placing EVDs. It is also important to remember that even if the patient does not have any of the indications mentioned above, if the treating physician believes that there is a reasonable probability of developing these signs and symptoms in the *near future*, EVD placement should be considered. (Technical details and further management strategies are discussed in Chapter 22.) Despite the lack of "level 1" evidence of randomized data for improved outcomes, the use of an EVD is important because it can be helpful in managing ICP and CPP and often is lifesaving in certain SAH patients.

This patient's level of arousal improves a few minutes after placing the EVD (opening pressure was 35 mm Hg). He is now able to localize to painful stimulations. Does the prognosis change with improved neurological examination after EVD placement?

Changing Neurologic Status *After* EVD Placement

Placement of an EVD frequently results in a significant improvement in neurologic status. Comatose patients may start to localize to painful stimulation and may even open their eyes. Although this is not always seen, when it happens, it may possibly indicate a favorable outlook (eg, a patient who presents with HH grade V after aneurysmal SAH wakes up after EVD placement and begins to follow verbal commands: if this patient remains awake and continues to follow commands throughout the course of his or her illness, then the patient is behaving at a low-grade HH [ie, grades I to III], not like a grade V patient who presents with and remains in coma).

Patients with HH grade V have extremely poor prognosis. Many physicians and surgeons disclose such a poor prognosis to the patient's family, and this disclosure often leads to withdrawal of life-sustaining care prior to any treatment. Although the decision to treat or not to treat should be made based on the prognosis and in the best interest of the patient, the initial prognosis is mostly based on the bedside neurologic assessment. Physicians should be aware that the patient's clinical status may *dramatically* change after placement of an EVD, which has significant implications for the prognosis.[7] When a patient with high-grade SAH is stuporous or comatose, it is difficult to determine whether

such exam is due to hydrocephalus and IVH or to the initial injury, which is independent of the hydrocephalus and/or IVH. Thus, it would be prudent to wait until the initial resuscitation (ABC and EVD) is completed before making any predictions.

There are several SAH grading systems worth mentioning here. In 1967, Hunt and Hess reported 275 consecutive patients who were treated at the Ohio State University over a 12-year period. They believed that the intensity of the meningeal inflammatory reaction, the severity of neurologic deficit, and the presence or absence of significant systemic disease should be taken into account when classifying SAH patients. From their original manuscript, their grading system (which is now known and widely used as the Hunt and Hess Grade) was a classification of patients with intracranial aneurysms according to surgical risk (Table 1-1).[8]

Higher grades are associated with increased surgical risk for the repair of ruptured intracranial aneurysms. The Hunt and Hess original report included the presence of significant systemic disease (such as "hypertension, diabetes, severe arteriosclerosis, chronic pulmonary disease, and severe vasospasm seen on angiography") as a negative sign, and the presence of such disease resulted in placement of the patient in the next less favorable (higher surgical risk) grading category.[8] This grading system is not flawless as it can be challenging sometimes to differentiate between categories. For example, consider a patient with SAH with mild headache and nuchal rigidity compared with another patient with moderate headache and nuchal rigidity (which means grades I and II, respectively, according to the original HH grading system). The only differentiating variable here would be the intensity of the headache, which can be problematic because the intensity of headache is subjective, and patients often cannot differentiate mild from moderate headache (most people would describe a "very bad" headache and cannot provide specific details).

This criticism had been actually predicted, and the original authors mentioned it in their journal article: "It is recognized that such classifications are arbitrary and that the margins between categories may be ill-defined."[8] For this reason, it has been pointed out that the HH system has poor interobserver reliability and reproducibility.[9] Nevertheless, the HH grading system is widely used, and numerous studies have shown that the higher grade (or sometimes called poor grade, which usually refers to HH grades IV and V) is associated with a poor outcome.[10-13]

Another grading system to consider is the one that is the most universally accepted system for patients presenting with an altered level of consciousness: the Glasgow Coma Scale (GCS). In 1975, Jennet and Bond, from the University of Glasgow, reported a scale called Assessment of Outcome After Severe Brain Damage, a Practical Scale (Table 1-2).[14]

Table 1-1. Hunt and Hess Grade for SAH[a]

Criteria	Category
Grade I	Asymptomatic, or mild headache and slight nuchal rigidity
Grade II	Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy
Grade III	Drowsiness, confusion, or mild focal deficit
Grade IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity and vegetative disturbances
Grade V	Deep coma, decerebrate rigidity, moribund appearance

[a]Classification of patients with intracranial aneurysms according to surgical risk.

(Reproduced with permission from Hunt W, Hess R. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg. 1968;28:14-20.)

Table 1-2. Glasgow Coma Scale

Category	Score
Eye opening	
Spontaneous	4
To loud voice	3
To pain	2
None	1
Verbal response	
Oriented and converses	5
Confused, disoriented	4
Inappropriate words	3
Incomprehensive sounds	2
None	1
Best motor response	
Obeys commands	6
Localizes to pain	5
Withdraws (flexion)	4
Abnormal flexion posturing	3
Extension posturing	2
None	1

(From Jennett B, Bond M. Assessment of outcome after severe brain damage. Lancet. 1975;1:480-484.)

The GCS is a more general grading system and was not developed specifically for SAH patients. However, studies show that for patients with aneurysmal SAH, the initial GCS score has positively correlated with long-term outcome.[15]

In 1988, the World Federation of Neurosurgical Societies (WFNS) developed a grading system that incorporated both the GCS and bedside neurologic assessment focusing on any focal deficit (Table 1-3).[16]

The HH and WFNS grading systems are by far the two most commonly used systems for grading patients with acute aneurysmal SAH. Despite the frequently raised criticisms regarding the interobserver variability, the HH grade is used even more commonly than the WFNS scale

Table 1-3. World Federation of Neurosurgical Societies Scale for SAH

Grade	Criteria
I	GCS 15 without focal deficit[a]
II	GCS 13-14 without focal deficit
III	GCS 13-14 with focal deficit
IV	GCS 7-12 with or without focal deficit
V	GCS 3-6 with or without focal deficit

Abbreviations: GCS, Glasgow Coma Scale; SAH, subarachnoid hemorrhage.

[a]Focal deficit is defined as either aphasia and/or motor deficit.

(From Drake C. Report of World Federation of Neurological Surgeons Committee on a Universal Subarachnoid Hemorrhage Grading Scale. J Neurosurg. 1988;68:985-986.)

Table 1-4. Fisher Scale of SAH

Group	CT finding description
1	No detectable SAH
2	Diffuse SAH, no localized clot > 3 mm thick or vertical layers > 1 mm thick
3	Localized clot > 5 × 3 mm in subarachnoid space or > 1 mm in vertical thickness
4	Intraparenchymal or intraventricular hemorrhage with either absent or minimal SAH

Abbreviation: SAH, subarachnoid hemorrhage.

(From Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. Neurosurgery. 1980;6:1-9.)

(71% of reported studies from 1985 to 1992 used the HH grade compared with 19% that used the WFNS scale),[17,18] and both grading systems have been shown to correlate reasonably well with the long-term outcome.[19]

In 1980, Fisher and colleagues reported the relationship between the amount of SAH and the risk of developing severe vasospasm (defined as delayed clinical symptoms and signs; Table 1-4).[20]

The Fisher group's grading system is based on the description of CT findings, mainly focusing on the actual volume of blood in the subarachnoid space. There is a linear relationship between the amount of hemorrhage and the rate of developing symptomatic vasospasm.[20] This grading system has been extensively studied and there are numerous clinical studies validating its usefulness.[21-25] In multiple studies, the risk of developing symptomatic cerebral vasospasm appears to increase along with the increasing amount of acute hemorrhage in the subarachnoid space. Although original report by Fisher et al does describe the low risk of vasospasm, there is a clearly observed risk of vasospasm even for patients with minimal blood in the subarachnoid space and for those with intraparenchymal or intraventricular hemorrhage.[20]

It is important to understand that the Fisher scale actually did report some incidence of vasospasm in groups 1, 2, and 4. Group 3 had the highest incidence of vasospasm, but other groups also had vasospasms, just much lower in frequency.[20] Like all other grading systems, the Fisher scale is not without limitations. There have been concerns in the literature reporting a low correlation between the Fisher grade and the incidence of symptomatic vasospasm (one recent study showed about 50% correlation between the Fisher grade and vasospasm).[26] Another criticism about the Fisher scale is its inevitable interpersonal variability in assessing the estimated blood volume. Also, according to the scale, all cases of CT of the head showing SAH with greater than 1 mm of vertical thickness is categorized as grade III, but this includes vast majority of patients with SAH who may not in fact have the same risk of developing vasospasm.[26,27]

In light of these concerns, Claassen et al's group, from Columbia University, proposed another grading system (Table 1-5): the modified Fisher scale (mFS).[28,29]

Note that the mFS incorporates the presence or absence of IVH, and if a patient has IVH, even if there is no blood in the subarachnoid space, the scale is 2 (as opposed to 1 [no blood seen] or 4 [minimal SAH and the presence of intraparenchymal hemorrhage or IVH] in the original Fisher scale). This scale emphasizes that the presence of IVH increases the risk of developing symptomatic vasospasm. This emphasis is stronger but not completely different from that of the Fisher scale, because the original Fisher scale does report some incidence (although low) of vasospasm in those with IVH and absent or minimal SAH. Furthermore, the mFS uses a subjective description and coding of the hemorrhage by the use of "thick" or "thin" clots in the subarachnoid space, and the description of IVH does not take the exact amount of IVH into account (this scale takes the "presence" versus the "absence" of IVH into account, not how much IVH there is). The mFS emphasizes the importance

Table 1-5. The Modified Fisher Scale

CT finding description	IVH	Modified Fisher Scale
Diffuse thick SAH	Present	4
	Absent	3
Localized thick SAH	Present	4
	Absent	3
Diffuse thin SAH	Present	2
	Absent	1
Localized thin SAH	Present	2
	Absent	1
No SAH	Present	2
	Absent	0

Abbreviations: IVH, intraventricular hemorrhage; SAH, subarachnoid hemorrhage.

(From Claassen J, Bernardini GL, Kreiter KT, et al. Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemorrhage: the Fisher Scale revisited. Stroke. 2001;32:2012-2020.)

of IVH, and it also highlights how the amount of hemorrhage once again plays an important role. Its grading system is easy and intuitive (unlike the classic Fisher scale in which group 4 actually has a lower incidence of vasospasm than lower grades), as the scale goes from 0 to 4, and the higher grade has the higher risk of developing delayed cerebral ischemia (DCI).

In order to minimize the interobserver variability in assessing the estimated volume of blood in the subarachnoid space, a volumetric quantification of Fisher grade 3 has been proposed and studied by Friedman and colleagues from the Mayo Clinic.[30] However, although quantification of SAH may provide a more accurate assessment of the volume of blood in the subarachnoid space, it requires *manual* outlining of the hemorrhage volume, which can be time consuming and less reliable.

In 2011, Ko and colleagues, from Columbia University, reported a study of volumetric analysis of SAH using a MIPAV (Medical Image Processing, Analysis, and Visualization; version 4.3; National Institutes of Health [NIH]) software package that *automatically* outlines the hemorrhage on CT at the click of a button.[31] This quantification analysis showed that patients with a higher volume of cisternal *plus* IVH clot burden developed a greater risk of developing DCI and poor outcome at 3 months (Figure 1-3).

It also validated the modified Fisher scale as a reasonable grading system in predicting DCI that can be done easily at the bedside. However, it is important to note that although both the Fisher scale and the mFS have demonstrated the association between blood burden and DCI, a question still remains: Do the location and exact thresholds of blood volume matter? Ko and colleagues reported.[31]

> Our data show that the quantitative blood volume in contact with the cisternal space, whether directly in the cisternal subarachnoid space or intraventricular space, acts as *cumulative* blood burden and is associated with an increased risk of DCI. The quantitative volume scale and the mFS were equivalent in predicting DCI, validating the accuracy of the mFS. However, volumetric analysis had no overlaps in the odds ratio for DCI in different blood burden groups, which may suggest more robust association between the total blood burden and DCI.

Klimo and Schmidt have eloquently summarized a historical review of the literature on the relationship between the CT findings and the rate of developing cerebral vasospasm after aneurysmal SAH using different scales.[32]

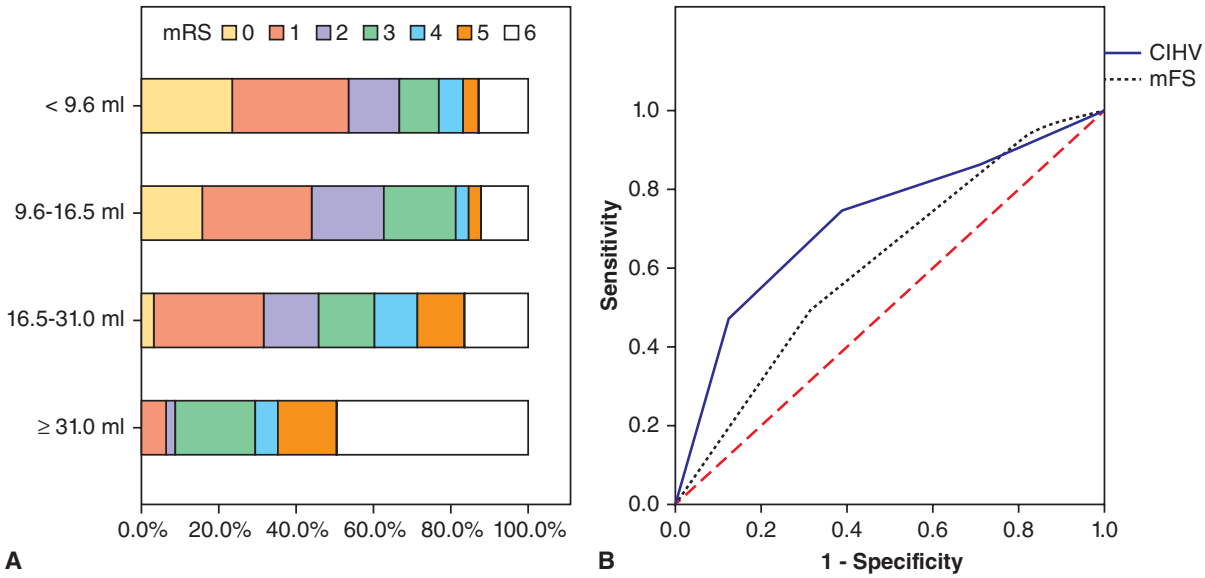


Figure 1-3. The effect of blood volume on functional outcome at 3 months. **A.** Using cisternal blood plus intraventricular hemorrhage volume (CIHV) criteria, patients with higher quartiles had higher risk of death or severe disability at 3 months. **B.** CIHV was better at predicting 3-months outcome than modified Fisher Scale (B). (*Reproduced with permission from Ko SB, Choi HA, Carpenter AM, et al. Quantitative analysis of hemorrhagic volume for predicting delayed cerebral ischemia after subarachnoid hemorrhage. Stroke. 2011 Mar;42(3):669-74. https://doi.org/10.1161/STROKEAHA.110.600775.*)

The elucidation of predictive factors of cerebral vasospasm following aneurysmal subarachnoid hemorrhage is a major area of both clinical and basic science research. It is becoming clear that many factors contribute to this phenomenon. The most consistent predictor of vasospasm has been the *amount* of SAH seen on the postictal CT scan. Over the last 30 years, it has become clear that the greater the amount of blood within the basal cisterns, the greater the risk of vasospasm. To evaluate this risk, various grading schemes have been proposed, from simple to elaborate, the most widely known being the Fisher scale. Most recently, volumetric quantification and clearance models have provided the most detailed analysis. IVH, although not supported as strongly as cisternal SAH, has also been shown to be a risk factor for vasospasm.

Angiography shows an anterior communicating (A-comm) artery aneurysm, and coiling was performed to secure the ruptured aneurysm. The patient returns to the ICU but now has elevated ICP of 50 to 55 mm Hg with MAP of 100 mm Hg.

What is the stepwise approach for treating high ICP for SAH patients?

The first battle in high-grade SAH: The battle against elevated ICP

The early phase of high-grade SAH is often complicated by the presence of ICP crisis. An ICP value out of the normal range (0-20 mm Hg) is considered abnormal, but the ICP alone as an absolute value may not always signify the need for an urgent treatment. A good example would be people with pseudotumor cerebri and high ICP who perform normal daily activities. ICP also rises when patients cough or are suctioned. Such an increase, if it is induced and transient, does not necessarily require any treatment. In the setting of acute, high-grade SAH, however, abnormally elevated ICP *is* a major concern owing to its direct, negative impact on the CPP. With persistently low or decreasing CPP, a certain degree of ischemic insult is inevitable. A step-by-step algorithm for managing refractory ICP crisis is outlined below. This is a recommendation that reflects the latest medical treatment available in the literature.