

REVIEW

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Acute management of spontaneous intracerebral hemorrhage (ICH) in the emergency department

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Background Spontaneous intracerebral hemorrhage (ICH) is a catastrophic form of stroke affecting more than 3.3 million individuals worldwide each year and accounting for approximately 10–20% of all strokes globally. Although less common than acute ischemic stroke, ICH carries a disproportionate burden of morbidity and mortality, with 30-day case fatality rates approaching 40–50%. Therapeutic advances for ICH have progressed more slowly than for ischemic stroke, and management remains largely supportive. Patients frequently present to the emergency department (ED) in critical condition, making early recognition and rapid, guideline-concordant intervention essential to improving outcomes.

Review This narrative review summarizes the most recent American Heart Association/American Stroke Association (AHA/ASA) guidelines for the acute management of spontaneous ICH, with emphasis on practical ED application. Hypertension remains the most important modifiable risk factor, particularly in younger patients, while cerebral amyloid angiopathy predominates in older adults. Additional risk factors include anticoagulant and antiplatelet therapy, alcohol and illicit drug use, smoking, advanced age, and genetic predisposition. Primary brain injury results from hematoma mass effect and elevated intracranial pressure, followed by secondary injury driven by edema, inflammation, and oxidative stress. Because hematoma expansion commonly occurs within the first hours after symptom onset and strongly predicts mortality, early ED management prioritizes rapid neuroimaging, controlled blood pressure reduction, timely anticoagulation reversal, seizure management, metabolic and temperature control, and prompt neurosurgical consultation when indicated. Emerging evidence supports bundled, time-sensitive care pathways to reduce delays and optimize outcomes.

Conclusion Optimal ICH outcomes depend on rapid, structured, guideline-concordant ED care focused on limiting hematoma expansion and secondary injury. Standardized workflows and bundled interventions represent effective strategies for improving survival and functional outcomes while avoiding premature prognostication in the acute phase.

Keywords Acute intracerebral hemorrhage, Blood pressure management, Anticoagulation reversal

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Introduction

Every year over 3.3 million people worldwide suffer spontaneous intracerebral hemorrhage (ICH), a devastating form of stroke characterized by bleeding directly into brain parenchyma [1]. Accounting for 10–20% of all strokes in the US, the mortality rate is as high as 50% at 30 days [2–4]. And yet until just a few years ago, guidelines for ICH management were sparse and therapeutic options remain few with questionable efficacy especially in comparison to the rapid progression in acute ischemic stroke (AIS) care. These patients present in critical condition to the emergency department where the acute management of their presentation is a crucial aspect of care and may impact long-term neurologic outcome. The purpose of this review is to provide a summary

of the new AHA guidelines for the acute management of ICH [5] as they pertain to emergency medicine.

Hypertension remains the most significant modifiable risk factor for primary ICH and the most common cause of spontaneous ICH in patients under 70 years of age. Chronic hypertension leads to remodeling of small penetrating arteries in deep brain structures predisposing them to rupture [6]. Another common cause of spontaneous ICH particularly in older populations is cerebral amyloid angiopathy (CAA) [7]. Amyloid deposits weaken vascular integrity generally leading to parenchymal bleeding within lobar regions. Additional risk factors for ICH Fig. 1 include anticoagulant and antiplatelet therapy [8, 9], alcohol intake, drug use, and smoking, each of which independently increases the likelihood of hemorrhage [10]. The prognosis of ICH is heavily influenced by

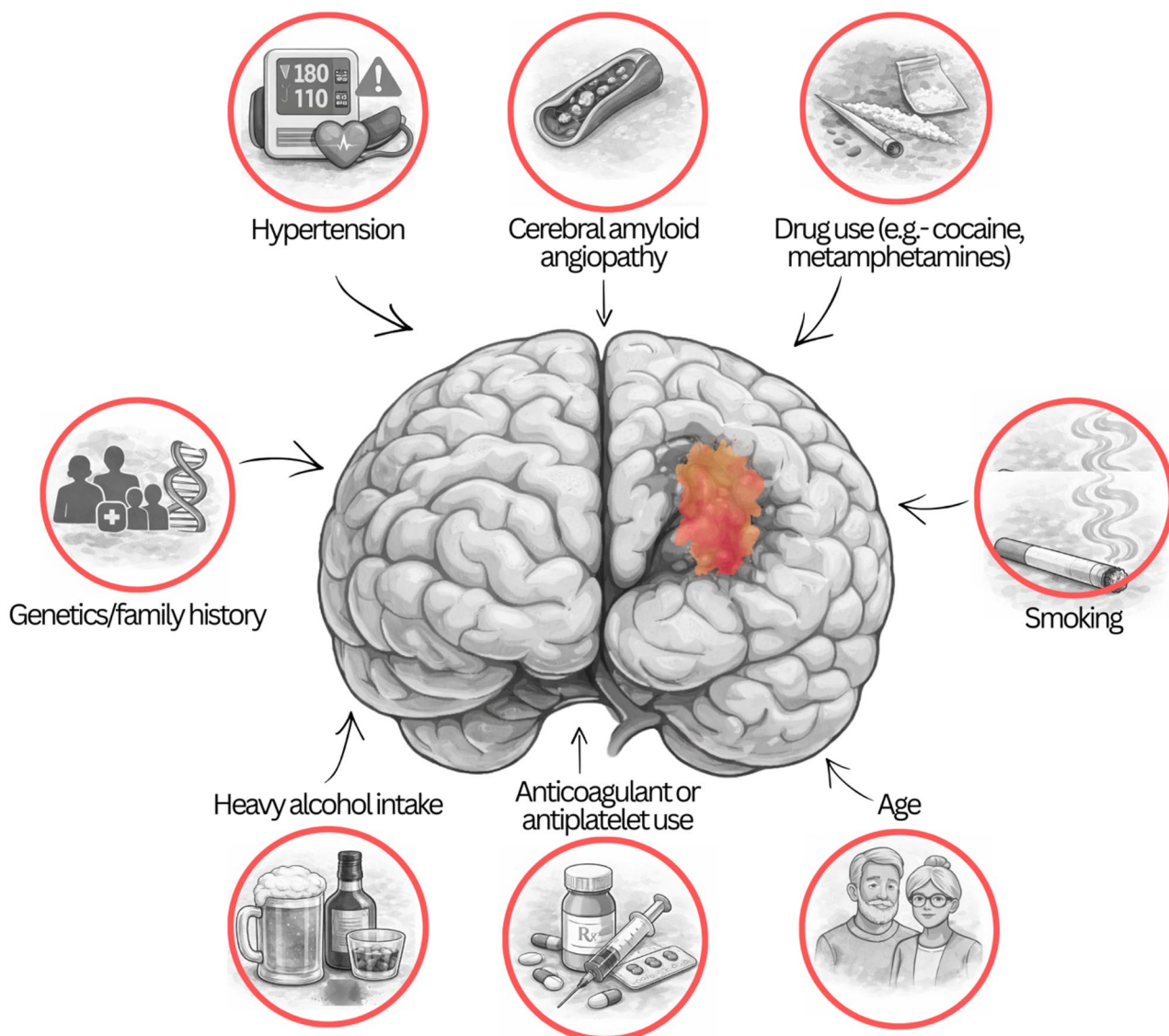


Fig. 1 Risk factors for spontaneous intracerebral hemorrhage (ICH). Graphic designed by L. Ganti in canva.com

volume of bleeding, location of hemorrhage, patient age and comorbidities, and timeliness of interventions [11].

Initial brain injury occurs through the mass effect of the hematoma which can cause increased intracranial pressure, decreased cerebral perfusion, and even lead to brainstem herniation. Secondary injury begins in the first few hours and progresses over days to weeks and includes edema, inflammation, and oxidative stress from breakdown of blood [7]. Given the critical nature of ICH and its rapid progression, early recognition and a systematic approach to management including prompt neuroimaging, acute blood pressure management, timely reversal of anticoagulation, and neurosurgical evaluation for select patients is key for patient outcomes.

Review

Initial management

Despite preclinical and clinical trials over the past decades, there is no single treatment that has been shown to significantly improve mortality and neurologic outcome after ICH. Thus, ICH management guidelines focus on prevention of ICH through risk management, medical management to prevent hematoma expansion in the acute phase of ICH, and consideration of surgical management for select patients. The mainstay of initial and impactful management of ICH is medical management in

the first few hours after presentation which often occurs in the emergency department. The timely interventions of ED providers critically impacts the clinical course and outcome of ICH patients so being adept at management of this patient population is key.

At initial presentation, differentiating hemorrhagic stroke, like ICH, from ischemic stroke can be difficult. Patients often present with abrupt alterations in mental status, acute neurologic deficit, seizure, headache, elevated blood pressure, and/or vomiting. However, acute management for all critically ill stroke patients consists of rapid assessment and stabilization that begins with the traditional ABCs (airway, breathing and circulation). If patients are not protecting their airway, or have a GCS \leq 8, a definitive airway should be secured. Oxygen supplementation should be administered with a goal of SpO₂ > 94% [12].

A thorough neurologic exam should be performed including obtaining an initial Glasgow Coma Scale (GCS), NIH stroke scale and ICH score Fig. 2. These initial scores can be used to monitor for changing clinical status and provide a tool to communicate to specialists the acuity and severity of clinical presentation.

Important pertinent history, obtained from the patient if able, emergency medical services (EMS), or family includes time of last known well, use of anticoagulation

Intracerebral Hemorrhage (ICH) Score		ICH Score	Mortality
ITEM	SCORE	0	0%
Glasgow Coma Score (GCS) 3-4	+2	1	13%
GCS 5-12	+1	2	26%
GCS 13-15	0	3	72%
Age > 80	Yes= +1 No= 0	4	94%
ICH volume > 30cc	Yes= +1 No= 0	5	100%
Intraventricular hemorrhage	Yes= +1 No= 0	6	100%
Infratentorial origin of hemorrhage	Yes= +1 No= 0		

Fig. 2 Intracerebral score. From Hemphill JC 3rd, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. *Stroke*. 2001 Apr;32(4):891-7. doi: <https://doi.org/10.1161/01.str.32.4.891>. PMID: 11,283,388

medications and time of last dose, known kidney disease, recent surgeries, and recent stroke or brain trauma. Rapid intravenous access and laboratory studies (such as complete blood count, blood glucose, electrolytes, kidney function, coagulation panel, and toxicology screen) should be promptly obtained.

Emergent neuroimaging provides a definitive diagnosis. Noncontrast computerized tomography (NCCT), widely and rapidly available, is simple and highly sensitive to diagnose ICH, determine location of hemorrhage, assess size of hematoma, presence of IVH extension, and detect complications including hydrocephalus or midline shift [13]. The most common locations for spontaneous intracerebral hemorrhage are the putamen, thalamus, and caudate nucleus, which account for 60%, with lobar, cerebellar and pontine hemorrhages accounting for the remaining 40%.

CT angiography (CTA) can be utilized when available to differentiate between stroke type when it is unclear by initial presentation provide additional information about secondary causes of ICH including aneurysm or AV malformation [11]. Additionally, it may be possible to see a spot sign, presence of contrast extravasation on CTA images, which is an independent predictor of hematoma expansion and poor outcome in patients with supratentorial ICH which may help guide surgical decision making [14–16].

MRI is useful for imaging of hematoma evolution over time and is more sensitive for identifying tumors, arterial or venous infarcts or other angiographically occult vascular malformations as cause of ICH – though it is much less likely that this would be obtained acutely in the ED and should not be part of initial acute care [17, 18]. Having a system in place to prioritize rapid neuroimaging of these patients is critical to treatment which relies on timely interventions after diagnosis as early hematoma expansion occurs in up to 1/3 of patients [19].

Hematoma expansion

ICH volume (due to physical disruption within a constrained/fixed space) is the strongest predictor of mortality with hematoma volume > 60mL associated with a 91% 30-day mortality rate. Hematoma expansion (HE) is common following ICH with > 70% of patients who present within the first 3 h of initial bleed exhibiting enlargement of hematoma within the first 24 h which is associated with worsened mortality and poor functional outcome [19–21]. If a patient is anticoagulated, HE is more common, associated with greater mortality, and occurs later in comparison to non-anticoagulated patients [22, 23]. The mainstay of acute medical management is aimed at preventing HE through timely BP management, anticoagulation reversal if needed, temperature and blood glucose control, ICP monitoring, and seizure management.

Figure 3 summarizes the hematoma expansion score, a tool that may be helpful in risk stratification. *It is important to emphasize that a DNR (do not resuscitate) order should be deferred for first 24 h, regardless of any clinical score* [5].

Blood pressure management

HTN is a common cause of spontaneous ICH and the majority of ICH patients present with elevated blood pressures in the acute phase [24]. Persistent HTN in the acute phase is associated with higher risk of HE and poorer outcomes [25–27]. Therefore, controlling blood pressure is critically important in the management of ICH aimed at preventing primary and secondary brain injury and improving outcomes.

As HE occurs in the first few hours after ICH, *early* BP control is a priority. Several trials, including INTERACT2 and ATACH-2 [28] evaluated the effect of intensive BP reduction on outcomes, while neither met their primary outcome, INTERACT-2 demonstrated improved functional outcomes with intensive BP control without adverse effects. And secondary study analyses demonstrated an association of early BP management with a decrease in HE and improved outcomes [28–30]. These studies informed current AHA/ASA guidelines which recommend that BP management be started as soon as possible, within the first hour of arrival, enacted in a smooth and sustained manner preferably with continuous antihypertensive infusion [28, 31]. IV antihypertensive agents including nicardipine and labetalol provide easily titratable, smooth, and sustained BP control. The recommended target SBP is 140 mmHg with the goal of maintaining a SBP range of 130–150 mmHg while avoiding drops in BP of > 60 mm Hg especially within the first hour and then sustained for the next several days [5]. In ICH patients with mild to moderate severity presenting with SBP > 150, it is important to avoid reduction of SBP < 130 mm Hg in the acute phase as it appears harmful.

Anticoagulation reversal

Use of anticoagulation and antiplatelet medications are known risk factors for spontaneous ICH and increase risk of larger bleeds, hematoma expansion and increased risk of mortality [8, 9, 32–34]. Determining patient's anticoagulation early is critical as well as obtaining the relevant laboratory tests to guide anti-coagulation reversal. As the benefit of reversal is likely time dependent, these agents should be administered within the first hour of arrival [34–36]. Having a systematic process in place to allow rapid lab testing, access to anticoagulation reversal, and staff trained in reconstitution and administration is critical to meeting these time goals.

Prediction Score for Hematoma Expansion	
Variable	Points
Warfarin sodium use	
No	0
Yes	2
Time to initial CT in hours	
<6	2
>6	0
Baseline ICH volume, mL	
<30	0
30-60	1
>60	2
CT angiography spot sign	
absent	0
present	3
unavailable	1
TOTAL	0-9

Performance of the Prediction Score for Hematoma Expansion and Mortality		
Score	Expansion	In-hospital mortality
0	5.7%	2.9%
1	11.1%	13%
2	7.7%	14.8%
3	17.9%	23.5%
4	29.6%	33.3%
5	35.4%	34.1%
6	53.6%	75%
7	45.5%	45.5%
8	n/a	100%
9	80%	100%

Fig. 3 Hematoma expansion score (left panel and suggested interpretation (right panel). From Brouwers et al. [21]

Specific reversal guidelines include (Fig. 4):

1. Vitamin K antagonists (VKAs) can be rapidly reversed with Vitamin K (10 mg IV; takes 6–24 h to work) and 4-factor prothrombin complex concentrates (4-F PCC/Kcentra®; rapid onset) and should be administered if INR ≥ 2 with the goal of achieving an INR ≤ 1.4 . In patients with INR of 1.3–1.9, it is reasonable to use PCC to achieve rapid corrected of INR and limit HE [33–36, 38, 39]. This approach is recommended by the European Stroke Organization and the American Heart Association with the benefit of this likely time dependent.
2. Factor Xa inhibitors (rivaroxaban, apixaban, and edoxaban) should be reversed with 4-factor prothrombin concentrates. Andexanet alf is a recombinant modified version of human Factor X that binds Factor Xa inhibitors and achieves hemostasis in patients treated within 18 h of last DOAC dose [39, 40]. The Annexa-I clinical trial **demonstrated that among** patients with intracerebral hemorrhage who were receiving factor Xa inhibitors, andexanet resulted in better control of hematoma expansion than usual care [41]. However

the increased risk of thrombotic complications led it to be pulled from the US market in December 2025 [42].

3. Factor IIa inhibitors (dabigatran) should be reversed with idarucizumab, a monoclonal antibody fragment that binds dabigatran for hemostasis [43–45]. In cases where Idarucizumab is unavailable, PCCs may be considered to improve hemostasis and renal replacement therapy (RRT) may be considered to reduce dabigatran concentration [38].
4. Unfractionated heparin (UFH) and low molecular weight heparin (LMWH) should be reversed *with* intravenous protamine.

Antiplatelet therapy

There are not enough data on the safety or utility of platelet transfusion in patients on antiplatelet therapy so this is not recommended in routine practice [5]. It may only be indicated in patients with severe thrombocytopenia between 50 and 100 platelets per microliter or when emergent neurosurgery is required (2b recommendation) [5]. Other general hemostatic treatments: there are ongoing trials assessing the efficacy of other hemostatic agents in acute ICH management (e.g., recombinant factor VIIa, TXA) but there are not enough data on the

Anticoagulation reversal in ICH

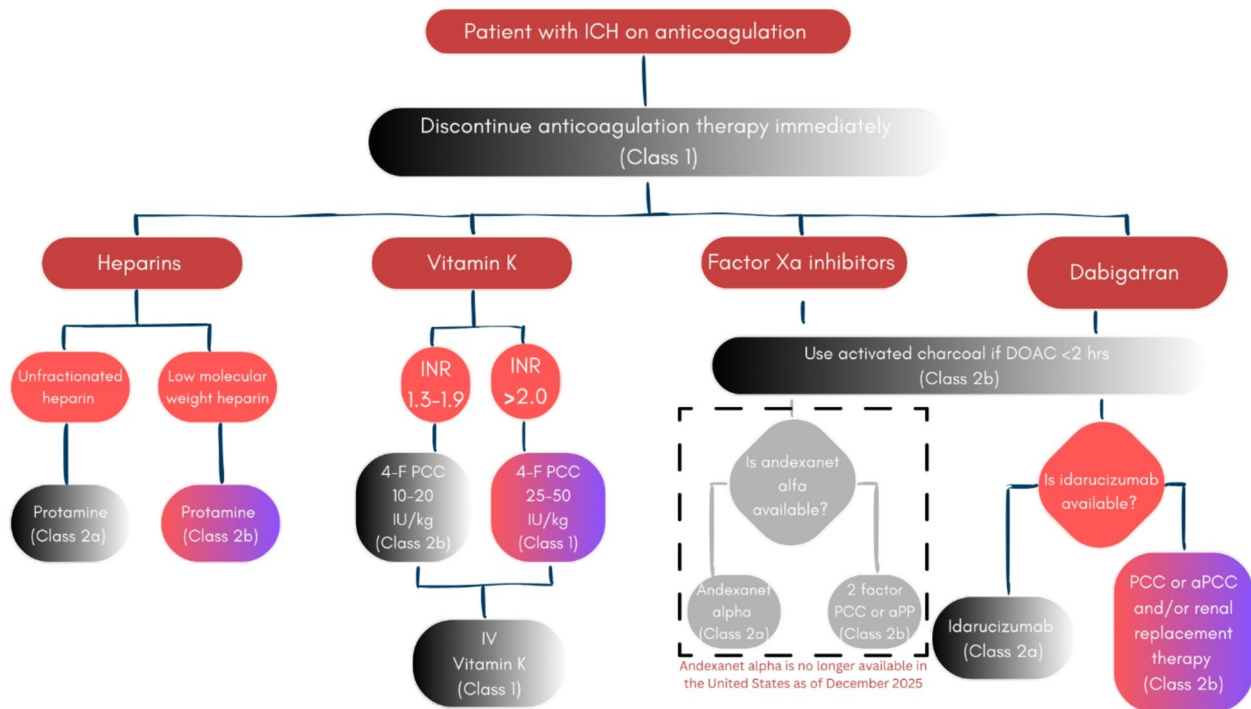


Fig. 4 Anticoagulation reversal for ICH. Based on article by Ward, S., Jamal, B.C. & Ganti, L. Frontoparietal intraparenchymal hemorrhage secondary to anticoagulation. *Int J Emerg Med* 17, 130 (2024). <https://doi.org/10.1186/s12245-024-00723-0> [37]. Adapted from 2022 AHA Guidelines for Management of ICH [5]

safety or utility of this practice so it is not recommended in routine practice [46–49].

Fluid management

The primary goal is to maintain adequate hydration without causing fluid overload that could worsen ICP and cerebral edema while avoiding hypotension and risk of cerebral hypoperfusion/ischemia [50].

Temperature control

Fever is common within the first 72 h occurring in 30–45% of patients and is independently associated with worse outcomes and mortality [51–53]. Pharmacologic treatment of elevated temperature may be reasonable to improve functional outcomes [52–55]. The usefulness of therapeutic hypothermia in ICH is unclear and not currently recommended in routine practice [56, 57].

Serum glucose

Hyperglycemia seen in up to 60% of patients within first 24 h regardless of history of diabetes and is independently associated with worse outcomes/early mortality

[58, 59] A Mayo clinic study found that hyperglycemia on presentation in non-diabetic patients is an independent predictor of early mortality and worse functional outcome in patients with intracerebral hemorrhage, and may actually reflect occult diabetes [60].

Monitoring blood glucose levels and treating hypoglycemia and hyperglycemia (> 180–220 mg/dL) is reasonable [61–65].

Seizure and anti-epileptic drugs

Reported incidence of seizures is 4–42% after ICH usually presents within the first 24–72 h. In patients with ICH and clinical or EEG evidence of seizures, anti-seizure medications should be initiated immediately to reduce morbidity [66, 67]. In patients *without evidence of seizures*, there is no evidence to suggest benefit of prophylactic antiseizure medication so is not commonly recommended in routine practice [68–70].

Management of elevated intracranial pressure (ICP)

Mass effect from ICH can lead to midline shift, herniation. Elevated ICP related to mass effect from initial

Table 1 Time metrics proposed for the evaluation of intracerebral hemorrhage, modeled after the US joint commission metrics for acute ischemic stroke

National joint commission ischemic stroke time metrics		CODE ICH proposed hemorrhagic stroke time metrics	
Door to Doc	≤ 10 min	Door to Doc	≤ 10 min
Door to Stroke Team	≤ 15 min	Door to Stroke Team	≤ 15 min
Door to CT scan start	≤ 25 min	Door to CT scan start	≤ 25 min
Door to CT scan read	≤ 45 min	Door to CT scan read	≤ 45 min
Door to Needle (lytic)	≤ 60 min	Door to Needle (anti-coagulation reversal)	≤ 60 min
Door to arterial puncture (for thrombectomy)	≤ 90 min	Door to first anti-hypertensive	≤ 60 min
		Door to Target Blood Pressure	≤ 90 min

hematoma, hematoma expansion or obstructive hydrocephalus from intraventricular hemorrhage can rapidly alter neuro exam which is a sign of herniation. Additionally, approximately 40% of patients will have IVH on initial imaging with increasing IVH volume associated with worsening outcomes and mortality related to obstructive hydrocephalus [71–73].

In patients with evidence of increasing ICP, bolus hyperosmolar therapy (including mannitol and hypertonic saline-preferred) may be considered for transiently reducing ICP but prompt neurosurgical consultation and intervention with EVD is considered a life-saving procedure. There is no role (i.e. potential for risk, and no benefit) for prophylactic steroid administration in ICH [5].

In cases of large volume hemorrhage or those causing significant mass effect, surgical intervention may be required. For select patients, early neurosurgical consideration is important with options including hematoma evacuation and decompressive craniectomy [74, 75]. In the most recent studies on hematoma evacuation, including the most recent MISTIE III, there may be some mortality benefit, compared to medical management alone, of minimally invasive hematoma evacuation for patients with supratentorial ICH > 20mL volume and GSC of 5–12 (2a recommendation). This will depend on availability of such expertise but early consultation is recommended. In patients with posterior fossa hemorrhage and deteriorating neuro status, signs of brainstem compression, cerebellar ICH volume ≥ 15 neurosurgical intervention is recommended [76–83]. Being familiar with available neurosurgical expertise and arranging rapid consultation and transfer if needed is critically important but should be done concurrently with medical management.

Bundles of care, ED order sets, time metrics/golden hour

Bundles of care refer to a set of evidence-based practices that are implemented together to improve patient outcomes. While all the interventions above are

independently important, all these therapies occurring in parallel as goal-directed care bundles, have been shown to be the most impactful ICH intervention. In several studies, bundling of care (including anticoagulation reversal, intensive BP reduction, glucose and temperature monitoring and management, and surgical evaluation/intervention) with specific time targets led to improved patient outcomes [84–86]. A recent focus on elevating the attention to “CODE ICH” in a similar fashion as “CODE STROKE” for AIS management shows promise as the future of acute phase ICH management [87] (Table 1).

Prognostication

Most patients who die in the hospital with ICH, do so after decisions by the care team and surrogate decision makers to limit life-saving therapies and interventions likely based on presumed low likelihood of a favorable outcome and surrogate/patient-aligned goals of care. But ICH is a high mortality disease that carries inherent prognostic uncertainty. After engaging in shared-decision making with the surrogate decision maker, it is reasonable in the acute phase of ICH management within the emergency department to avoid prognostication and instead focus on initial guideline-concordant care for all patients with ICH unless previous care limitations had been implemented prior to ICH. AHA recommends that care limitations or withdrawal of support *should not be recommended* by treating physicians in the first 24-hours after ICH [5, 88, 89].

Emerging therapies and future directions

One of the most important goals in improving stroke care is the identification of gaps and barriers that would be highly relevant to enhancing prognosis outcomes. Compared to AIS management, there continues to be a paucity of effective ICH therapies. Thus, it is critical that the field continues to investigate novel prognostic biomarkers and new pharmacologic agents for ultra-early hemostatic therapies, neuroprotection, and minimally invasive surgery. Other areas of improving care includes optimizing bundle components and developing more precise and individualized protocols for goal-directed therapy. Investigating new methods to minimize delays in treatment and enhance adherence to time metrics.

Conclusion

The acute management of intracerebral hemorrhage requires a multifaceted approach, combining immediate clinical intervention, consideration of surgical options, and supportive care. Ongoing research and advances in treatment strategies continue to refine and improve outcomes for patients with ICH. Staying updated with the

latest literature and guidelines is crucial for clinicians involved in the management of this severe condition.

Author contributions

MP and LG drafted the manuscript.

Funding

No funding was received for this study.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Competing interests

Dr. Ganti has an editorial role at Springer.

Received: 31 December 2025 / Accepted: 14 January 2026

Published online: 30 January 2026

References

1. Parry-Jones AR, Krishnamurthi R, Ziai WC, Shoamanesh A, Wu S, Martins SO, Anderson CS. World stroke organization (WSO): global intracerebral hemorrhage factsheet 2025. *Int J Stroke*. 2025;20(2):145–50. <https://doi.org/10.1177/17474930241307876>. Epub 2025 Jan 6. PMID: 39629687; PMCID: PMC11786522.
2. van Asch CJ, Luitse MJ, Rinkel GJ, van der Tweel I, Algra A, Klijn CJ. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. *Lancet Neurol*. 2010;9.
3. Fogelholm R, Murros K, Rissanen A, Avikainen S. Long term survival after primary intracerebral haemorrhage: a retrospective population based study. *J Neurol Neurosurg Psychiatry*. 2005;76.
4. Pinho J, Costa AS, Araújo JM, Amorim JM, Ferreira C. Intracerebral hemorrhage outcome: a comprehensive update. *J Neurol Sci*. 2019;398.
5. Greenberg SM, Ziai WC, Cordonnier C, Dowlatshahi D, Francis B, Goldstein JN, Hemphill JC, Johnson R, Keigher KM, Mack WJ, et al. 2022 guideline for the management of patients with spontaneous intracerebral hemorrhage: a guideline from the American heart association/American stroke association. *Stroke*. 2022;53.
6. Broderick M, Rosignoli L, Lunagariya A, Nagaraja N. Hypertension is a leading cause of nontraumatic intracerebral hemorrhage in young adults. *J Stroke Cerebrovasc Dis*. 2020;29.
7. Magid-Bernstein J, Girard R, Polster S, Srinath A, Romanos S, Awad IA, Sansing LH. Cerebral hemorrhage: Pathophysiology, Treatment, and future directions. *Circ Res*. 2022;130:1204–29.
8. Stead LG, Jain A, Bellolio MF, Odufuye AO, Dhillon RK, Manivannan V, Gilmore RM, Rabinstein AA, Chandra R, Serrano LA, Yerragonda N, Palamari B, Decker WW. Effect of anticoagulant and antiplatelet therapy in patients with spontaneous intra-cerebral hemorrhage: does medication use predict worse outcome? *Clin Neurol Neurosurg*. 2010;112(4):275–81. Epub 2009 Dec 29. PMID: 20042270.
9. Morotti A, Goldstein JN. Anticoagulant-associated intracerebral hemorrhage. *Brain Hemorrhages*. 2020;1.
10. Faghih-Jouybari M, Raof MT, Abdollahzade S, Jamshidi S, Padegane T, Ehteshami S, Fateh S. Mortality and morbidity in patients with spontaneous intracerebral hemorrhage: a single-center experience. *Curr J Neurol*. 2021;20.
11. McGurgan IJ, Ziai WC, Werring DJ, Al-Shahi Salman R, Parry-Jones AR. Acute intracerebral haemorrhage: diagnosis and management. *Pract Neurol*. 2021;21.
12. Hillal A, Sultani G, Ramgren B, Norrvig B, Wassélius J, Ullberg T. Accuracy of automated intracerebral hemorrhage volume measurement on non-contrast computed tomography: a Swedish stroke register cohort study. *Neuroradiology*. 2023;65.
13. Goldstein JN, Fazen LE, Snider R, Schwab K, Greenberg SM, Smith EE, Lev MH, Rosand J. Contrast extravasation on CT angiography predicts hematoma expansion in intracerebral hemorrhage. *Neurology*. 2007;68.
14. Wada R, Aviv RI, Fox AJ, Sahlas DJ, Gladstone DJ, Tomlinson G, Symons SP. CT angiography spot sign predicts hematoma expansion in acute intracerebral hemorrhage. *Stroke*. 2007;38.
15. Delgado Almandoz JE, Romero JM. Advanced CT imaging in the evaluation of hemorrhagic stroke. *Neuroimaging Clin N Am*. 2011;21.
16. Demchuk AM, Dowlatshahi D, Rodriguez-Luna D, Molina CA, Blas YS, Dzialowski I, Kobayashi A, Boulanger JM, Lum C, Gubitz G, et al. Prediction of haematoma growth and outcome in patients with intracerebral haemorrhage using the CT-angiography spot sign (PREDICT): a prospective observational study. *Lancet Neurol*. 2012;11.
17. Wijman CAC, Venkatasubramanian C, Bruins S, Fischbein N, Schwartz N. Utility of early MRI in the diagnosis and management of acute spontaneous intracerebral hemorrhage. *Cerebrovasc Dis*. 2010;30.
18. Young N, Vladica P, Soo YS, Ho D. Acute intracerebral haematomas: assessment for possible underlying cause with MRI scanning. *Australas Radiol*. 1993;37.
19. Brouwers HB, Greenberg SM. Hematoma expansion following acute intracerebral hemorrhage. *Cerebrovasc Dis*. 2013;35:195–201.
20. Davis SM, Broderick J, Hennerici M, Brun NC, Diringer MN, Mayer SA, Begtrup K, Steiner T. Hematoma growth is a determinant of mortality and poor outcome after intracerebral hemorrhage. *Neurology*. 2006;66.
21. Brouwers HB, Chang Y, Falcone GJ, Cai X, Ayres AM, Battley TW, Vashkevich A, McNamara KA, Valant V, Schwab K, Orzell SC, Bresette LM, Feske SK, Rost NS, Romero JM, Viswanathan A, Chou SH, Greenberg SM, Rosand J, Goldstein JN. Predicting hematoma expansion after primary intracerebral hemorrhage. *JAMA Neurol*. 2014;71(2):158–64. <https://doi.org/10.1001/jamaneurol.2013.5433>. PMID: 24366060; PMCID: PMC4131760.
22. Brott T, Broderick J, Kothari R, Barsan W, Tomsick T, Sauerbeck L, Spilker J, Duldner J, Khoury J. Early hemorrhage growth in patients with intracerebral hemorrhage. *Stroke*. 1997;28.
23. Dowlatshahi D, Demchuk AM, Flaherty ML, Ali M, Lyden PL, Smith EE. Defining hematoma expansion in intracerebral hemorrhage: relationship with patient outcomes. *Neurology*. 2011;76.
24. Britton M, Carlsson A, de Faire U. Blood pressure course in patients with acute stroke and matched controls. *Stroke*. 1986;17.
25. Chen ST, Chen SD, Hsu CY, Hogan EL. Progression of hypertensive intracerebral hemorrhage. *Neurology*. 1989;39.
26. Kazui S, Minematsu K, Yamamoto H, Sawada T, Yamaguchi T. Predisposing factors to enlargement of spontaneous intracerebral hematoma. *Stroke*. 1997;28.
27. Willmot M, Leonardi-Bee J, Bath PMW. High blood pressure in acute stroke and subsequent outcome: a systematic review. *Hypertension*. 2004;43.
28. Wang X, Di Tanna GL, Moullaali TJ, Martin RH, Shipes VB, Robinson TG, Chalmers J, Suarez JJ, Qureshi AI, Palesch YY, et al. J-shape relation of blood pressure reduction and outcome in acute intracerebral hemorrhage: A pooled analysis of INTERACT2 and ATACH-II individual participant data. *Int J Stroke*. 2022;17:1129–36.
29. Qureshi AI, Palesch YY. Antihypertensive treatment of acute cerebral hemorrhage (ATACH) II: design, methods, and rationale. *Neurocrit Care*. 2011;15:559–76.
30. Anderson CS, Heeley E, Huang Y, Wang J, Stapf C, Delcourt C, Lindley R, Robinson T, Lavados P, Neal B, et al. Rapid Blood-Pressure Lowering in patients with acute intracerebral hemorrhage. *N Engl J Med*. 2013;368:2355–65.
31. Ma L, Hu X, Song L, Chen X, Ouyang M, Billot L, Li Q, Malavera A, Li X, Muñoz-Venturelli P, et al. The third intensive care bundle with blood pressure reduction in acute cerebral haemorrhage trial (INTERACT3): an international, stepped wedge cluster randomised controlled trial. *Lancet*. 2023;402:27–40.
32. Flibotte JJ, Hagan N, O'Donnell J, Greenberg SM, Rosand J. Warfarin, hematoma expansion, and outcome of intracerebral hemorrhage. *Neurology*. 2004;63.
33. Cucchiara B, Messe S, Sansing L, Kasner S, Lyden P. Hematoma growth in oral anticoagulant related intracerebral hemorrhage. *Stroke*. 2008;39.
34. Ha ACT, Bhatt DL, Rutka JT, Johnston SC, Mazer CD, Verma S. Intracranial hemorrhage during dual antiplatelet therapy. *J Am Coll Cardiol*. 2021;78.
35. Hanger HC, Geddes JAA, Wilkinson TJ, Lee M, Baker AE. Warfarin-related intracerebral haemorrhage: better outcomes when reversal includes prothrombin complex concentrates. *Intern Med J*. 2013;43.

36. Steiner T, Poli S, Griebel M, Hüsing J, Hajda J, Freiburger A, Bendszus M, Bösel J, Christensen H, Dohmen C, et al. Fresh frozen plasma versus prothrombin complex concentrate in patients with intracranial haemorrhage related to vitamin K antagonists (INCH): a randomised trial. *Lancet Neurol*. 2016;15:566–73.
37. Mayer SA, Brun NC, Begtrup K, Broderick J, Davis S, Diringer MN, Skolnick BE, Steiner T. Efficacy and safety of recombinant activated factor VII for acute intracerebral hemorrhage. *N Engl J Med*. 2008;358.
38. Sweidan AJ, Singh NK, Conovaloff JL, Bower M, Groysman LI, Shafie M, et al. Coagulopathy reversal in intracerebral haemorrhage. *Stroke Vascular Neurol*. 2020;5. <https://doi.org/10.1136/svn-2019-000274>.
39. Siegal DM, Curnutte JT, Connolly SJ, Lu G, Conley PB, Wiens BL, Mathur VS, Castillo J, Bronson MD, Leeds JM, Mar FA, Gold A, Crowther MA. Andexanet alfa for the reversal of factor Xa inhibitor activity. *N Engl J Med*. 2015;373(25):2413–24. <https://doi.org/10.1056/NEJMoa1510991>. Epub 2015 Nov 11. PMID: 26559317.
40. Connolly SJ, Sharma M, Cohen AT, Demchuk AM, Czlonkowska A, Lindgren AG, Molina CA, Berezcki D, Toni D, Seiffge DJ, Tanne D, Sandset EC, Tsvigoulis G, Christensen H, Beyer-Westendorf J, Coutinho JM, Crowther M, Verhamme P, Amarencu P, Roine RO, Mikulik R, Lemmens R, Veltkamp R, Middeldorp S, Robinson TG, Milling TJ Jr, Tedim-Cruz V, Lang W, Himmelmann A, Ladenvall P, Knutsson M, Ekholm E, Law A, Taylor A, Karyakina T, Xu L, Tsiplova K, Poli S, Kallmünzer B, Gumbinger C, Shoamanesh A. ANNEXA-1 investigators. Andexanet for factor Xa inhibitor-associated acute intracerebral hemorrhage. *N Engl J Med*. 2024;390(19):1745–1755. <https://doi.org/10.1056/NEJMoa2313040>. PMID: 38749032.
41. Anticoagulant Reversal Drug Pulled from U.S. Market. <https://www.medpage.com/publichealthpolicy/fdageneral/119001>. Retrieved December 30, 2025.
42. Pollack CV, Reilly PA, van Ryn J, Eikelboom JW, Glund S, Bernstein RA, Dubiel R, Huisman MV, Hylek EM, Kam C-W, et al. Idarucizumab for Dabigatran Reversal — Full cohort analysis. *N Engl J Med*. 2017;377:431–41.
43. Schulman S, Bijsterveld NR. Anticoagulants and their reversal. *Transfus Med Rev*. 2007;21.
44. Schulman S, Ritchie B, Nahiriak S, Gross PL, Carrier M, Majeed A, Hwang HG, Zondag M. Reversal of dabigatran-associated major bleeding with activated prothrombin concentrate: a prospective cohort study. *Thromb Res*. 2017;152.
45. Ward S, Jamal BC, Ganti L. Frontoparietal intraparenchymal hemorrhage secondary to anticoagulation. *Int J Emerg Med*. 2024;17:130. <https://doi.org/10.1186/s12245-024-00723-0>.
46. Liu J, Nie X, Gu H, Zhou Q, Sun H, Tan Y, Liu D, Zheng L, Zhao J, Wang Y, et al. Tranexamic acid for acute intracerebral haemorrhage growth based on imaging assessment (TRAIge): a multicentre, randomised, placebo-controlled trial. *Stroke Vasc Neurol*. 2021;6.
47. Meretoja A, Yassi N, Wu TY, Churilov L, Sibolt G, Jeng JS, Kleinig T, Spratt NJ, Thijs V, Wijeratne T, et al. Tranexamic acid in patients with intracerebral haemorrhage (STOP-AUST): a multicentre, randomised, placebo-controlled, phase 2 trial. *Lancet Neurol*. 2020;19.
48. Sprigg N, Flaherty K, Appleton JP, Salman RAS, Berezcki D, Beridze M, Christensen H, Ciccone A, Collins R, Czlonkowska A, et al. Tranexamic acid for hyperacute primary intracerebral haemorrhage (TICH-2): an international randomised, placebo-controlled, phase 3 superiority trial. *Lancet*. 2018;391.
49. Rincon F, Mayer SA. Clinical review: critical care management of spontaneous intracerebral hemorrhage. *Crit Care*. 2008;12.
50. Commichau C, Scarmeas N, Mayer SA. Risk factors for fever in the neurologic intensive care unit. *Neurology*. 2003;60.
51. Schwarz S, Häfner K, Aschoff A, Schwab S. Incidence and prognostic significance of fever following intracerebral hemorrhage. *Neurology*. 2000;54.
52. Hajat C, Hajat S, Sharma P. Effects of poststroke pyrexia on stroke outcome: a meta-analysis of studies in patients. *Stroke*. 2000;31.
53. Hervella P, Rodríguez-Yáñez M, Pumar JM, Ávila-Gómez P, da Silva-Candal A, López-Loureiro I, Rodríguez-Maqueda E, Correa-Paz C, Castillo J, Sobrino T, et al. Antihyperthermic treatment decreases perihematomal hypodensity. *Neurology*. 2020;94.
54. Broessner G, Beer R, Lackner P, Helbok R, Fischer M, Pfaußler B, Rhoter J, Küppers-Tiedt L, Schneider D, Schmutzhard E. Prophylactic, endovascularly based, long-term normothermia in ICU patients with severe cerebrovascular disease: bicenter prospective, randomized trial. *Stroke*. 2009;40.
55. den Hertog HM, van der Worp HB, van Gemert HMA, Algra A, Kappelle LJ, van Gijn J, Koudstaal PJ, Dippel DW. The Paracetamol (Acetaminophen) in stroke (PAIS) trial: a multicentre, randomised, placebo-controlled, phase III trial. *Lancet Neurol*. 2009;8.
56. Kollmar R, Staykov D, Dörfler A, Schellinger PD, Schwab S, Bardutzky J. Hypothermia reduces perihemorrhagic edema after intracerebral hemorrhage. *Stroke*. 2010;41.
57. Staykov D, Schwab S, Dörfler A, Kollmar R. Hypothermia reduces perihemorrhagic edema after intracerebral hemorrhage: but does it influence functional outcome and mortality? *Ther Hypothermia Temp Manag*. 2011;1.
58. Staykov D, Wagner I, Volbers B, Doerfler A, Schwab S, Kollmar R. Mild prolonged hypothermia for large intracerebral hemorrhage. *Neurocrit Care*. 2013;18.
59. Godoy DA, Piñero GR, Svampa S, Papa F, Di Napoli M. Hyperglycemia and short-term outcome in patients with spontaneous intracerebral hemorrhage. *Neurocrit Care*. 2008;9.
60. Kimura K, Iguchi Y, Inoue T, Shibasaki K, Matsumoto N, Kobayashi K, Yamashita S. Hyperglycemia independently increases the risk of early death in acute spontaneous intracerebral hemorrhage. *J Neurol Sci*. 2007;255:90–4.
61. Stead LG, Jain A, Bellolio MF, Odufuye A, Gilmore RM, Rabinstein A, Chandra R, Dhillon R, Manivannan V, Serrano LA, et al. Emergency department hyperglycemia as a predictor of early mortality and worse functional outcome after intracerebral hemorrhage. *Neurocrit Care*. 2010;13.
62. Intensive versus Conventional. Glucose control in critically ill patients. *N Engl J Med*. 2009;360.
63. Oddo M, Schmidt JM, Carrera E, Badjatia N, Connolly ES, Presciutti M, Ostapovich ND, Levine JM, Roux P, Le, Mayer SA. Impact of tight glycemic control on cerebral glucose metabolism after severe brain injury: a Microdialysis study. *Crit Care Med*. 2008;36.
64. Wang LC, Lei S, Wu JN, Wang LF, Jiang HF, Ni HX, Ye XH. Intensive insulin therapy in critically ill patients. *Chin Crit Care Med*. 2006;18.
65. Béjot Y, Aboa-Eboulé C, Hervieu M, Jacquin A, Osseby GV, Rouaud O, Giroud M. The deleterious effect of admission hyperglycemia on survival and functional outcome in patients with intracerebral hemorrhage. *Stroke*. 2012;43.
66. Capes SE, Hunt D, Malmberg K, Pathak P, Gerstein HC. Stress hyperglycemia and prognosis of stroke in nondiabetic and diabetic patients: a systematic overview. *Stroke*. 2001;32.
67. Mehta A, Zusman BE, Shutter LA, Choksi R, Yassin A, Antony A, Thirumala PD. The prevalence and impact of status epilepticus secondary to intracerebral hemorrhage: results from the US nationwide inpatient sample. *Neurocrit Care*. 2018;28.
68. Vespa PM, O'Phelan K, Shah M, Mirabelli J, Starkman S, Kidwell C, Saver J, Nuwer MR, Frazee JG, McArthur DA, et al. Acute seizures after intracerebral hemorrhage: a factor in progressive midline shift and outcome. *Neurology*. 2003;60.
69. Angriman F, Tirupakuzhi Vijayaraghavan BK, Dragoi L, Lopez Soto C, Chapman M, Scales DC. Antiepileptic drugs to prevent seizures after spontaneous intracerebral hemorrhage. *Stroke*. 2019;50.
70. Zandieh A, Messé SR, Cucchiara B, Mullen MT, Kasner SE. Prophylactic use of antiepileptic drugs in patients with spontaneous intracerebral hemorrhage. *J Stroke Cerebrovasc Dis*. 2016;25.
71. Spoelhof B, Sanchez-Bautista J, Zorrilla-Vaca A, Kaplan PW, Farrok S, Mirski M, Freund B, Rivera-Lara L. Impact of antiepileptic drugs for seizure prophylaxis on short and long-term functional outcomes in patients with acute intracerebral hemorrhage: a meta-analysis and systematic review. *Seizure*. 2019;69.
72. Broderick JP, Brott TG, Duldner JE, Tomsick T, Huster G. Volume of intracerebral hemorrhage. A powerful and easy-to-use predictor of 30-day mortality. *Stroke*. 1993;24:987–93.
73. Tuhim S, Horowitz DR, Sacher M, Godbold JH. Volume of ventricular blood is an important determinant of outcome in supratentorial intracerebral hemorrhage. *Crit Care Med*. 1999;27.
74. Chan E, Anderson CS, Wang X, Arima H, Saxena A, Moullaali TJ, Heeley E, Delcourt C, Wu G, Wang J, et al. Significance of intraventricular hemorrhage in acute intracerebral hemorrhage intensive blood pressure reduction in acute cerebral hemorrhage trial results. *Stroke*. 2015;46.
75. Akhigbe T, Okafor U, Sattar T, Rawluk D, Fahey T. Stereotactic-guided evacuation of spontaneous supratentorial intracerebral hemorrhage: systematic review and meta-analysis. *World Neurosurg*. 2015;84.
76. Guo G, Pan C, Guo W, Bai S, Nie H, Feng Y, Li G, Deng H, Ma Y, Zhu S, et al. Efficacy and safety of four interventions for spontaneous supratentorial intracerebral hemorrhage: a network meta-analysis. *J Neurointerv Surg*. 2020;12.
77. Hanley DF, Thompson RE, Rosenblum M, Yenokyan G, Lane K, McBee N, Mayo SW, Bistran-Hall AJ, Gandhi D, Mould WA, et al. Efficacy and safety of minimally invasive surgery with thrombolysis in intracerebral haemorrhage evacuation (MISTIE III): a randomised, controlled, open-label, blinded end-point phase 3 trial. *Lancet*. 2019;393.

78. Li M, Mu F, Su D, Han Q, Guo Z, Chen T. Different surgical interventions for patients with spontaneous supratentorial intracranial hemorrhage: a network meta-analysis. *Clin Neurol Neurosurg*. 2020;188.
79. Zhou X, Chen J, Li Q, Ren G, Yao G, Liu M, Dong Q, Guo J, Li L, Guo J, et al. Minimally invasive surgery for spontaneous supratentorial intracerebral hemorrhage: A meta-analysis of randomized controlled trials. *Stroke*. 2012;43.
80. Sondag L, Schreuder FFBM, Boogaarts HD, Rovers MM, Vandertop WP, Dammers R, Klijn CJM. Neurosurgical intervention for supratentorial intracerebral hemorrhage. *Ann Neurol*. 2020;88.
81. Tang Y, Yin F, Fu D, Gao X, Lv Z, Li X. Efficacy and safety of minimal invasive surgery treatment in hypertensive intracerebral hemorrhage: A systematic review and meta-analysis. *BMC Neurol*. 2018;18.
82. Yao Z, Hu X, You C, He M. Effect and feasibility of endoscopic surgery in spontaneous intracerebral hemorrhage: A systematic review and Meta-Analysis. *World Neurosurg*. 2018;113.
83. Pradilla G, Ratcliff JJ, Hall AJ, Saville BR, Allen JW, Paulon G, McGlothlin A, Lewis RJ, Fitzgerald M, Caveney AF, et al. Trial of early minimally invasive removal of intracerebral hemorrhage. *N Engl J Med*. 2024;390:1277–89.
84. Zhou X, Xie L, Altinel Y, Qiao N. Assessment of evidence regarding minimally invasive surgery vs. Conservative treatment on intracerebral hemorrhage: A trial sequential analysis of randomized controlled trials. *Front Neurol*. 2020;11.
85. Parry-Jones AR, Järhult SJ, Kreitzer N, Morotti A, Toni D, Seiffge D, Mendelow AD, Patel H, Brouwers HB, Klijn CJ, et al. Acute care bundles should be used for patients with intracerebral haemorrhage: an expert consensus statement. *Eur Stroke J*. 2023.
86. Parry-Jones AR, Sammut-Powell C, Paroutoglou K, Birleson E, Rowland J, Lee S, Cecchini L, Massyn M, Emsley R, Bray B, et al. An intracerebral hemorrhage care bundle is associated with lower case fatality. *Ann Neurol*. 2019;86:495–503.
87. Middleton S, McElduff P, Ward J, Grimshaw JM, Dale S, D'Este C, Drury P, Griffiths R, Cheung NW, Quinn C, et al. Implementation of evidence-based treatment protocols to manage fever, hyperglycaemia, and swallowing dysfunction in acute stroke (QASC): a cluster randomised controlled trial. *Lancet*. 2011;378:1699–706.
88. Li Q, Yakhkind A, Alexandrov AW, Alexandrov AV, Anderson CS, Dowlatshahi D, Frontera JA, Hemphill JC, Ganti L, Kellner C, et al. Code ICH: A call to action. *Stroke*. 2024;55:494–505.
89. Lun R, Yogendrakumar V, Ramsay T, Shamy M, Fahed R, Selim MH, Dowlatshahi D. Predicting long-term outcomes in acute intracerebral haemorrhage using delayed prognostication scores. *Stroke Vasc Neurol*. 2021;6:536–41.

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