Management of acute intracranial and intraventricular hemorrhage

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Objective: **Acute intracranial hemorrhage and intraventricular hemorrhage are devastating disorders. The goal of this review is to familiarize clinicians with recent information pertaining to the acute care of intracranial hemorrhage and intraventricular hemorrhage.**

Data Sources: **PubMed search and review of the relevant medical literature.**

Summary: **The management of intracranial hemorrhage and intraventricular hemorrhage is complex. Effective treatment should include strategies designed to reduce hematoma expansion and limit the medical consequences of intracranial hemorrhage and intraventricular hemorrhage. At present, there are a number of new approaches to treatment that may reduce mortality and improve clinical outcomes. Clinicians should recognize that patients with large hematomas may make a substantial recovery.**

Conclusions: **Patients with intracranial hemorrhage and intraventricular hemorrhage should be cared for in an intensive care unit. New therapies designed to stabilize hematoma growth and reduce hematoma burden may improve outcomes. (Crit Care Med 2010; 38:946–953)**

KEY WORDS: intracranial hemorrhage; intraventricular hemorrhage; hypertension; treatment; thrombolysis

Intracranial hemorrhage (ICH) is associated with the highest case fatality of any stroke subtype. One month after ICH, 50% of patients will be dead and only 20% of patients will have regained functional independence at 1 yr (1, 2). On presentation, patients with ICH are often only mildly impaired and cognitively intact. Over minutes to hours, the patient's clinical picture can change rapidly as a result of a number of events, including hematoma expansion, cerebral edema, and hydrocephalus. New strategies designed to reduce the impact of these events involve prothrombotic agents, such as factor VIIa (fVIIa), aggressive blood pressure control, and aggressive reversal of prolonged international normalized ratios, to reduce hematoma expansion. New strategies that use recombinant tissue plasminogen activator (rt-PA) as a thrombolytic to reduce clot burden in intraventricular hemorrhage (IVH) and ICH are now being tested and appear to be efficacious. These approaches applied in the critical care setting are changing outcomes in ICH and IVH.

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SUBJECTS AND METHODS

Epidemiology

ICH has a worldwide incidence that varies from 10 to 20 per 100,000 and affects an estimated 37,000 to 52,400 people annually in the United States. It has an associated 40% to 50% 30-day mortality (1–4). ICH occurs twice as often as subarachnoid hemorrhage, more frequently in men than in woman, and more often in certain ethnic populations, such as blacks, Japanese, Mexican Americans, Latin Americans, Native Americans, and Chinese (5–7).

IVH occurs in 12% to 45% of patients with ICH (8–10). It also can occur independently of ICH without a significant parenchymal component. Mortality estimates for IVH in any setting range from 45% to 80% (1, 2). The most common cause of IVH is spontaneous ICH (11). Approximately 40% of patients with primary ICH experience IVH (7, 10, 12, 13). The total annual incidence of IVH in the United States is estimated to be approximately 22,000 adults per year (1, 10, 14). IVH is associated with approximately 15% of the 700,000 strokes that occur annually in the United States (10, 15).

DISCUSSION

Clinical Presentation

The onset of ICH and IVH is apoplectic. Initial symptoms include headache, hemiparesis, altered mental status, and coma. Other lesser symptoms include nausea and vomiting, altered vision, and diplopia. Initially, the patient may be clinically stable with only mild to moderate symptoms. However, after this initial phase, patients often experience a relentless decline that ends in coma and death. Rapidly rising intracranial pressure (ICP) associated with cerebral edema can cause herniation. Blood pressure is usually elevated because of uncontrolled essential hypertension and hypertension associated with systemic catecholamine release presumed to be from the effects of the ICH or IVH on the brain (16).

Patients with supratentorial lesions will present with hemiparesis contralateral to the hemorrhage. Those patients with infratentorial lesions have a more devastating course that progresses rapidly to clinical brain death. Unique infratentorial symptoms include coma, intranuclear ophthalmoplegias, pupillary abnormalities, quadriparesis, and decorticate posturing.

Imaging

Computed tomography (CT) of the brain is the modality of choice for diagnosing ICH and IVH, and it allows for the rapid diagnosis of all types of ICH. Blood is easily identified on CT—it appears as a white hyperdense lesion. CT also can be used to identify other important associated clinical features, such as cerebral edema and hydrocephalus. Magnetic resonance imaging (MRI) is equally effective

Table 1. Evolution of intracranial hemorrhage in the 1.5 Telsa magnetic resonance imaging

ICH Stage	Age of Hematoma	Hemoglobin State	T1 Sequence	T2 Sequence
Hyperacute	<24 hrs	Oxyhemoglobin	Isointense	Slightly hyperintense
Acute	1–3 days	Deoxyhemoglobin	Slightly hypointense	Hypointense
Subacute early	>3 days	Methemoglobin	Very hyperintense	Hypointense
Subacute late	>7 days	Methemoglobin	Very hyperintense	Very hyperintense
Chronic center	>30 days	Hemochromes	Slightly isointense	Slightly hyperintense
Chronic rim	>30 days	Hemosiderin	Hypointense	Very hypointense

ICH, intracranial hemorrhage.
Adapted from Allkemper et al: *Radiology* 2004; 232:874–881.

at identifying acute hemorrhage. It can readily distinguish between hemorrhage and acute ischemic stroke, although it is rarely obtained in the emergency department (17). Special MRI images called gradient echo images allow for easy identification of blood regardless of hematoma age or the heme oxidation state. The appearance of hemoglobin on the T1- and T2-weighted images from patients with acute ICH changes at different times. This chronology is outlined in Table 1.

Pathophysiology

ICH can occur in all locations of the brain, whereas primary IVH occurs most often as the result of hemorrhage of the thalamus and caudate nucleus (8). The areas of the brain most frequently involved with ICH are the basal ganglia, thalamus, brainstem, cerebellum, and cerebral cortex. In general, ICH is a disorder of the small and medium arteries of the brain, on average 70–300 μm in diameter (18). Areas susceptible to ICH are perfused by arterial systems composed of small arteries such as the lenticulostriate, thalamogeniculate, paramedium, and circumlinear arteries of the brain stem (4, 18, 19). After hemorrhage, blood disperses between the ascending fiber tracts within the parenchyma of the brain. In these areas there are locations of preserved brain surrounded by necrotic tissue. Hemoglobin, thrombin, and their metabolites disrupt the molecular and cellular activity of tissue surrounding the hematoma, induce inflammation, and disrupt the integrity of the blood–brain barrier (18, 20–26). These activities facilitate cerebral edema and damage neurons directly (18, 27, 28).

Numerous studies have identified risk factors and underlying pathologic changes associated with ICH and IVH. Over time, the integrity of affected vessels declines as a result of damage from hypertension, protein deposition, tumors, or congenital defects. The underlying pathophysiologic damage that leads to ICH has been identified as a subintimal change in the submuscularis zone and occurs most frequently at the bifurcation of small arteries (29–31). This damage is characterized by fibrin deposition and disruption of the elastic lamina, as well as vacuolization and necrosis of smooth muscle cells associated with dissection of the walls of small vessels (18, 29, 32). Charcot-Bouchard microaneurysms are small vascular abnormalities that have been described in this context. When ICH occurs, it usually does not occur in one small vessel alone, but rather in many similar small vessels distributed across a single vascular distribution (31).

Risk Factors

The most important epidemiologic risk factor in ICH is hypertension (33, 34). This risk is augmented by smoking (33, 34). Other important risk factors include alcohol, hypocholesterolemia, and genetic factors (4, 35, 36). Both therapeutic and illicit drug use are associated with ICH. Warfarin use is a common risk factor for ICH and is associated with larger hematomas (37, 38). Over-the-counter vasoconstrictors such as phenylpropylamine are associated with ICH (39). Illicit drugs associated with ICH include cocaine and methamphetamine (40). Common causes of secondary ICH include vascular malformations, arteriovenous malformations, cavernous angiomas, small arterial telangiectasia, and primary and secondary brain tumors. Different tumor types along with other common causes of ICH are listed in Table 2.

Cerebral Amyloid Angiopathy

Amyloid angiopathy is a common cause of ICH in the elderly. It is visualized by birefringent green, Congo red staining of β-amyloid deposition present in the walls of small to medium cerebral arteries (4). Special consideration of this disorder should be given in the setting of lobar hemorrhages, particularly if they occur in a posterior parietal or occipital distribution on CT or MRI. Cerebral amyloidosis occurs as a sporadic form and as hereditary Icelandic and Dutch forms that are attributed to genetic errors in the amyloid precursor protein (41–43). Alzheimer disease is associated with amyloid deposition resulting in ICH (44). Disorders associated with peripheral vascular deposition of amyloid do not commonly cause ICH (45). Central nervous system amyloidosis is identified by the association of microhemorrhages, which are detected by gradient echo-weighted MRI in patients with lobar ICH (46).

Secondary Imaging

Four-vessel digital angiography and MRI are used to detect occult vascular defects and other lesions that may have caused ICH. In general, if a patient is older than age 50 yrs with a known history of hypertension, and if the ICH occurs in the basal ganglia, a CT scan is all that is necessary (47). In patients younger than age 50 yrs, or in patients with unusual hemorrhages, a four-vessel digital angiogram and MRI should be completed during the initial hospitalization. CT angiographies are often used instead of traditional angiography, particularly as the first screening tool for ICH. Initial studies suggest that they are nearly as effective as standard angiograms in screening ICH for underlying vascular abnormalities (48). However, the sensitivity of CT angiography for the detection of vascular abnormalities involving small blood vessels has not been well-established, and many centers rely on conventional four-vessel digital angiograms. These modalities allow for the detection of underlying disorders that cause hemorrhages, such as ischemia, amyloidosis, arteriovenous malformations, dural arteriovenous fistulas, cavernous angiomas, arterial telangiectasias, brain tumors, and aneurysms. If nothing is found, and if the region of interest is obscured by the hematoma, then these tests should be repeated in 6 wks to 90 days to allow time for the hematoma to be absorbed.

Table 2. Other causes of intracranial hemorrhage and mode of diagnosis

Cause of ICH	Mode of Diagnosis
Hypertension	CT, classic location, clinical history
Warfarin	Head CT, clinical history
Cocaine, methamphetamine, over-the-counter drugs	Head CT, drug test, clinical history
Amyloid angiopathy	MRI, clinical history
Subarachnoid hemorrhage	CT, angiogram, subarachnoid blood, clinical history
Cavernous angioma	MRI, clinical history
Venous angioma	MRI, clinical history, conventional angiography
Arteriovenous malformation	MRI, conventional angiography, clinical history
Dural arteriovenous fistula	MRI, conventional angiography, clinical history
Tumor: melanoma, renal cell, breast, lung, colon	MRI with gadolinium, CT with contrast, blush on conventional angiography, location in grey–white junction and cerebellum
Sagittal sinus thrombosis or transverse sinus thrombosis	CT, hyperdense sagittal sinus, MRI, clot visualized in sinus, enhanced CT or MRI: empty delta sign, conventional angiography

ICH, intracranial hemorrhage; CT, computed tomography; MRI, magnetic resonance imaging. Adapted from Qureshi et al: *N Engl J Med* 2001; 344:1450–1460.

Mortality Prediction

Modern trials have revealed mortality rates for ICH in the range of 18% at 30 days, substantially lower than the 23% to 58% reported in older studies (3, 4, 49–53). Patients treated in intensive care units (ICU) by clinicians with experience in the care of ICH and IVH have better outcomes (54, 55). In young patients with ICH caused by underlying lesions, such as tumors, chronic and acute ischemic strokes, and preeclampsia, most models of mortality do not apply (56, 57). The number one cause of mortality in ICH is withdrawal of care. The possibility of "self-fulfilling prophecies" should be considered in all patients with ICH in which withdrawal of support is contemplated (53).

A number of prospective observational studies have validated outcome scales that aid in counseling families about predicated mortality and morbidity in ICH caused by hypertension (6, 10, 50, 58). The ICH score is perhaps the best-validated scale (58, 59). This scale incorporates estimated hematoma size, Glasgow Coma Scale (GCS), the presence of IVH, and age to help estimate the probability of death and disability (Table 3).

Management

The patient's airway should be evaluated in the emergency department. An endotracheal tube is placed in an estimated 30% of all patients with cortical ICH and in most patients with infratentorial ICH (60, 61). A CT or MRI should be completed emergently, and all preliminary laboratory tests should be performed. Emergent therapy for blood pressure management or reversal of anticoagulation should be initiated. Neurosurgical evaluation for potential surgical interventions such as intraventricular catheter placement or removal of a clot located in a superficial location such as the cortex should be performed as soon as possible, preferably in the emergency department. After initial evaluation and stabilization, the patient should be transported to the ICU.

Clot Stabilization

Stabilization of hematoma growth is the goal of acute ICH management. Data from recent observational and interventional trials suggest that the mortality hazard ratio increases 1.05 points for every 10% increase in hematoma size (9, 62–65). Hematoma expansion within the first 24 hrs occurs in 38% of ICH patients (63). Estimates of this figure vary from 20% to 72.9% in different studies (9, 50, 63, 65). In 33% of those patients who experience hematoma expansion within the first 24 hrs, the most rapid phase occurs within the first hour after the initial bleed (63). This observation suggests a window for early aggressive intervention.

The recent trial called Efficacy and Safety of Recombinant Activated Factor VII for Acute Intracerebral Hemorrhage used 80 μg/kg of fVIIa within 3 hrs of onset to reduce hematoma expansion in cases of acute ICH. This procedure successfully reduced clot growth as measured by CT. However, the study failed to demonstrate reductions in severe disability or death, which are the primary outcome measures of the study. The drug fVIIa currently is not approved for use in ICH by the Food and Drug Administration (51, 66, 67).

Subgroup analysis offers opportunities for future trials with fVIIa in patients with acute ICH. Retrospective analysis of previous clinical trials suggests that patients treated with fVIIa who were age 70 yrs or younger and had baseline ICH volume <60 mL, IVH volume <5 mL, and time from onset to treatment ≤ 2.5 hrs may have had improved clinical outcomes (66). Other retrospective reviews indicate that treatment with fVIIa may reduce the cost of hospitalization (68). At this time an ongoing NINDS trial (NCT00810888) called The Spot Sign for Predicting and Treating ICH Growth Study to test the efficacy of fVIIa in such patients is underway. This study identifies patients with active hematoma growth through extravasation of dye in the acute hematoma (spot sign) on CT angiography (11, 69, 70).

Warfarin Treatment and Hematoma Growth

ICH is often a side effect of treatment with warfarin. Studies have shown that international normalized ratio reversal with fresh-frozen plasma or vitamin K takes many hours (71, 72). The use of such agents as procoagulant complexes and fVIIa at the 20 μg/kg dose can reverse prolonged international normalized ratios in minutes. Their use in patients with acute ICH who are using warfarin has been advocated (73–76). At present, the literature lacks clinical trials that demonstrate the efficacy of these agents to change clinical outcomes in ICH in the setting of prolonged international normalized ratio. A clinical trial called Factor VII, Prothrombin Complex Concentrate, and Fresh-Frozen Plasma in Warfarin-Related Intracranial Hemorrhage began in 2008 and is ongoing (72, 77, 78).

Deep Venous Thrombosis Prophylaxis

Deep venous thrombosis occurs in 1.6% of all patients with ICH (1). Therefore, deep venous thrombosis prophylaxis should be used because it is an important part of the management of any stroke patient. The use of subcutaneous heparin, 5000 units two or three times daily, or

Table 3. Intracranial hemorrhage score

Component	ICH Score Points
GCS score	
3–4	2
5–12	1
13–15	0
ICH volume, cm^3[a]	
≥30	1
<30	0
IVH	
Yes	1
No	0
Infratentorial origin of ICH	
Yes	1
No	0
Age	
≥80	1
<80	0
Total ICH score	0–6[b]

ICH, intracranial hemorrhage; GCS, Glasgow Coma Scale; IVH, intraventricular hemorrhage.

[a]ICH volume is determined by the formula (clot height × clot width × clot width)/2 (50). This formula is an estimate of the volume of the clot based on the formula for a volume of a sphere. The height of the bleed is determined by counting the number of slices that contain blood. The width is determined by measuring the width of the widest slice. The length is determined by measuring the slice with the largest length; [b]thirty-day mortality rates for patients with ICH scores of 1, 2, 3, and 4 were 13%, 26%, 72%, and 97%, respectively. Adapted from Hemphill et al: *Stroke* 2001; 32:891–897.

Table 4. Recommended guidelines from the American Heart Association for treating elevated blood pressure in spontaneous intracranial hemorrhage

If SBP is >200 mm Hg or MAP is >150 mm Hg, then consider aggressive reduction of blood pressure with continuous intravenous infusion, with frequent blood pressure monitoring every 5 mins
If SBP is >180 mm Hg or MAP is >130 mm Hg and there is evidence of or suspicion of elevated ICP, then consider monitoring ICP and reducing blood pressure using intermittent or continuous intravenous medications to keep cerebral perfusion pressure >60 to 80 mm Hg
If SBP is >180 mm Hg or MAP is >130 mm Hg and there is no evidence of or suspicion of elevated ICP, then consider a modest reduction of blood pressure (e.g., MAP of 110 mm Hg or target blood pressure of 160/90 mm Hg) using intermittent or continuous intravenous medications to control blood pressure; clinically reexamine the patient every 15 mins

ICP, intracranial blood pressure; MAP, mean arterial blood pressure; SBP, systolic blood pressure. Adapted from Broderick et al: *Circulation* 2007, 116:e391–e413 (1).

Table 5. Intravenous medications that may be considered for control of elevated blood pressure in patients with intracranial hemorrhage as recommended by the American Heart Association

Drug	Intravenous Bolus Dose	Continuous Infusion Rate
Labetalol	5–20 mg every 15 mins	2 mg min^{-1} (maximum 300 mg/day)
Nicardipine	NA	5–15 mg hr^{-1}
Esmolol	250 μg kg^{-1} IVP loading dose	25–300 μg kg^{-1} min^{-1}
Enalapril	1.25–5 mg IVP every 6 hrs[a]	NA
Hydralazine	5–20 mg IVP every 30 mins	1.5–5 μg kg^{-1} min^{-1}
Nipride (140, 141)	NA	0.1–10 μg kg^{-1} min^{-1}

IVP, intravenous push; NA, not applicable.

[a]Because of the risk of precipitous blood pressure lowering, the first test dose for enalapril should be 0.625 mg. Adapted from Broderick et al: *Circulation* 2007; 116:e391–e413 (1).

low-molecular-weight heparin, 40 mg once daily, is considered safe in such patients. These medication regimens are usually implemented within 24 hrs after acute ICH, when the clot has stopped enlarging and blood pressure is stable (1).

Blood Pressure Reduction and Hematoma Growth

Debate about the control of blood pressure in acute ICH and IVH focuses on two issues: hematoma growth and salvage of hypoperfused tissue surrounding the hematoma. Mayer et al (79) published a CT perfusion study identifying an ischemic penumbra in patients with acute ICH. Since that early work, three studies using CT perfusion, positron emission tomography, and MRI perfusion have been completed. None was able to identify an ischemic penumbra in acute ICH (80–83).

The association of high blood pressure and hematoma expansion is controversial. High blood pressure in patients with ICH may cause increased bleeding and hematoma growth. Two studies have failed to show increased blood pressure as a risk factor for hematoma expansion (84–86). Yet, another observational study suggests that lowering blood pressure in patients with ICH may help to reduce neurologic deterioration (87). Two other prospective randomized controlled studies—Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage trial (INTERACT) and Antihypertensive Treatment of Acute Cerebral Hemorrhage (ATACH)—have not demonstrated changes in clinical outcomes with intensive blood pressure management (88–90).

American Heart Association guidelines on ICH management advocate appropriate blood pressure control. These guidelines are outlined in Table 4. In addition, the agents used to control blood pressure should be chosen carefully. Agents such as nitroprusside are known to elevate ICP when brain injury is present (1). Agents considered safe in acute ICH are listed in Table 5.

Cerebral Edema and ICP Management

In acute ICH and IVH, cerebral edema is a predictor of mortality (64, 91, 92). The natural history of cerebral edema in ICH is characterized by an initial rapid increase in volume over the first 72 hrs, followed by a period of continued slow edema growth for at least 7 days after the initial bleed. If elevated ICP or herniation is present, then immediate action to reduce ICP is required. The first measure is controlled hyperventilation to a $Paco_2$ of 25 to 35 mm Hg. This method decreases ICP rapidly and effectively. Prophylactic or chronic hypoventilation is associated with worse outcomes, and hyperventilation should be used only in acute cases as a temporizing measure (93).

Osmotherapy can decrease cerebral edema from ICH in a limited fashion. Cerebral edema in ICH is often resistant to treatment with traditional osmotherapy (94). The use of prophylactic osmotic therapy with mannitol to prevent the development of cerebral edema in ICH does not improve outcomes (95–97). Mannitol is usually the first osmotic agent administered because it can be given through an intravenous line at a dose of 0.5–1.0 mg/kg with a serum osmolality goal of >310 mOsm/kg. Substantial data support the effectiveness of mannitol in reducing ICP, but no data suggest that it alters clinical outcomes in ICH or IVH (1). Hypertonic saline is another osmotic agent used to reduce ICP.

It can be administered in 3%, 7%, or 23% solutions through a central line. It is administered as a bolus (23%) or as a constant infusion of 2% or 3%. The usual serum sodium goal is 145–155 mmol/L but can be as high as 160 mmol/L (1, 95–97). Data do exist to support its effectiveness in reducing ICP, but no evidence suggests that it alters clinical outcomes in ICH or IVH (1).

For refractory elevations in ICP, additional options include pharmacologically induced coma or decompressive hemicraniectomy (98–100). At present, there is no indication for the internal or external surgical decompression of hematoma in the setting of supratentorial ICH. Only one case series has been published addressing this issue (100). Pharmaceutical coma requires the use of anesthetics such as propofol or pentobarbital at high doses. Data that adequately describe the clinical effectiveness of this strategy are lacking (1). Steroids have no role in the management of cerebral edema or increased ICP (1, 101).

Temperature Control

The role for induced hypothermia to manage ICP in patients with ICH and IVH is unclear (1). Whereas animal data suggest that hypothermia may reduce neurologic injury and help to control ICP, small human trials indicate that induced hypothermia is not effective at altering clinical outcomes (1). Although the use of hypothermia in humans may have some efficacy in reducing ICP, it is associated with a large number of delayed sequelae associated with rewarming (102, 103).

Seizure Management

Overt seizures occur in 10% to 15% of patients after ICH. Recent continuous electroencephalography data suggest a higher prevalence of electrographic seizures in the range of 60% (104–106). Currently, the use of an antiepileptic drug for prophylaxis is accepted for the acute period after all ICH, although no large randomized trials have been completed to verify its efficacy (1). In the absence of seizures, antiepileptic drug prophylaxis is discontinued 2–4 wks after the initial hemorrhage (107). Antiepileptic drug prophylaxis in hemorrhages of the basal ganglia and infratentorial region appear to be unnecessary (108).

Surgical Management

At present, aggressive surgical management to evacuate the clot in patients with acute ICH is not indicated, except in cases of cerebellar ICH (109, 110). The International Surgical Trial in Intracerebral Hemorrhage (ISTICH) suggested that there was no clinical benefit from conventional surgical clot evacuation when compared with conservative medical management in acute ICH (111–113). A new clinical trial (STICH 2) is ongoing to test the hypothesis that minimally invasive surgical evacuation of cortical clots may be beneficial (114, 115). Several trials have suggested that clinical outcomes improve with surgery designed to reduce clot burden in patients with cortical hematomas (115, 116). Others have reported increased risks associated with this approach (117). The management of other complications, such as hydrocephalus and IVH, require surgical intervention that can include placement of an intraventricular catheter (1, 10, 19, 91). An intraventricular catheter reduces increased ICP caused by hydrocephalus and improves clot drainage in IVH (118). Patients who have cerebellar ICH with hematomas >40 mL in volume or who have a GCS <14 require early surgical intervention (119). Neurosurgical consultation for posterior decompression of large cerebellar hematomas should always be considered for patients with cerebellar ICH (1, 119–121).

Future Directions

Treatment strategies that focus on the reduction of hematoma burden through clot thrombolysis may improve outcomes for patients with ICH and IVH. Thrombolysis with rt-PA is used to reduce hematoma volume in the ventricles and parenchyma. Although urokinase was initially used for this purpose, rt-PA has become the thrombolytic of choice (122–130). Both animal and human models support the efficacy of rt-PA for clot lysis and removal (131–134). Hanley et al (135, 136) have completed a study in which rt-PA was used to facilitate removal of clots for the treatment of IVH; they demonstrated safety and efficacy as well as improved outcomes when compared with historic controls. These findings have led to a large, ongoing, National Institutes of Health, randomized prospective trial of intraventricular rt-PA in IVH (NCT00784134).

Minimally invasive surgery and reduction of hematoma volume through thrombolysis may also be effective in reducing clot burden. This approach incorporates a catheter placed stereotactically into the hematoma through which thrombolytics are infused and the dissolved clot is removed. Current trials that use rt-PA as the thrombolytic have demonstrated the efficacy and safety of this technique for hematoma reduction in acute ICH but have not demonstrated clinical improvement (135, 137–139).

CONCLUSION

ICH and IVH are the subtypes of stroke associated with the highest rates of morbidity and mortality. Support of these patients in an ICU staffed by clinicians who are familiar with these diseases is the best strategy for optimization of clinical outcomes. New techniques emphasizing hematoma stabilization and clot reduction are on the horizon.

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