INTRACEREBRAL HEMORRHAGE

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OVERVIEW

Intracerebral hemorrhage (ICH) is defined as bleeding into the brain parenchyma that is distinct from subarachnoid hemorrhage (SAH) and isolated intraventricular hemorrhage (IVH). Currently there is no approved treatment of proven benefit in decreasing morbidity and mortality after ICH. However, guidelines do exist regarding management and these address a variety of issues including blood pressure control, the role of surgery, and other aspects of initial medical therapy(1).

EPIDEMIOLOGY

Intracerebral hemorrhage accounts for 10-15% of the approximately 700,000 annual strokes in the United States (2). The incidence of ICH in the United States is approximately 12-15 cases per 100,000.

Although ICH comprises a minority of strokes that occur in the United States, it accounts for a disproportionately large amount of the total morbidity, mortality, and economic burden of stroke. The economic burden of ICH is tremendous, approaching 6 billion dollars annually in the United States, and approximately $165,000 per patient/year (3).

Less than one-third of patients will be functionally independent after experiencing an ICH. The short-term mortality of ICH in most series is approximately 40% and has not improved significantly in recent years despite the growth of neurointensive care. While this is a direct reflection of the absence of any effective proven treatments for ICH, there is also concern that heterogeneity in care and early care limitations may lead to self-fulfilling prophecies of poor outcome (4-6). Because of this concern, recent ICH guidelines recommend careful consideration of aggressive care for at least the first full day in all ICH patients and deferring of new do-not-resuscitation orders during that time (1).

PATHOPHYSIOLOGY

Primary ICH is due to rupture of small arterioles. Hypertension accounts for approximately 60-70% of all ICH. Typical locations for hypertensive ICH include the basal ganglia, thalamus, cerebellum, pons, and deep lobar white matter. Cerebral amyloid angiopathy (CAA) is an increasingly recognized cause of primary ICH, particularly in elderly patients.
Recurrent hemorrhage risk from amyloid deposition is tripled with the presence of apolipoprotein E gene ε2 and ε4 alleles (7). Hemorrhages due to CAA are usually located in the peripheral lobar white matter near the grey and white matter junction.

Secondary ICH occurs in the context of an underlying pathology that predisposes the patient to hemorrhage (e.g. vascular malformation or tumor). Other important etiologies of both primary and secondary ICH include coagulopathy, sympathomimetic drugs of abuse, vasculitides, and Moya-Moya. The etiology of ICH is usually ascribed from consideration of the combination of the clinical presentation, patient risk factors, and imaging characteristics of the hemorrhage.

The underlying pathologic process that causes the rupture of small arterioles (<100 micron diameter) in hypertensive ICH has been termed lipohyalinosis. This process is characterized by subintimal fibroblast proliferation, deposition of lipid-filled macrophages, and replacement of smooth muscle cells in the tunica media of the larger vessels with collagen. This results in reduced blood vessel elasticity and increased susceptibility to spontaneous rupture.

Primary brain injury in ICH is due to tissue destruction caused by the initial hemorrhage as blood transects white matter tracts and destroys neurons. More recently, the importance of damage caused by mechanical effects of the hematoma has been augmented by interest in mechanisms of secondary brain injury. This has been spurred by the observations that early hematoma growth is common and that many patients also deteriorate clinically without hematoma growth, in the same time frame in which edema is developing and clot absorption and breakdown are occurring. There is increasing evidence that plasma proteins abundant in vasogenic edema and increased by clot resorption are harmful to the brain. Patients with a higher ratio of edema to hematoma volume have been retrospectively shown to have poorer outcomes (8). Thrombin, as well as hemoglobin and its breakdown products, have been demonstrated to be neurotoxic via glutamate mediated excitotoxicity, exacerbate acute peri-hematoma edema, and contribute to disruption of the blood-brain barrier. Additionally, interleukin-1 and matrix metalloproteinases (MMPs) are upregulated in the neurons and astrocytes of the peri-hematoma region. Peri-hematoma edema is reduced in experimental models of ICH with both MMP–9 knockout mice and with administration of IL-1 receptor antagonists. Overall, the potentially toxic effects of iron as well as a range of inflammatory mediators have led to the concept of “neurohemoinflammation” as a descriptor for a variety of different pathways which may result in secondary brain injury after ICH. These small molecules and their biochemical signaling pathways, or even iron chelating agents such as deferoxamine, represent potential targets for future acute ICH therapy (9-11).

Concern for peri-hematoma ischemia has now lessened as a major mechanism of secondary injury in ICH. Cerebral blood flow studies using SPECT and MRI perfusion and diffusion have attempted to demonstrate a peri-hematoma penumbra that is at risk for additional injury and neuronal loss due to hypoperfusion.
The significance of these findings have been called into question by more recent CT perfusion studies that failed to show a penumbra, by PET studies that have found that these areas of “penumbra” may in fact be appropriately perfused in the setting of reduced metabolic activity, and by animal studies which suggest a zone of hypoperfusion without impaired oxygen metabolism (12, 13).

In the past, ICH has been thought of as a monophasic event with an initial hemorrhage that grew to its maximal size within moments, with rehemorrhage or hematoma expansion as rare events suggestive of coagulopathy or underlying vascular anomaly. However, numerous studies have now demonstrated that hematoma expansion is common early after acute ICH, even in the absence of an underlying lesion or coagulopathy. In a single center prospective study, substantial hematoma growth, defined as > 33% enlargement of the baseline hematoma volume, occurred during the first day in 38% of patients who underwent CT scanning within three hours of the initial ictus; 26% of patients demonstrated this enlargement within 1 hour after initial CT scan (14). Retrospective studies have found similar rates of hematoma expansion ranging from 18-36% with substantially lower rates of delayed re-bleeding beyond six hours of 2-10%. Of note, when examining the placebo group of a phase IIa study of recombinant Factor VIIa for acute ICH, 73% of patients demonstrated some degree of hematoma expansion over the first day (15). Because hematoma expansion is an important independent determinant of overall outcome, it is now being strongly considered as a potential target for intervention with hemostatic agents or even aggressive blood pressure control in order to limit hematoma growth.

CLINICAL FEATURES

The clinical presentation of ICH is characterized by the sudden onset of focal neurologic dysfunction that is generally accompanied by severe headache. However, headache at onset does not reliably distinguish ICH since it occurs in up to 30% of patients with ischemic strokes. Patients with large hemispheric ICH that acts as a mass or who have significant IVH that obstructs cerebrospinal drainage may have profoundly elevated intracranial pressure and often present with nausea and vomiting, in addition to focal neurologic deficits that may rapidly progress to herniation and coma. Coma with pin-point pupils is suggestive of pontine tegmental hemorrhage.

Various baseline clinical and neuroimaging characteristics are predictive of outcome in intracerebral hemorrhage. These include hematoma volume, Glasgow Coma Scale (GCS) score, IVH, advanced age, and infratentorial ICH location (16-18). Numerous outcome prediction models and grading scales have been developed using these parameters which are available at the time of initial ICH evaluation and may be overall predictive of short-term (30-day) mortality or long-term functional outcome. The most commonly used validated scale, the ICH Score (17, 19), is calculated as the sum score of points assigned for each of five different parameters (Table 2).
Hematoma volume can be estimated from the head CT scan using the ABC/2 method, which approximates the volume of a spheroid. From the axial CT slice containing the largest hematoma area, A is the longest diameter and B is the longest diameter perpendicular to A. C is the number of slices on which hemorrhage is seen multiplied by the slice thickness (note that slices containing an estimated < 25% of the largest volume are not counted, slices with 25-75% are counted as a half slice, and slices > 75% are counted as a full slice). (20)

**DIAGNOSIS**

The etiology of ICH is usually ascribed from consideration of the combination of the clinical presentation, patient risk factors, and imaging characteristics of the hemorrhage.

The widespread use of CT scanning has made the diagnosis of ICH relatively straightforward, and it remains the most widely used neuroimaging technique. Acute stroke MRI protocols utilizing susceptibility-weighted imaging that exploit the paramagnetic properties of hemoglobin can also accurately identify ICH with very high sensitivity and specificity as compared to CT (21). Intracranial vascular imaging with conventional angiography has a high yield in identifying vascular malformations in cases of ICH in younger patients (age < 45), atypical hemorrhage location for hypertension, if the patient has no history of hypertension, or in primary IVH. Multi-slice CT angiography has been proposed as a surrogate for conventional angiography in the investigation of neurovascular disorders, but the sensitivity is insufficiently adequate if a vascular malformation is not identified to advocate foregoing conventional angiography. Additionally, in cases of acute ICH, CT angiography with early and delayed image acquisition may be helpful in identifying patients with active contrast extravasation as the presence of this “spot sign” is a predictor of hematoma expansion and worsened outcome (22, 23). This type of imaging could theoretically help target interventions toward patients most likely to experience ongoing hematoma expansion, but this has not yet been tested prospectively.

**TREATMENT**

**Blood Pressure**

Elevated blood pressure (BP) is extremely common in the setting of acute ICH. Blood pressure management in this setting remains controversial because of concerns over balancing the competing interests of limiting hematoma expansion or rebleeding while avoiding the theoretical risk of secondary ischemic brain injury by hypoperfusing peri-hematoma brain parenchyma. Studies have conflicted over whether elevated blood pressure predisposes to hematoma expansion after acute ICH (24, 25). However, recent studies have suggested that peri-hematoma ischemia is unlikely to be a major contributor to ICH-related brain injury in most cases (12, 13).
Even so, there still remains a relative dearth of data to support specific blood pressure goal recommendations and recent American Heart Association/American Stroke Association guidelines for the management of ICH continue to recommend individualized blood pressure goals based upon individual patient characteristics such as presumed etiology of hemorrhage (hypertension versus underlying vascular anomaly), history of chronic hypertension and baseline blood pressure, and known or suspected major vessel arterial stenosis where a significant decline in blood pressure could cause secondary organ damage (1). These guidelines suggest the following potential approaches: [1] if systolic blood pressure (SBP) is >200 mmHg or mean arterial pressure (MAP) >150 mmHg then consider aggressive BP reduction with a continuous intravenous infusion and frequent monitoring of BP and neurologic examination; [2] if SBP is >180 mmHg or MAP is >130 mmHg and there is evidence of or suspicion of elevated intracranial pressure (ICP), then consider monitoring ICP and reducing BP using intermittent or continuous intravenous medications to keep the cerebral perfusion pressure (CPP) between 60 and 80 mmHg; [3] if SBP is >180 or MAP is >130 and there is not evidence of or suspicion of elevated ICP, then consider a modest reduction of BP (e.g. MAP < 110 mmHg or target BP < 160/90 mmHg) using continuous or intermittent IV medications to control BP with frequent monitoring of BP and neurologic examination.

The results of two early phase clinical trial of blood reduction in acute ICH have recently been published, INTERACT and ATACH. INTERACT was a multi-center randomized prospective trial which demonstrated that intensive lowering of SBP to goal < 140 as opposed to SBP to goal < 180 decreased the absolute risk of significant hematoma growth (defined as ≥ 33% of baseline hematoma volume) by 8% without increasing the rate of adverse events (26), a larger clinical trial based upon these results is underway to test whether this lower SBP goal can improve clinical outcomes.(27) Another recently published dose escalation study (ATACH) evaluated the tolerability and safety of targeting 3 different BP goals (SBP 170-200, SBP 140-170, SBP 110-140) using nicardipine infusion found that patients tolerated acute lowering of SBP to the three tiers without significant differences in neurologic deterioration between the three tiers (28).

While the choice of BP lowering agent should be individualized based on factors such as heart rate and medical comorbidities (e.g. renal or heart failure), our usual preference is to use agents that preferentially affect cardiac output or are arterial vasodilators such as bolus doses of intravenous labetalol or continuous intravenous infusion of nicardipine. We try to avoid medications which might cause significant venodilation such as hydralazine or nitroprusside. INTERACT and ATACH have now been expanded to pivotal phase III clinical trials to test whether aggressive BP treatment limits hematoma expansion and improves clinical outcome after ICH (29).

Coagulopathy

ICH is more frequent in patients treated with anticoagulants and fibrinolytics, and the risk of warfarin-related ICH increases with increasing INR. Warfarin-related ICH is associated with an even higher rate of mortality than ICH in the absence of coagulopathy and ongoing bleeding in warfarin-related ICH continues for a more prolonged duration (30, 31).
The obvious goal is to urgently reverse the coagulopathy as soon as possible. While this has historically been done using vitamin K and fresh frozen plasma (FFP), it is now recognized that this approach is suboptimal and often leads to excessively slow correction or failure to correct the coagulopathy entirely (32). Current guidelines (33) recommend the use of vitamin K 5-10 milligrams usually administered intravenously by slow push and concurrent treatment with a more rapidly acting reversal agent as it usually takes hours after vitamin K administration for reversal of warfarin-induced coagulopathy. Full warfarin correction usually necessitates the administration of large volumes of FFP and the logistics surrounding cross-matching, thawing, and infusion rates makes this generally a slower option for correction. Consequently, recent interest has turned to the use of concentrated factor preparations such as prothrombin complex concentrate (PCC) or hemostatic agents such as recombinant Factor VIIa. Prothrombin complex concentrate administration generally reverses an elevated INR more rapidly than FFP (34) and consequently may be more advantageous in limiting hematoma growth due to ongoing warfarin-related coagulopathy. However, in a study comparing PCC and FFP, there was no difference in hematoma growth between FFP and PCC in patients whose INR was corrected within 2 hours (35). This strongly suggests that it is timing of coagulopathy reversal, not a specific agent, that makes the difference. Various current guidelines for warfarin-reversal in the setting of life-threatening hemorrhage now emphasis the use of a rapid reversal agent such as PCC or recombinant Factor VIIa in addition to Vitamin K (33, 36, 37).

Hemostatic Agents

The recognition that hematoma expansion worsens outcome and is common even in the absence of coagulopathy has generated significant interest in the potential use of hemostatic agents to limit hematoma growth. Developed as an agent for the treatment of a subset of hemophilic patients, recombinant Factor VIIa (rFVIIa) has now been investigated in a wide range of bleeding disorders in patients with normal coagulation, including ICH (38). In a phase IIa trial, 399 acute ICH patients who had initial CT diagnosis within 3 hours of symptom onset received either placebo or one of three doses of recombinant Factor VIIa (40, 80, or 160 μg/kg) within one hour of CT scan. Overall, patients who received rFVIIa had less hematoma expansion and this translated to a lower risk of mortality and improved functional outcome, despite a small increase in thrombotic events such as myocardial infarction (39). Given these encouraging results, a larger phase III trial including 821 patients was conducted with essentially the same inclusion criteria, but comparing placebo and two doses of rFVIIa (20 and 80 μg/kg). In this pivotal phase III trial, hematoma expansion was once again dramatically reduced by treatment with rFVIIa. However, there was no statistically significant change in the proportion of patients who died or were severely disabled (40). Post-hoc analysis suggested that the subset of the study population who were ≤ 70 years old and who had baseline ICH volumes < 60 mL, intraventricular hemorrhage volume < 5 mL, and time from onset-to-treatment < 2.5 hours may have clinically benefited from being administered the drug (41). However, this should be considered as an exploratory analysis for identifying a target population for an additional clinical trial. At present hemostatic therapy cannot be recommended as routine treatment for ICH patients without coagulopathy.
Antiplatelet agents and ICH

There are conflicting reports as to the role of prior antiplatelet therapy on hematoma expansion and outcome for patients presenting with ICH (1, 42, 43). Consequently, there is wide heterogeneity in clinical practice ranging from practitioners who advocate platelet transfusion in patients with ICH while taking antiplatelet agents such as aspirin or clopidogrel, to those who advocate the use of laboratory tests for platelet function, to those who choose not to treat. Evaluation of the placebo group from a neuroprotective ICH study did not find an association between antiplatelet use and hematoma expansion or outcome.(44) In contrast, recently published work on antiplatelet use and platelet function has suggested that the results of platelet activity assays (but not merely the history of aspirin usage) correlated with occurrence of IVH, a greater ICH Score, hematoma growth, and worse outcomes in ICH.(45-47) Given the widespread use of antiplatelet agents, further clarification of the impact of antiplatelet use and platelet dysfunction on ICH occurrence, growth, and outcome is an important future direction.

Intensive Care Management

Intracranial Pressure

Patients with moderate or large ICH or intraventricular hemorrhage often have increased intracranial pressure (ICP) or hydrocephalus that warrants consideration of treatment. The AHA/ASA guidelines advocate a graded stepwise approach with initial routine use of less invasive measures prior to instituting more invasive measures. These less invasive measures include elevation of the head of the bed to 30 degrees, maintenance of the neck in a neutral position to facilitate jugular venous drainage, and adequate analgesia and sedation. More invasive measures include CSF drainage via an extraventricular drain (EVD) placed directly into the ventricles. An EVD allows continuous measurement of intracranial pressure as well as drainage of CSF to treat elevated ICP, but does carry a small risk of hemorrhage or infection. Osmotic agents such as mannitol and hypertonic saline may be used to decrease ICP, but overuse of mannitol may cause hypovolemia, renal failure, and cerebral vasoconstriction. Neuromuscular blockade may also be considered in patients with refractory elevated ICP but is likely associated with an increased risk of infection and critical illness neuromuscular disease. While hyperventilation may rapidly reduce elevated ICP by causing cerebral arterial vasoconstriction, this effect is generally transient (few hours) and reduces cerebral blood flow which might potentially engender secondary brain injury. Thus, hyperventilation is usually used as a temporizing measure in preparation for other more definitive medical or surgical treatments. Finally, barbiturate coma may be considered in patients that have failed other therapies but is associated with a significant risk of hypotension and requires continuous electroencephalographic monitoring to titrate effective dosing. Induced hypothermia to 32 to 34 degrees Celsius may also be attempted for a brief period, but is associated with a high rate of complications. The use of barbiturate coma and induced hypothermia have not been systematically investigated in ICH and are presently considered salvage second-tier therapies.
Fever, Glucose, DVT Prophylaxis

Fever is a common occurrence in patients with intracerebral hemorrhage and increased fever duration is associated with poor outcomes. Thus, fever should be aggressively treated even as appropriate testing for systemic infection is being undertaken. Hyperglycemia on admission is predictive of 14-day and 28-day mortality in patients with ICH. Intensive insulin therapy treatment of hyperglycemia during critical illness has been shown to decrease systemic morbidity and mortality as well as decrease the incidence of critical illness polyneuropathy (48). Thus, it is reasonable to vigilantly avoid hyperglycemia in patients with ICH and to institute aggressive approaches to achieve normoglycemia. Even so, randomized trials specifically in patients with ICH have not been performed and concerns have been raised about the possibility of hypoglycemic episodes and their particularly detrimental effects in patients with brain injury. Deep venous thrombosis (DVT) and pulmonary embolism are frequent in patients presenting with ICH, and DVT is diagnosed in approximately 2% of patients during their acute hospitalization. In one study, the combination of compression stockings plus intermittent pneumatic compression decreased the rate of asymptomatic DVT detected at day 10 from 15.9% to 4.7% in patients with ICH (49). The other intervention study published to date in patients specifically with ICH examined the initiation of low-dose subcutaneous heparin for DVT prophylaxis (50). Unfractionated heparin 5000 Units three times a day was initiated on the second, fourth or tenth day following presentation with ICH. There was a statistically significant decrease in the incidence of pulmonary embolism in those patients in whom heparin was started on the second day when compared with the other groups, and importantly there was no increase in intracranial rebleeding. This small study suggests that low-dose subcutaneous heparin can be started as early as the second day in patients who present with ICH and that it may decrease the incidence of pulmonary embolism without profoundly increasing the risk of hematoma expansion or new ICH during hospitalization.

Anticonvulsants

Clinically apparent seizures occur in 4-8% of patients with ICH within the first 30 days. Lobar ICH location increases the likelihood of seizures. Additionally, non-convulsive seizures identified on continuous EEG (cEEG) monitoring may occur in about 20% of ICH patients and have been suggested to be associated with worsened outcome and other parameters such as midline shift and higher NIH Stroke Scale scores. Despite this, it is unknown whether anticonvulsant prophylaxis reduces the incidence of clinically overt or non-convulsive seizures after ICH. In fact, evaluation of the placebo group from a neuroprotective trial of ICH treatment found that those patients who received prophylactic antiepileptic treatment had a worsened outcome even after adjusting for other factors (51). Current guidelines recommend against the use of routine anticonvulsant prophylaxis, but do recommend treatment of seizures if they occur (1).
Surgery

The decision to undertake surgical evacuation of the hematoma in spontaneous ICH remains controversial and fraught with clinical uncertainty, being still significantly influenced by the bias of practitioners and consultants caring for the patient (52). Until recently there had only been a few small mostly single center trials, the preponderance of which did not favor a mandatory approach of craniotomy for evacuation of the hematoma in ICH. These prior trials set the stage for a landmark study entitled the International Surgical Trial in Intracranial Haemorrhage (STICH) (53). This international, multi-center trial randomized 1033 patients presenting within 72 hours of ictus of spontaneous supratentorial ICH in which the local neurosurgeon decided that there was clinical equipoise about whether or not the patient would benefit from surgery. The patient was randomized to either early surgical intervention (within 24 hours of randomization) or initial medical management. The primary outcome measure was death and disability as measured by the extended Glasgow Outcome Score (GOS) at 6 months; different outcome cutpoints were used depending on expected prognosis from the initial hemorrhage. Method of hematoma evacuation and medical management were left to the discretion of local treating physicians and outcomes were assessed using questionnaires sent to the patients or their families. 506 patients were randomized to early surgical intervention; 530 were randomized to initial medical management but 26% of these patients ultimately underwent surgery for hematoma evacuation (mostly due to neurologic deterioration). In an intention-to-treat analysis, early surgery was neither beneficial nor harmful as there was no statistically significant difference in either mortality or functional outcome. Given the design limitations, the results of STICH cannot be used to conclude that surgical evacuation has no role in supratentorial ICH. However, it does demonstrate that a large scale surgical ICH trial can be successfully completed and that early surgery is unlikely to be a panacea for most patients. Of note, pre-specified subgroup analyses identified that subjects with hematomas < 1 cm from the cortical surface and subjects who underwent craniotomy as the surgical procedure had a nonsignificant trend towards benefit with early surgery. Based upon the results of the subgroup analysis, a second international multi-center trial (STICH II) is currently underway to test early hematoma evacuation versus initial conservative management in patients with lobar hematoma 1 cm or less from the cortical surface (http://www.ncl.ac.uk/stich/).

There are a number of case series which report that patients with spontaneous cerebellar hemorrhage who present with large cerebellar hematomas (> 3 cm in diameter) or with compression of the brain stem or hydrocephalus may still have a favorable outcome with surgical intervention. However, there has not been a prospective randomized trial of surgery for cerebellar ICH analogous to STICH. Even so, cerebellar ICH is generally considered as a potentially surgical lesion by most neurologists and neurosurgeons, especially in patients with obstructive hydrocephalus or clinical deterioration. The 2010 AHA/ASA ICH management guidelines recommend surgical removal of the hematoma as soon as possible in patients with cerebellar hemorrhage who are deteriorating neurologically or who have brain stem compression and/or hydrocephalus from ventricular obstruction (1).
A number of minimally invasive surgical alternatives to open craniotomy have been also been considered and studied in small case series or pilot clinical trials. These techniques include: simple aspiration of the hematoma; mechanical aspiration with a screw and suction technique; instillation of a thrombolytic such as urokinase or recombinant tissue plasminogen activator into the hematoma with aspiration of contents; and endoscopic aspiration of the hematoma with lavage of the hematoma cavity and photocoagulation of oozing vessels. An NIH sponsored multicenter trial is currently underway comparing catheter directed t-PA treatment for hematoma evacuation versus conventional medical management for patients presenting with ICH (http://mistietrial.com/default.aspx). Another phase III trial (CLEAR III) is evaluating the effectiveness of catheter-directed t-PA for the treatment of intraventricular hemorrhage (http://biosgroup–johnshopkinsmedicine.health.officelive.com/default.aspx).

REFERENCES


**TABLE 1. Etiology of Intracerebral Hemorrhage**

**Primary ICH**
- Hypertension
- Cerebral Amyloid Angiopathy
- CADASIL
- Coagulopathy
- Sympathomemetic Drug Abuse (phenylpropylalimine, cocaine, amphetamines)

**Secondary ICH**
- Vascular Malformations
  - Arteriovenous Malformations
  - Cavernous Malformation
  - Intracranial Aneurysms
- Dural fistualas
- Mycotic Aneurysm
- Moya Moya Disease

- Ischemic Stroke
  - Hemorrhagic Conversion of an Ischemic Stroke
  - Dural/Cortical Sinus Thrombosis

- Trauma
- Tumor associated ICH (Primary or metastatic)
- Vasculitis
**TABLE 2. The ICH Score (17)**

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<td><strong>Total ICH Score</strong></td>
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*GCS = GCS score on initial presentation (or post-resuscitation); ICH Volume on initial CT scan calculated using ABC/2 method; IVH = presence of any intraventricular hemorrhage on initial CT*