TABLE 446-7 THE ICH SCORE

Clinical or Imaging Factor	Point Score
Age	
<80 years	0
≥80 years	1
Hematoma Volume	
<30 cc	0
≥30 cc	1
Intraventricular Hemorrhage Present	
No	0
Yes	1
Infratentorial Origin of Hemorrhage	
No	0
Yes	1
Glasgow Coma Scale Score	
13–15	0
5–12	1
3–4	2
Total Score	Sum of each category above

ICH Score Total	% Mortality at 30 Days (95% CI)	% Walk Independently at 12 Months (95% CI)
0	0 (0–13)	70 (53–84)
1	13 (4–29)	60 (47–73)
2	26 (11–46)	33 (21–48)
3	72 (53–86)	13 (5–25)
4	97 (82–100)	3 (0–16)
5	100 (54–100)	8 (0–38)

Note: Although an ICH Score of 6 is possible with the scale, this is rarely observed and is considered highly likely to be fatal.

Abbreviations: CI, confidence interval; ICH, intracerebral hemorrhage.

Sources: JC Hemphill et al: Stroke 32:891, 2001; JC Hemphill et al: Neurology 73:1088, 2009.

Hematomas may expand for several hours following the initial hemorrhage, even in patients without coagulopathy. However, the precise mechanism is unclear. A phase 3 trial of treatment with recombinant factor VIIa reduced hematoma expansion; however, clinical outcomes were not improved, so use of this drug cannot be advocated at present. The theoretical risk of acutely elevated blood pressure on hematoma expansion forms the basis of the consideration for recently completed and ongoing clinical trials of acute blood pressure lowering.

Evacuation of supratentorial hematomas does not appear to improve outcome for most patients. The International Surgical Trial in Intracerebral Haemorrhage (STICH) randomized patients with supratentorial ICH to either early surgical evacuation or initial medical management. No benefit was found in the early surgery arm, although analysis was complicated by the fact that 26% of patients in the initial medical management group ultimately had surgery for neurologic deterioration. The follow-up study STICH-II found that surgery within 24 h of lobar, supratentorial hemorrhage did not improve overall outcome, but might have a role in select severely affected patients. Therefore, existing data do not support routine surgical evacuation of supratentorial hemorrhages in stable patients. However, many centers still consider surgery for patients deemed salvageable and who are having progressive neurologic deterioration due to herniation. Surgical techniques continue to evolve, and minimally invasive endoscopic hematoma evacuation is currently being investigated in clinical trials.

For cerebellar hemorrhages, a neurosurgeon should be consulted immediately to assist with the evaluation; most cerebellar hematomas >3 cm in diameter will require surgical evacuation. If the patient is alert without focal brainstem signs and if the hematoma is <1 cm in diameter, surgical removal is usually unnecessary. Patients with hematomas between 1 and 3 cm require careful observation for signs of impaired consciousness, progressive hydrocephalus, and precipitous respiratory failure. Hydrocephalus due to cerebellar hematoma should not be treated solely with ventricular drainage.

Tissue surrounding hematomas is displaced and compressed but not necessarily infarcted. Hence, in survivors, major improvement commonly occurs as the hematoma is reabsorbed and the adjacent tissue regains its function. Careful management of the patient during the acute phase of the hemorrhage can lead to considerable recovery.

Surprisingly, ICP is often normal even with large ICHs. However, if the hematoma causes marked midline shift of structures with consequent obtundation, coma, or hydrocephalus, osmotic agents can be instituted in preparation for placement of a ventriculostomy or parenchymal ICP monitor (Chap. 330). Once ICP is recorded, CSF drainage (if available), osmotic therapy, and blood pressure management can be tailored to the individual patient to keep cerebral perfusion pressure (MAP minus ICP) above 60 mmHg. For example, if ICP is found to be high, CSF can be drained from the ventricular space and osmotic therapy continued; persistent or progressive elevation in ICP may prompt surgical evacuation of the clot. Alternately, if ICP is normal or only mildly elevated, interventions such as osmotic therapy may be tapered. Because hyperventilation may actually produce ischemia by cerebral vasoconstriction, induced hyperventilation should be limited to acute resuscitation of the patient with presumptive high ICP and eliminated once other treatments (osmotic therapy or surgical treatments) have been instituted. Glucocorticoids are not helpful for the edema from intracerebral hematoma.

PREVENTION

Hypertension is the leading cause of primary ICH. Prevention is aimed at reducing chronic hypertension, eliminating excessive alcohol use, and discontinuing use of illicit drugs such as cocaine and amphetamines. Patients with amyloid angiopathy should generally avoid OACs, but antiplatelet agents may be administered if there is an indication based on atherothrombotic vascular disease.

VASCULAR ANOMALIES

Vascular anomalies can be divided into congenital vascular malformations and acquired vascular lesions.

CONGENITAL VASCULAR MALFORMATIONS

True *arteriovenous malformations* (AVMs), venous anomalies, and capillary telangiectasias are lesions that usually remain clinically silent through life. AVMs are probably congenital, but cases of acquired lesions have been reported.

True AVMs are congenital shunts between the arterial and venous systems that may present with headache, seizures, and intracranial hemorrhage. AVMs consist of a tangle of abnormal vessels across the cortical surface or deep within the brain substance. AVMs vary in size from a small blemish a few millimeters in diameter to a large mass of tortuous channels composing an arteriovenous shunt of sufficient magnitude to raise cardiac output and precipitate heart failure. Blood vessels forming the tangle interposed between arteries and veins are usually abnormally thin and histologically resemble both arteries and veins. AVMs occur in all parts of the cerebral hemispheres, brainstem, and spinal cord, but the largest ones are most frequently in the posterior half of the hemispheres, commonly forming a wedge-shaped lesion extending from the cortex to the ventricle.

Bleeding, headache, and seizures are most common between the ages of 10 and 30, occasionally as late as the fifties. AVMs are more frequent in men, and rare familial cases have been described. Familial AVM may be a part of the autosomal dominant syndrome of hereditary hemorrhagic telangiectasia (Osler-Rendu-Weber) syndrome due to mutations in either endoglin or activin receptor-like kinase 1, both involved in transforming growth factor (TGF) signaling and angiogenesis.