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Emergency Neurological Life Support: Intracerebral Hemorrhage

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Abstract Intracerebral hemorrhage (ICH) is a subset of stroke due to spontaneous bleeding within the parenchyma of the brain. It is potentially lethal, and survival depends on ensuring an adequate airway, proper diagnosis, and early management of several specific issues such as blood pressure, coagulopathy reversal, and surgical hematoma evacuation for appropriate patients. ICH was chosen as an Emergency Neurological Life Support (ENLS) protocol because intervention within the first hours may improve outcome, and it is critical to have site-specific protocols to drive care quickly and efficiently.

Keywords Intracerebral hemorrhage · Blood pressure · Hematoma · Coagulopathy · Surgery

Introduction

Intracerebral hemorrhage (ICH) results from spontaneous direct bleeding into the brain. In the U.S., ICH accounts for 10–15% of all strokes, but it carries a disproportionately high risk of death or long-term disability. It is considered an acute neurological emergency because of the potential to treat or mitigate injury, and the risk of ongoing secondary brain injury.

The availability of treatments proven to benefit ICH patients has lagged behind that of ischemic stroke and aneurysmal subarachnoid, and this has resulted in variability in care that ranges from aggressive treatment to a nihilistic approach. Guidelines exist for the management of ICH, and the purpose of this ENLS protocol is to emphasize initial management, with the goal of optimizing recovery. Acknowledging that there is variability in the strength of evidence for treatment recommendations for certain interventions, aggressive initial care of the ICH patient is recommended, in accordance with existing guidelines [1, 2].

Management of the ICH patient during the initial "golden hour" emphasizes the following aspects:

1. Stabilization and reassessment of the patient's airway, breathing, and circulation (ABC's)
2. Rapid and accurate diagnosis using neuroimaging
3. Concise clinical assessment regarding ICH characteristics and patient condition
4. Targeted assessment for potential early interventions including:
   a. Control of elevated blood pressure
   b. Correction of coagulopathy
   c. Need for early surgical intervention
5. Anticipation of specific patient care needs such as:
   a. Specific treatment aspects related to underlying ICH cause
   b. Risk for early clinical deterioration and hematoma expansion
   c. Need for intracranial pressure (ICP) or other neuromonitoring

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d. Patient disposition from the emergency department (ED)

The ENLS suggested algorithm for the initial management of ICH is shown in Fig. 1. Suggested items to complete within the first hour of evaluating a patient with ICH are shown in Table 1

Diagnosis

ICH may result from a variety of underlying etiologies. Rupture of a small arteriole due to chronic hypertension accounts for approximately 60% of cases. Other common causes include cerebral amyloid angiopathy, coagulopathy due to treatment with antithrombotic medications, sympathomimetic drugs such as cocaine, and underlying vascular anomalies such as arteriovenous malformations (AVMs) or cavernous malformations. Less common causes include cerebral vasculitis, Moya–Moya syndrome, and rupture of a saccular or mycotic aneurysm. Secondary hemorrhagic transformation of an arterial or venous infarct may also occur.

Most patients with acute ICH develop the sudden onset of a focal neurological abnormality. Without neuroimaging, the ICH neurological syndrome often cannot be reliably distinguished from an acute ischemic stroke. Headache, progressive neurological signs and symptoms, acute severe hypertension, and decreased level of consciousness occur more frequently in ICH than in ischemic stroke.

The initial prehospital and ED resuscitation is similar across stroke subtypes, with rapid neuroimaging being essential to diagnosis. Because treatments for ICH and acute ischemic stroke are different, ICH-specific interventions are not provided until the diagnosis is made. Thus, prehospital care focuses on management of the ABCs and rapid transport to a designated stroke receiving hospital. Non-contrast computed tomography (CT) is the most commonly used modality given that it can be done quickly, can be used for critically ill patients, and has a very high sensitivity and specificity for acute parenchymal hemorrhage. Magnetic resonance imaging (MRI) may have a similar sensitivity to identify ICH, but logistics related to availability and the clinical condition of the patient limits its use as a primary modality [3, 4].

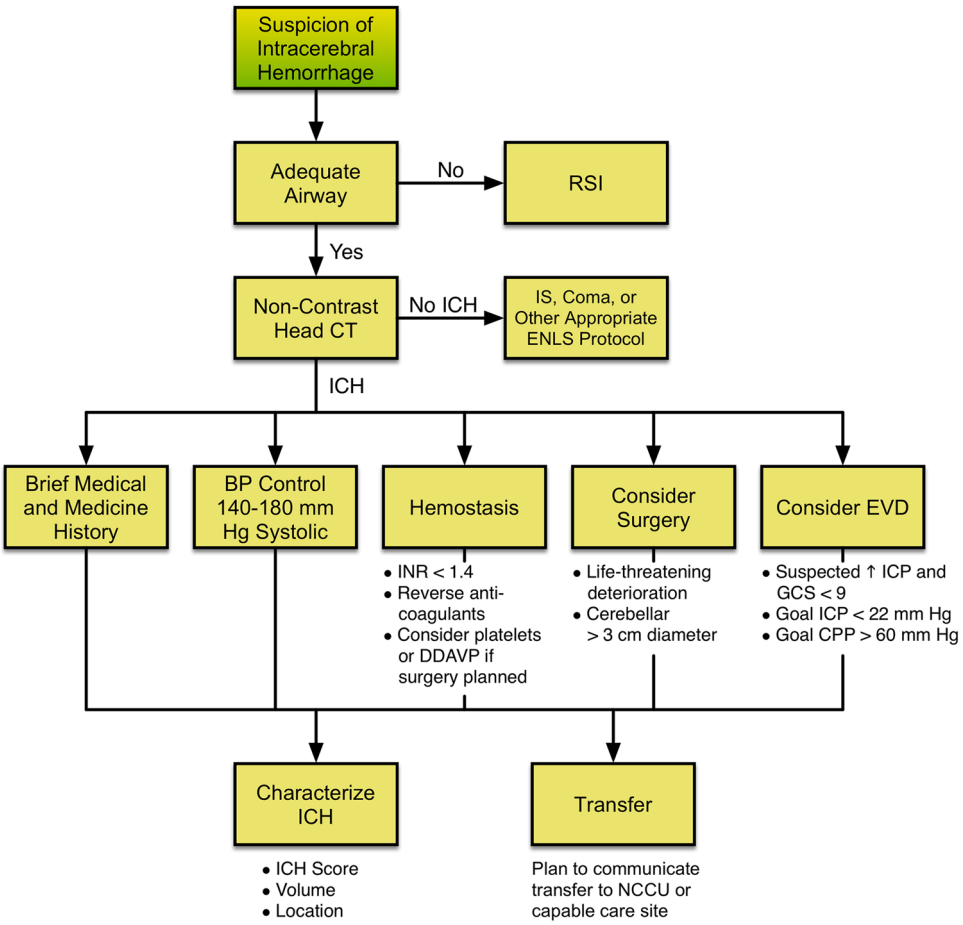

Fig. 1 ENLS Intracerebral Hemorrhage protocol

Table 1 Intracerebral hemorrhage checklist for the first hour

Intracerebral hemorrhage checklist for the first hour
☐ Complete blood count with platelet count, PT, PTT, INR
☐ Head imaging results: hematoma size, location, presence of intraventricular hemorrhage
☐ Glasgow Coma Scale (GCS) score
☐ Calculate ICH Score
☐ Interventions:
☐ Coagulopathy reversal (goal INR <1.4)
☐ Blood pressure lowering (goal systolic 140–180 mmHg)
☐ Surgical hematoma evacuation (if indicated)
☐ Airway/ventilation management

Interpreting the ICH CT Scan: Location, Volume, and Spot Sign

ICH tends to occur in characteristic locations, with hypertensive ICH most frequently located in the basal ganglia, thalamus, pons (brainstem), and cerebellum. ICH due to cerebral amyloid angiopathy or AVM tends to have a lobar location. The origin of the hematoma is usually evident from the initial CT scan, and its location influences outcome and treatment (Fig. 2).

While ICH location is important, ICH hematoma volume is a stronger predictor of patient outcome. The ability to calculate hematoma volume quickly from the initial CT scan is an advantage in directing communication and treatment decisions. Automated CT software algorithms can be used to calculate hematoma volume. However, the manual ABC/2 formula, which approximates the volume of an ellipsoid, is simple and reasonably accurate compared to computerized methods [5].

When using the ABC/2 method for calculating volume, the axial CT image is selected with the largest cross sectional area of hemorrhage. Measure the largest hemorrhage diameter (A). Next, perpendicular to this line, measure the largest hemorrhage diameter on the same image (B). Then, multiply the total number of CT slices with hemorrhage by the slice thickness to obtain (C). For (C), if the hematoma area on a slice is approximately 25–75% of the hematoma area on the reference slice used to determine (A), then this slice is considered half a hemorrhage slice, and if the area is less than 25% of the reference slice, the slice is not considered a hemorrhage slice [5]. Alternately, (C) can be assessed by measuring the largest diameter, superior to inferior, that is seen on coronal or sagittal images. Multiply (A) times (B) times (C), then divide by 2 in order to obtain the hematoma volume. Figure 3 demonstrates an example.

Many ICH patients experience hematoma growth after initial presentation, and the ability to anticipate expansion is desirable, as expansion is associated with worse clinical outcome [6]. Several retrospective reports have suggested that the use of intravenous (IV) contrast administration during the initial CT scan may identify extravasation into the hematoma and that this "spot sign" (contrast within the hematoma) is predictive of hematoma growth (Fig. 4) [7–9].

Thus, the use of a "stroke CT" that includes non-contrast CT as well as CT angiography (and possibly CT perfusion and post-contrast images) may be considered in patients with acute ICH in order to detect a "spot sign," as well as to reveal an underlying vascular anomaly. Ongoing studies are seeking to use the "spot sign" as a way to identify those at risk for hemorrhage expansion and to determine if hemostatic agents may benefit these specific high-risk patients.

Management

Initial Patient Assessment and Primary Intervention: ABCs and the ICH Score

As with all emergency medical care, initial assessment of the ABCs is critical. Until the diagnosis of ICH is made from neuroimaging, overall airway and hemodynamic management proceeds in a common pathway with other stroke subtypes. However, immediately following the ICH diagnosis, disease-specific treatment can be instituted.

Because many ICH patients are obtunded or comatose, airway management (specifically the need for intubation for airway protection) should be considered throughout the early treatment course. Thus, while "Airway" is listed under secondary treatment in the ENLS ICH protocol (Fig. 1), it is concurrent with the initial evaluation. In general, if an ICH patient is comatose, rapid sequence intubation (RSI) should be undertaken, with a goal of normoventilation (see the ENLS *Airway, Ventilation, and Sedation protocol).*

An initial clinical assessment of the patient's condition and stroke severity is essential to rapid treatment planning and communication among providers. While performance of a complete, detailed neurological examination is ideal,

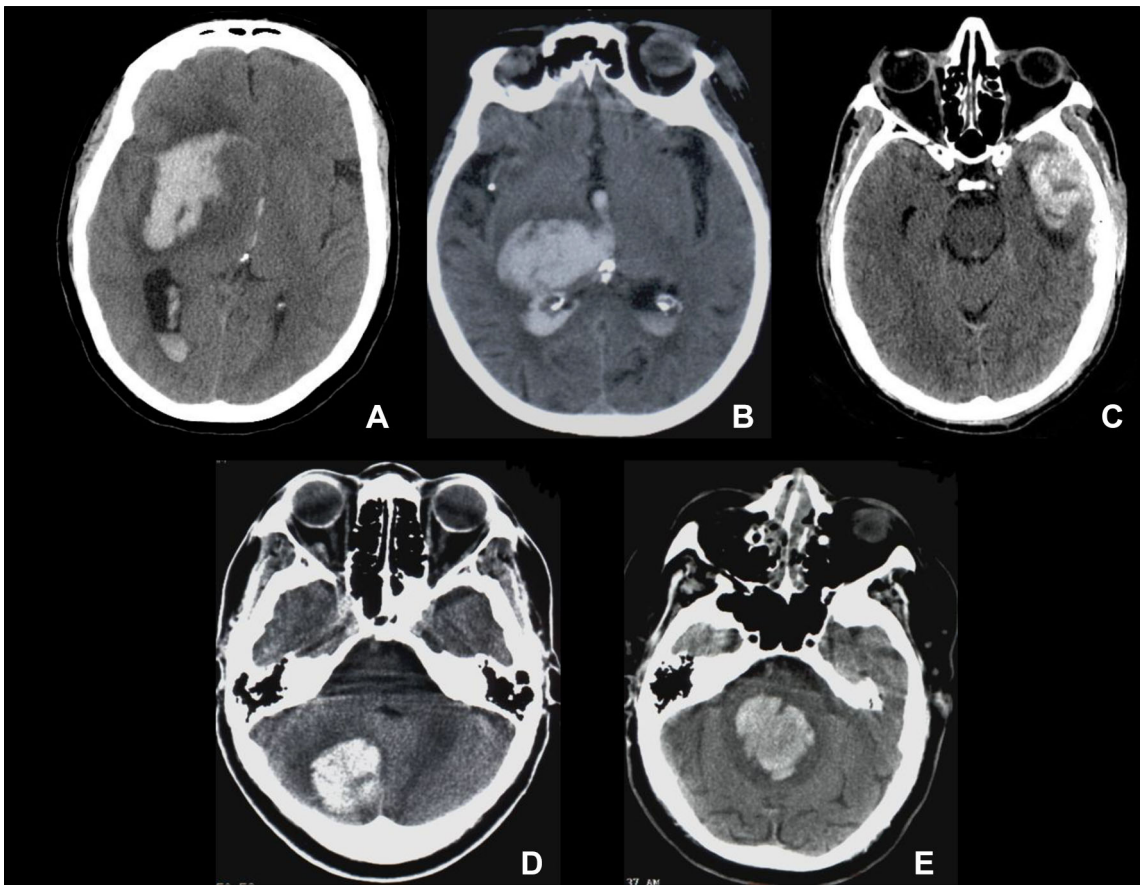

Fig. 2 Typical locations for intracerebral hemorrhage (ICH). ICH due to chronic hypertension is usually due to rupture of small penetrating arterioles and typically occurs in the basal ganglia (**a**), thalamus (**b**), cerebellum (**d**), and pons (**e**). ICH from cerebral amyloid angiopathy and sympathomimetic drugs of abuse such as cocaine or methamphetamine often occurs in lobar regions such as the temporal lobe (**c**). Supratentorial ICH would be considered as basal ganglia, thalamic, or lobar (**a**–**c**), whereas ICH originating in the cerebellum or pons would be considered infratentorial (**d**, **e**). **a**, **b**, and **e** also demonstrate IVH

much information can be gleaned from a quick assessment using existing clinical grading scales. The ICH Score is the most commonly used validated clinical grading scale for patients with intracerebral hemorrhage, combining elements related to patient demographics, clinical condition, and neuroimaging findings that are readily available at the time of hospital admission [10, 11]. Several other useful clinical grading scales are also available [12–14].

Components of the ICH Score include age, initial Glasgow Coma Scale (GCS) score, ICH hematoma volume, ICH hematoma location (supratentorial or infratentorial), and presence of IVH. Table 2 demonstrates the components of the ICH score, with the full score being the sum of points given for each component. Each point increase in the ICH Score is associated with an increased risk of mortality and a decreased likelihood of good functional outcome. The ICH Score is best used as a communication tool among providers and with patients or family members regarding a patient's condition rather than as a tool to precisely prognosticate outcome. While it is tempting to utilize clinical grading scales to triage severely impaired patients toward less-aggressive intervention, this approach is not recommended. Rather, in general, initial aggressive therapy is recommended in order to avoid the potential for a self-fulfilling prophecy of poor outcome in the context of early care limitations [1, 15, 16].

Primary Intervention: Blood Pressure, Coagulopathy, and Surgery

Following the diagnosis of ICH, immediate consideration should be given to the need for (a) acute control of elevated blood pressure, (b) correction of coagulopathy due to medications or underlying medical conditions, and (c) the need for urgent surgical hematoma evacuation. These are common themes that should form part of the initial ICH evaluation and treatment plan. Decisions regarding these interventions will influence the succeeding aspects of ICH

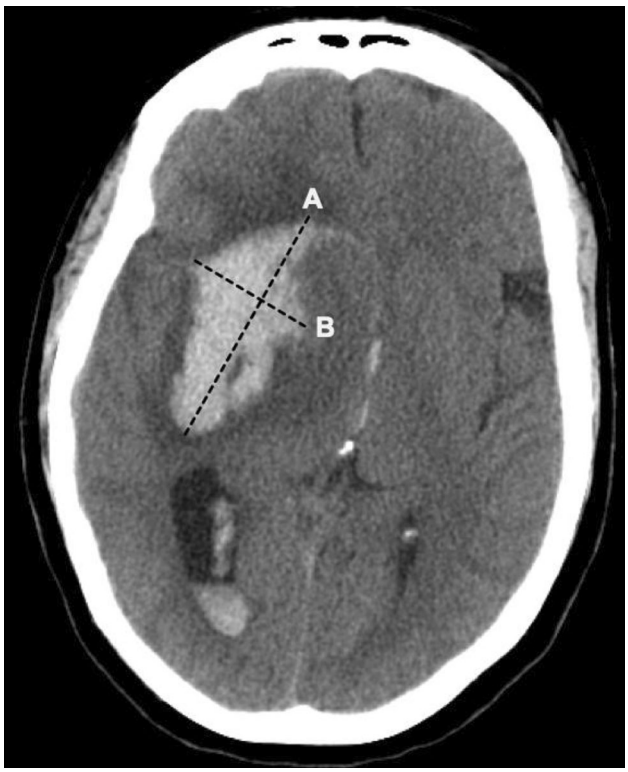

Fig. 3 ABC/2 method for estimating ICH hematoma volume [5]. Right basal ganglia intracerebral hemorrhage. The axial CT image with the largest cross sectional area of hemorrhage is selected. In this example, the largest diameter (*A*) is 6 cm, the largest diameter perpendicular to (*A*) on the same image (*B*) is 3 cm, and hemorrhage is seen on 6 slices of 0.5 cm (5 mm) thickness for a (*C*) of 3 cm (not shown). Thus, the hematoma volume is $(6 \times 3 \times 3)/2 = 27$ cc. Note that for (*C*), if the hematoma area on a slice is approximately 25–75% of the hematoma area on the reference slice used to determine (*A*), then this slice is considered half a hemorrhage slice, and if the area is less than 25% of the reference slice, the slice is not considered a hemorrhage slice

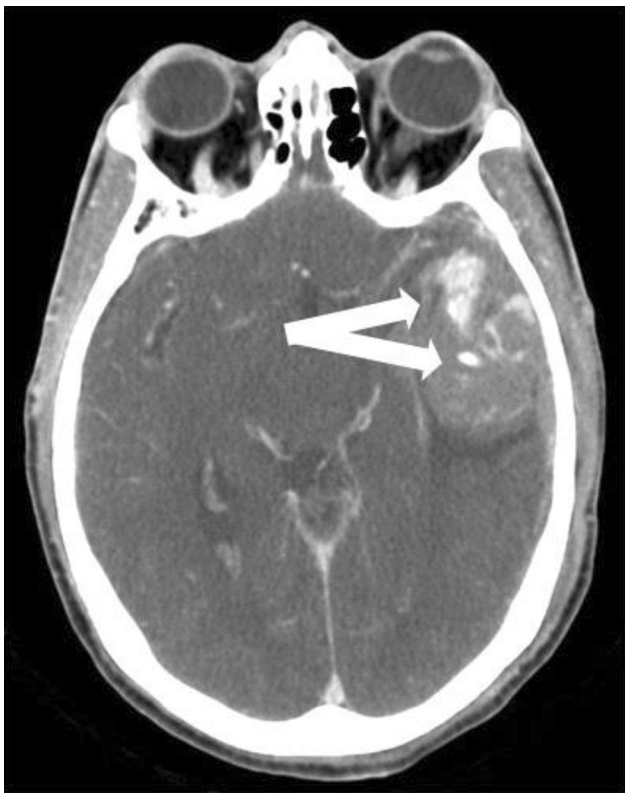

Fig. 4 Contrast extravasation ("spot sign") in acute ICH. In this post-contrast image obtained after administration of IV contrast during a "code stroke" CT (non-contrast study, CT angiogram, CT perfusion study), contrast extravasation is present in this acute left temporal lobe ICH. This finding is commonly referred to as a "spot sign" (*arrows*) and is associated with increased risk of hematoma expansion

care, such as disposition from the ED, planning for repeat imaging, and need for ICP monitoring or continuous electroencephalography (cEEG).

Hematoma expansion is common in patients with acute ICH, and this is associated with worsened outcomes [6, 17]. Though the pathophysiology that leads to hematoma expansion is incompletely understood, it tends to occur early (within a few hours of onset) and coagulopathy increases the frequency of its occurrence and its extent [18]. However, hematoma expansion is common even in patients without coagulopathy or who are not receiving antithrombotic medications. Thus, intervention to address treatable aspects should not be delayed pending patient disposition.

Blood Pressure

Elevated blood pressure is extremely common in patients with acute ICH. While it seems intuitive that elevated blood pressure may predispose to hematoma expansion due to increased bleeding or to elevated ICP from worsening edema, clinical studies have had conflicting results regarding the impact of acutely elevated blood pressure and the value of acutely lowering the blood pressure [19, 20]. There has been a concern that acutely lowering blood pressure could lead to ischemic brain injury in the perihematoma region, but this risk has not been supported by recent studies [21, 22].

While blood pressure management has remained controversial, current approaches favor rapid lowering of moderately elevated blood pressures [1, 2]. Two pilot randomized clinical trials, INTERACT and ATACH, suggested that acutely lowering systolic blood pressure to below 140 mmHg is safe [23, 24]. These were followed by pivotal phase III efficacy trials to test the impact of blood pressure lowering on clinical outcome. INTERACT2 was a

Table 2 The ICH Score [10]

Component	ICH Score Points
Glasgow Coma Scale	
3–4	2
5–12	1
13–15	0
ICH volume (cc)	
≥30	1
<30	0
Presence of IVH	
Yes	1
No	0
Infratentorial origin of ICH	
Yes	1
No	0
Age (years)	
≥80	1
<80	0
Total ICH Score	0–6

phase III clinical trial of acute blood pressure lowering in ICH patients presenting with a systolic blood pressure between 150 and 200 mmHg [25]. Patients were randomized to two different systolic blood pressure thresholds: a standard threshold of less than 180 mmHg and an intensive threshold of less than 140 mmHg for the initial seven days after ICH occurrence. Patients in the intensive arm had modestly better outcomes with about 3% fewer patients having death or severe disability (defined as a modified Rankin Scale score of 3 to 6). Interestingly, there was no difference in hematoma expansion between groups. ATACH 2 had a similar design and tested the two systolic blood pressure thresholds of 180 and 140 mmHg for the initial 24 h after ICH using the titratable intravenous agent nicardipine [26]. ATACH 2 did not demonstrate a difference in outcome between treatment groups.

Both the current American Heart Association/American Stroke Association Guidelines for the Management of Intracerebral Hemorrhage and the guidelines from the European Stroke Organization recommend a target blood pressure of less than 140 mmHg in patients like those studied in INTERACT2 [1, 2]. The most recent version of both of these evidence-based guidelines were developed after the publication of INTERACT 2 and prior to the completion of ATACH 2. Given these new clinical trial results, it may be reasonable to target a systolic blood pressure between 140 and 180 mmHg with the specific threshold determined based on patient comorbidities and level of chronic hypertension. Although the clinical difference between these two systolic blood pressure thresholds may be modest and debatable, none of the current guidelines recommend allowing blood pressure to remain extremely elevated without treatment [1, 2]. Acute lowering of blood pressure is reasonable in patients presenting with more extreme levels of hypertension, but less is known about the specific safety and efficacy of treatment [1].

Basic principles of blood pressure lowering in ICH are that management should be initiated immediately and a titratable agent should be used to ensure that the target value is reached quickly and with minimal potential for overshoot. IV beta-blockers and calcium-channel blockers are the most commonly used medications for this indication in the ED and the intensive care unit (ICU).

Labetalol is rapid acting, has mixed alpha and beta adrenergic antagonism, and is commonly used in the ED in an initial bolus dose of 5–20 mg. Nicardipine is a calcium channel blocker of the dihydropyridine family that is more selective for coronary and cerebral vascular beds. A common initial nicardipine dose of 5 mg/hr is often used, with titration up every 15 min as needed. Clevidipine is another calcium channel blocker that acts even more rapidly than nicardipine. If possible, nitroprusside should be avoided due to its potential for cerebral vasodilation, disturbed cerebral autoregulation, and elevated ICP. ICU admission is recommended, due to the close monitoring and frequent medication changes required to lower blood pressure. A more detailed discussion of common anti-hypertensive medications utilized in neurologic emergencies can be found in the *Pharmacotherapy* Module.

Coagulopathy: Anticoagulants, Antiplatelet Agents, and Heparin

The use of antithrombotic medications for prevention and treatment of ischemic stroke, cardiovascular disease, and systemic venous thromboembolism is common and is increasing as the population ages. Antithrombotic medications are a risk factor for the occurrence of ICH, as well as for hematoma expansion if an ICH occurs. Given the range of antithrombotic medications, including warfarin, heparin, antiplatelet agents such as aspirin and clopidogrel, and newer agents such as dabigatran, rivaroxaban and apixaban, the specific risks and interventions to reverse coagulopathy vary. Additionally, coagulopathies may be due to underlying medical conditions, such as liver disease or hematologic malignances.

The second focus in ICH is on treatment of coagulopathy. As part of the initial evaluation of the ICH patient, a medical history and medication list should be obtained from the patient, family, prehospital providers, or medical record; specifically the use of antithrombotic medication and, if possible, when the last dose was taken should be noted. Urgent laboratory tests should include a complete

blood count (CBC) with platelet count, an international normalized ratio (INR), and a partial thromboplastin time (PTT). A general principle is that any ICH occurring in a patient on antithrombotic medications should be considered life-threatening due to the risk of hematoma expansion. Interventions to treat coagulopathy are based on this history and laboratory information more than on size or location of the hematoma or clinical scores.

Patients taking a vitamin K antagonist such as warfarin and whose INR is > 1.4 should receive agents to normalize the INR to 1.4 or below. Options have included the administration of fresh frozen plasma (FFP), vitamin K, prothrombin complex concentrates (PCC), and the hemostatic agent recombinant Factor VIIa (rFVIIa) although PCC is now the recommended approach [27, 28]. The most important principle is to normalize the INR as soon as possible, ideally within minutes.

While FFP is widely used for reversing the effect of warfarin, it may not be optimal in other medical conditions. FFP contains factors I (fibrinogen), II, V, VII, IX, X, XI, XIII, and antithrombin. Fairly large volumes of FFP (10–15 ml/kg) are often required for full reversal of anticoagulation, and this places patients at risk for volume overload and pulmonary edema [29]. FFP, like other blood products, also carries a risk for transfusion related events and requires thawing after cross-matching by a blood bank.

PCCs contain factors II, IX, X (and varying amounts of VII, depending on the specific preparation) with much higher concentrations of clotting factors in smaller amounts of volume than FFP. The term 4-factor PCC is used to designate that sufficient concentrations of factors II, VII, IX, and X are present in the specific preparation. PCCs can correct the INR within minutes, faster than FFP, and with fewer cardiopulmonary complications [30]. In a prior observational study comparing PCC and FFP, there was no difference in hematoma growth in patients whose INR was corrected within 2 h [31], suggesting that the timing of coagulopathy reversal, not the specific agent, makes the greatest impact. However, the recent INCH clinical trial demonstrated the superiority of 4-factor PCC in a dose of 30 IU/kg over FFP 20 ml/kg in rapidly reversing an elevated INR to ≤ 1.2 in 54 ICH patients on a vitamin K antagonist [32]. Furthermore, hematoma expansion was less in patients receiving PCC. There was a trend for lower mortality and better functional outcome in the PCC treated patients; however, the study was stopped early by its regulatory oversight body because of the finding of less hematoma expansion in the PCC group and therefore was underpowered to formally assess a clinical outcome difference.

The most recent guidelines recommend weight-based dosing for PCC (or FFP only if PCC is not available) with the dose adjusted based on INR [28]. However, the specific dose may vary based on the PCC formulation used at a specific hospital. Information on reversal of warfarin, direct thrombin inhibitors, and factor –Xa inhibitors may be found in the *Pharmacotherapy* module. Current guidelines [1, 28] recommend the use of vitamin K 10 mg administered intravenously by slow push, in conjunction with another more rapidly acting agent (e.g. PCC), as it typically takes hours after vitamin K administration for reversal of warfarin-induced coagulopathy, but it has a more long-lasting effect than PCC or FFP [27].

While rFVIIa also quickly reverses an elevated INR, this may reflect a specific effect on the INR laboratory test and a clinically important coagulopathy may remain. rFVIIa has been shown to decrease hematoma growth in non-coagulopathic ICH patients, but this did not translate into improved clinical outcome [33]. Thus, rFVIIa is not recommended for use in ICH patients with or without warfarin-related coagulopathy [1]; however, it is occasionally used in patients with coagulopathy related to liver failure.

Observational studies have varied regarding the impact of concurrent antiplatelet therapy on hematoma expansion and outcome for patients presenting with ICH, though increased risk of hematoma growth while on these agents is suggested [34–37]. There has been heterogeneity in clinical practice, ranging from the empiric use of platelet transfusions, to determining the need for transfusion by laboratory tests for platelet function, to complete avoidance of platelet treatment. The PATCH study was an open-label clinical trial testing the efficacy and safety of platelet transfusion in patients with ICH occurring while on an antiplatelet agent for at least a week [38]. Platelet transfusions did not improve outcome and were associated with a significant increase in risk of death and more adverse events. Thus, platelet transfusion is not recommend for most patients with ICH occurring while on an antiplatelet agent [28]. Few patients in PATCH were on clopidogrel and those undergoing neurosurgical procedures were excluded. The most recent antithrombotic reversal guidelines from the Neurocritical Care Society recommend platelet transfusion for patients on antiplatelet medications who are undergoing a neurosurgical procedure [28]. They also recommend considering a single intravenous dose of 0.4 mcg/kg of DDAVP (desmopressin) in antiplatelet medication-related ICH. Additional trials are assessing platelet transfusion in ICH patients as well as the role of platelet-function assays in directing treatment.

Newer anticoagulants, such as direct thrombin inhibitors (e.g., dabigatran) or direct Xa inhibitors (e.g., rivaroxaban and apixaban), do not have evidence that reversal decreases hematoma expansion or improves outcome. Idarucizumab is a targeted monoclonal antibody that binds to the thrombin binding site of dabigatran [39]. It is approved for

use and is recommended as the initial reversal agent for patients with ICH while on dabigatran [28]. Activated charcoal (50 gm) should also be given if ICH occurs within 2 h of the most recent dabigatran dose. Less-recommended alternatives for reversal of direct thrombin inhibitors if idarucizumab is not available are the activated PCC FEIBA (factor VIII inhibitor bypassing activity) or 4-factor PCC; however, these approaches have not been formally tested and do not fully reverse dabigatran coagulopathy [28, 40, 41]. Direct Xa inhibitors do not currently have specific reversal agents available. There is some suggestion that PCCs may have limited effectiveness in reversing the effect of rivaroxaban and apixaban [42]. The currently recommended approach is to use FEIBA or 4-factor PCC with the addition of charcoal if the last dose of direct Xa inhibitor was within 2 h [28]. It should be noted that additional laboratory tests, such as endogenous thrombin potential and thrombin clotting time, may have some value in assessing the activity of these newer anticoagulant agents. Vitamin K is of no value and FFP is of unclear utility. Specific antidotes for direct Xa inhibitors are in development [28].

Unfractionated heparin is used for many medical conditions, including acute coronary syndromes, pulmonary embolism, and endovascular surgery, as well as for maintaining the patency of indwelling catheters. Heparin binds to and activates antithrombin III, thus inactivating thrombin and favoring thrombolysis. The reversal agent for heparin is protamine sulfate, administered 1 mg for every 100 units of heparin received in the prior 2 h, with a maximum dose of 50 mg [43]. Protamine sulfate binds to and inactivates heparin, allowing it to be broken down by the reticuloendothelial system. Given the short half-life of heparin, reversal is likely unnecessary if the last dose was received greater than 4 h prior to ICH onset. Protamine sulfate can also be used in the same dose in an attempt to reverse the effect of low molecular weight heparin that was given within the prior 8 h. However, this reversal may be incomplete.

Surgical Hematoma Evacuation

Though most patients with acute ICH do not require surgery for removal of the hematoma, it is worthwhile to address the option of surgery immediately after ICH diagnosis, since the theoretical benefits of surgery include prevention of brain herniation, improvement in elevated ICP, and removal of blood and blood degradation products that may produce cytoxic secondary brain injury.

After decades of ambiguity, the effects of surgical evacuation were addressed in the Surgical Trial in Intracerebral Haemorrhage (STICH) that found early surgical evacuation of a supratentorial ICH was not harmful, but there was no difference in long-term mortality or functional outcome [44]. Because the subgroup of patients in STICH with lobar ICH within 1 cm of the cortical surface may have benefited from surgical evacuation, the STICH II clinical trial was undertaken for this group of patients [45]. However, STICH II did not demonstrate a significant benefit to early hematoma evacuation in these patients either. Minimally invasive techniques, including endoscopic hematoma aspiration or instillation of a thrombolytic such as urokinase or recombinant tissue plasminogen activator into the hematoma with aspiration of contents, are also being studied [46–48]. At present, routine removal of supratentorial hematoma cannot be endorsed, but it is still undertaken as a life-saving measure in selected patients.

In contrast, several case series suggest that patients with cerebellar ICH $>$ 3 cm in diameter or with compression of the brain stem or hydrocephalus may benefit from surgical hematoma evacuation [49, 50]. There has not been a randomized trial of cerebellar hematoma evacuation analogous to STICH, but it is not clear there is equipoise to justify such a trial.

Current American Heart Association ICH guidelines recommend that patients with cerebellar hemorrhage who are deteriorating neurologically or have brainstem compression should undergo surgical removal of the hemorrhage as soon as possible. Initial treatment of these patients with ventricular drainage alone rather than surgical evacuation is not recommended [1]. Supratentorial hematoma evacuation or decompressive hemicraniectomy might be considered as a life-saving measure in deteriorating patients. Correction of coagulopathy is critical in patients undergoing surgical hematoma evacuation.

Secondary Intervention: Hospital Admission, ICP Management, and Seizures

Ideally, patients with acute ICH should be admitted to an ICU based on the need for close monitoring of neurological and hemodynamic condition and the risk for early deterioration from hematoma expansion, cerebral edema, hydrocephalus, or airway compromise. Admission to a neurological ICU has been associated with improved outcomes compared with admission to a non-neurological ICU [51]. Acknowledging that certain patients will require transfer between hospitals for neurological intensive care management, neurosurgical intervention, or neurointerventional capabilities, all aspects of ICH primary intervention can and should take place without delay in the initial presenting hospital.

Specifically, correction of coagulopathy with appropriate agents, blood pressure control, and treatment of acute seizures should be initiated in the ED of the presenting

hospital and not deferred until after transfer. It is critical that the above-discussed aspects of acute ICH evaluation and treatment are initiated at the time of original diagnosis and that transitions in care are smooth from ED to ICU (or operating room, interventional radiology, or comprehensive stroke center).

While this ENLS ICH protocol is principally concerned with the initial evaluation and treatment period, it is important to anticipate the health care needs of the following 24–72 h as part of care planning. The first 24 h are critical for blood pressure management, identification of seizures, ICP management, and maintaining a secure airway. Avoidance of fever, hyperglycemia/hypoglycemia, and hypoxia are also important, as these may impact outcomes [1, 52, 53]. In addition, patients with ICH are at increased risk for the development of deep venous thrombosis (DVT); current guidelines recommend use of intermittent pneumatic compression devices at hospital admission, as well as initiation of prophylaxis-dose unfractionated or low-molecular weight heparin within 1–4 days following onset (assuming cessation of bleeding) [1, 54].

The incidence and impact of elevated ICP in ICH has received limited study, but it is undoubtedly a factor in management [55–58]. Patients with IVH are at risk for hydrocephalus and elevated ICP. Current guidelines for ICP monitoring in ICH follow the approach in severe traumatic brain injury, with ICP monitoring recommended in patients with GCS ≤ 8, large hematomas with mass effect suggestive of elevated ICP, or hydrocephalus. As a goal, an ICP < 20 mmHg should be maintained, with a minimal CPP of 60 mmHg, adjusted based on an individual patient's cerebral autoregulation status [1]. Ventricular catheters are beneficial in their ability to both measure ICP and drain cerebrospinal fluid (CSF); therefore, they should be used in patients with hydrocephalus. In contrast, intraparenchymal fiberoptic monitors have a lower risk of hemorrhage and infection, but cannot be used to drain CSF. Correction of coagulopathy prior to ICP monitor insertion is desirable.

While seizures may occur in ICH patients, their incidence and impact on outcome have varied across studies [59, 60]. In a single study, prophylactic anticonvulsants reduced seizure occurrence in lobar ICH [60]. However, two more recent studies found worse functional outcomes in patients routinely given prophylactic anticonvulsants (primarily phenytoin) [61, 62]. While comatose ICH patients may have a high risk (approximately 20%) of nonconvulsive seizures, the impact of prophylactic anticonvulsants on their occurrence is also unclear [63, 64]. Current guidelines do not recommend routine use of prophylactic anticonvulsants [1], though some practitioners still use a short course in patients with lobar ICH and those undergoing surgical hematoma evacuation. Clinical seizures should be treated, and continuous EEG monitoring should be performed in patients with inadequately explained decreased level of consciousness.

Algorithm

An algorithm for the acute management of the ICH patient according to the principles of ENLS is presented in Table 3. This could be used as a checklist for proceeding throughout the domains of care from prehospital, to ED, to disposition in the Neurocritical Care Unit, OR, or interfacility transfer and can be shared across medical providers as this care proceeds. Note that frequent reassessment of ABCs and clinical neurological status is a key component throughout the care pathway as is revisiting the effectiveness of initial interventions such as blood pressure lowering and coagulopathy reversal in rapidly achieving the desired targets.

Pediatric Considerations

Since chronic hypertension and chronic anticoagulation therapy are less common in children, ICH is seen with much less frequency in pediatric patients. However, children may present with life threatening ICH due to vascular malformations, sickle cell disease, stroke or infection. Trauma can also lead to significant ICH that requires emergent intervention.

While less than 2% of cerebral aneurysms are found in pediatric patients, as many as 24% of children with intracranial aneurysms may have ICH at the time of their initial presentation [65]. For children with significant ICH, the same emergent care principles described earlier in this chapter apply regarding need for establishment of the airway, and providing adequate oxygenation and maintaining blood pressure (see pediatric section in Airway, Ventilation and Sedation Chapter). Hypotension is defined as systolic blood pressure (SBP) below the 5th percentile for age (SBP 5th percentile = 70 mmHg + age in years X 2). Careful attention should also be given to the detection and treatment of seizures, which may be present in as many as 21% of children with intracranial aneurysms [66], and treatment of ICH in need of emergent surgical evacuation. In children with SAH, admission to a center with expertise in diagnosing and treating vasospasm is indicated, as cerebral vasospasm may be seen in as many as 67% of children with SAH [67]. The diagnosis of cerebral vasospasm is particularly challenging in children given cerebral blood flow velocity is age and gender dependent [68, 69]. When clinically indicated, cerebral angiography has similar

Table 3 Standardized ICH management

Prehospital care
☐ ABCs
☐ Determine time of onset and circumstances
☐ Perform prehospital stroke screen
☐ Brief medical history and medication list
☐ Triage to stroke center
☐ Perform prehospital notification of pending stroke patient
ED Care
☐ Emergent triage to high acuity area
☐ Perform primary assessment—ABCs
☐ Perform focused neurologic exam (GCS, NIHSS)
☐ Obtain baseline screening labs (CBC and platelet count, electrolytes, INR and PTT, glucose)
☐ Obtain cerebrovascular imaging as soon as possible (non-con CT, stroke CT/CTA/CTP, or MRI)
☐ Obtain brief medical history and medication list
After confirmation of ICH
☐ Reassess ABCs (consider intubation if comatose)
☐ Initiate blood pressure intervention (target SBP 140-180 mmHg)
☐ Quantify ICH volume (ABC/2 calculation)
☐ Perform ICH Score (0–6)
☐ Begin correction of anticoagulation as required (goal INR $\leq$ 1.4)
☐ Consult neurosurgery for potential hematoma evacuation or ICP monitor placement
☐ Admit to (Neuro) ICU (may require transfer)
In-hospital setting
☐ Continue to reassess ABCs
☐ Continue neurologic reassessment
☐ ICP monitor and/or ventriculostomy for treatment of elevated ICP or hydrocephalus
☐ Continue management of blood pressure
☐ Place arterial blood pressure catheter as needed
☐ Place central venous catheter as needed
☐ Urine toxicology screen (if not already done)
☐ Foley catheter (needed for most ICH patients early)
☐ Feeding tube (goal to begin feeding within first day)
☐ DVT prophylaxis with sequential compression devices (consider heparin/LWMH on day 2)
☐ Recheck INR and PTT if patient was coagulopathic and receiving reversal agents
☐ No anticonvulsant prophylaxis; treat clinical seizures; continuous EEG if level of consciousness impaired out of proportion to ICH or IVH
☐ Consider need for repeat head CT
☐ Consider need for catheter cerebral angiography

complication rates compared to those reported in adults, even in children younger than 3 years of age [70].

There are no established parameters for treatment of hypertension in children with ICH, but a SBP target of 140-180 mmHg is reasonable in older children. Nicardipine is well tolerated and the recommended dose is 0.5 mcg/kg/min, titrated by 0.5 mcg/kg/min every 15 min to a maximum of 5 mcg/kg/min. In older children (adult weight) the initial dose is 2.5 mg/hr, with titration by 2.5 mg/hr every 15 min up to a maximum of 15 mg/hr. Esmolol is a reasonable alternative and generally well tolerated. Finally, while anticoagulation therapy is less common in children, pediatric patients with ICH may present with coagulation abnormalities that require careful evaluation and treatment to prevent hematoma expansion and facilitate surgical therapy.

Table 4 Intracerebral hemorrhage communication regarding assessment and referral

Communication
☐ Age
☐ GCS
☐ Hematoma volume and location
☐ Other CT findings (intraventricular hemorrhage, hydrocephalus, spot sign)
☐ ICH Score
☐ Airway status
☐ Blood Pressure, target, and treatment initiated
☐ Coagulation parameters (INR, PT, PTT, platelet count) and reversal treatment
☐ Plan for surgery
Sample Sign-Off Narrative
"I am signing out a 62 yo man with known hypertension and atrial fibrillation who is presumed to be on warfarin."
"He was found at home this morning at 9 AM by his wife who last saw him normal at 7 AM. He was talking to EMS and had left-sided weakness, GCS in the field was 13, and BP was 170/100."
"On arrival to the ED here, he was the same, so we took labs and sent him for a head CT."
"CT completed at 10 AM showed a 20 ml right thalamic ICH with mild IVH, but no hydrocephalus. There is about 4 mm of right-to-left midline shift. CTA/CTP showed no AVM or aneurysm, but there is a positive spot sign."
"When he returned to the ED, he was sleepier, with a GCS of 10, and his left-sided weakness was worse. So he has an ICH Score of 2. His labs came back with an INR of 1.9."
"We intubated him using rocuronium and etomidate. PCC infusion of 2250 IU (estimated weight 90 kg; dose of 25 IU/kg) is going in now. He also had 10 mg of IV vitamin K."
"Neurosurgery has been called, and they are on their way to see him. He is in ED Resuscitation Room 1, intubated and sedated now on propofol at 60 mcg/kg/min. His BP is 140/85 with no other treatment."
"They are ready to take him in Bed 2 in the Neurocritical Care Unit in 5 min. Nursing is also calling report."

Communication

When communicating to an accepting or referring physician about a patient with ICH, consider including the key elements listed in Table 4.

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