COMPLICATION AVOIDANCE AND MANAGEMENT

Patients with aneurysmal SAH are at risk of various complications, which can generally be considered neurologic or nonneurologic. Some of the more significant and prevalent complications (and corresponding management considerations) are discussed in this section.

NEUROLOGIC COMPLICATIONS

Vasospasm. Cerebral vasopasm is the delayed narrowing of large capacitance arteries and arterioles that occurs after SAH. Typically, it develops 3 to 5 days after hemorrhage and is maximal after 7 to 10 days. It abates over the course of 2 to 3 weeks (Fig. 23). There is no apparent sex predeliction, and the incidence of vasospasm does not appear to vary with age. In most series, the incidence of symptomatic vasospasm occurring after aneurysmal SAH is in the range of 25% to 30% (Awad LA., Barnett G.H., 1994). As many as 70% of patients undergoing angiography at 1-2 weeks after SAH, manifested by vasospasm. Apporoximately 50% of patients with symptomatic vasospasm will develop infarction despite therapy/

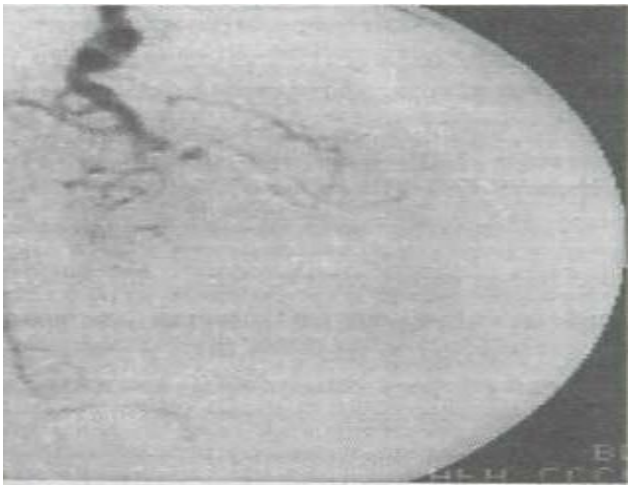

Fig. 2. Transcranial doppler (TCD) study of patient with subarachnoid hemorrhage (SAH) who underwent clipping of *a* giant left middle cerebral artery aneurysm 5 days after SAH (A) and 12 days after SAH (B). (C) Angiogram demonstrating vasospasm in a patient who had undergone clipping of a left middle cerebral artery aneurysm.

Although many potential risk factors for development of vasospasm have been identified, the amount and location of subarachnoid blood visualized on CT scan is the most significant. Patients with thick clot in the basal cisterns are considerably more likely to develop vasospasm than those who have thin scant amounts of subarachnoid blood. Despite a wealth of observational and experimental data, the substance causing vasospasm is not known. One or more components of subarachnoid blood or its breakdown products probably are responsible (Inagawa T, 1992). Onset of symptomatic vasospasm is usually heralded by worsening headache or diminished level of consciousness. Less frequently, patients may develop focal neurological deficits. They may also experience fever or meningeal signs. Transcranial Doppler (TCD) ultra sonography can be useful to diagnose vasospasm (Fig. 2). Peak TCD blood flow velocities greater than 200 cm/s and/or mean velocities greater than 120 cm/s correlate well with angiographically severe vasospasm (Inagawa T., 1992). TCD can evaluate only certain segments of the intracranial vasculature.

Catheter angiography is more sensitive than TCD in evaluating vasospasm, but it is also more invasive, more expensive, and less convenient. The value of angiography in patients with vasospasm is not so much that of a diagnostic study but rather as an aid in endovascular therapy. Transluminal angioplasty has been shown to lead to significant clinical and radiographic improvement in many patients (Mayberg M.R. et al., 1994). Complications, including aneurysmal or parent vessel rupture, occur in about 5% of cases. For patients in diffuse spasm who have worsened despite best medical management, treatment with intraarterial papaverine can be considered. As with transluminal angioplasty, preliminary results of such treatment are encouraging, although its effects are short-lived (particularly as compared with those of angioplasty). The two therapies must be compared in controlled trials. They can be used judiciously in individual cases when best medical management for spasm appears to be failing but before infarction is evident on imaging studies.

The mainstay of treatment of patients in cerebral vasospasm is **hypertensive, hypervolemic nemo dilution.** Several reports have documented improved outcome for patients receiving **«triple H» therapy** as compared with historical controls. Increasing circulating blood volume, raising mean arterial pressure (MAP), and reducing the hematocrit (Hct) level to (30% to 35%) are believed to optimize cerebral perfusion. This treatment can be used more intensively (systolic blood pressure to 200 mm Hg) in patients

who have had their aneurysms occluded. Triple H therapy is administered in an intensive care unit (ICU) (Awad I.A., Barnett G.H., 1994). Generally, the patient has an arterial line, urinary bladder catheter, pulse-oximeter, and a central venous or pulmonary artery catheter. Clinical examination and TCD studies help guide treatment. CT scans can be made to rule out infarction or other adverse sequelae. Treatment is continued until the patient shows clinical improvement, at which time therapy can be gradually discontinued.

Calcium channel antagonists have been shown to reduce the incidence of death due to vasospasm and of poor clinical outcome in many studies (Haley E.J., Kassell N.F., Torner J.C., 1993). In the United States, oral nimodipine is the drug that has been used, with few reported complications or side effects. Other agents currently being evaluated for treatment of patients in vasospasm include intracisternal fibrinolytics, antioxidants, and antiinflammatory medications.

Rebleeding. Initial and recurrent bleeding appear to be the major causes of death following SAH. In a recent study, Broderick et al. (1992) reported that 36 of 80 (45%) patients who had had aneurysmal SAH died within 30 days of the event. Eight of the 36 patients died as a result of rebleeding. Mortality rates associated with rebleeding are higher than those associated with the initial bleed, with the case-fatality rate approximating 70% (Mayberg M.R. et al., 1994). The Cooperative Aneurysm Study demonstrated that rebleeding is maximal in the first 24 hours after aneurysmal SAH (approximately 4%) and decreases to 1% to 2% a day for the first 2 weeks. After the first month, the rate stabilizes at about 3% a year. To prevent rebleeding, the clinician must focus on pertinent risk factors. The patient's blood pressure must be closely monitored (i.e., arterial line) and controlled with intravenous antihypertensive medication and mild sedation. Patients are treated with stool softeners and anticonvulsants. If a patient is stable, expeditious angiography is warranted.

Hydrocephalus. Acute hydrocephalus occurs in approximately 25% of patients with aneurysmal SAH, most often with intra ventricular blood and/or blood in the ambient cisterns. Although acute hydrocephalus can lead to rapid neurological deterioration, most patients exhibit a decline in their level of consciousness in 1 to 2 days. Placement of a ventriculostomy is warranted in such patients, and most will show clinical improvement after this procedure is performed. Every attempt should be made to avoid overdrainage of CSF (maintain TCP 15 to 30 mm Hg), which might precipitate rebleeding. Infection (ventriculitis, meningitis) is also a concern, occurring in 5% to 10% of cases with prolonged ventricular drainage (Bogdahn U. et al., 1992). Prophylactic antibiotics are administered routinely. To reduce the risk of infection, removal of the ventriculostomy catheter is recommended as soon as drainage is no longer necessary. When prolonged diversion of CSF is necessary, use of an indwelling shunt is indicated. Many patients manifest chronic (communicating) hydrocephalus. Such patients often benefit from placement of a ventriculoperitoneal or lumboperitoneal shunt.

Seizures. There is a 10% to 25% incidence of seizures in patients with aneurysmal SAH (King W A, Martin N.A., 1994). Because a seizure in a patient with an untreated aneurysm might cause rebleeding, prophylactic anticonvulsant therapy is indicated for all patients. The optimal duration of anticonvulsant therapy is not known. Long-term administration (>1 week) does not appear to be necessary, unless the patient has had documented seizure activity or has sustained intraparenchymal brain injury.

NON-NEUROLOGIC COMPLICATIONS

Cardiac. Electrocardiographic (ECG) changes are evident in 50% to 90% of patients who have had aneurysmal SAH. Thirty-seven percent to 80% of patients studied will have abnormally high creatinine kinase-myocardial fraction (CK-MB) levels (Lanzino G., Kongable G.L., Kassell N.F., 1994). These changes are particularly evident within the first 72 hours after hemorrhage. Left ventricular wall motion abnormalities are observed in 10% to 30% of patients. Cardiac complications appear to be more common in the elderly and in those who have more severe neurologic deficits. These complications are believed to result from excessive activation of the sympathetic nervous system, with catecholamine-mediated myocardial injury. Because hemodynamic instability occurs most often in the acute phase after aneurysmal SAH, it is imperative that all patients receive continuous cardiac monitoring at the outset. A pulmonary artery (Swan-Ganz) catheter is very useful in evaluation and treatment. Beta-blockers are the medication of choice for most patients with cardiac instability or injury because they control ventricular tachyarrythmias and have a subendocardial protective effect.

Life-threatening arrhythmias have been reported and appear to be most common in the elderly and in the setting of hypokalemia and QT prolongation. If patients are appropriately managed through the acute phase of SAH, most cardiac complications are self-limited and largely reversible.

Pulmonary. Patients who have had aneurysmal SAH may develop pulmonary complications for a variety of reasons. Those who are of poor neurologic grade (and by definition of the **brain deficiency:** stuporous, soporous or comatose) are at increased risk for atelectasis, aspiration, and pneumonia.
Positive airway pressure, bronchodilators, chest physiotherapy, and frequent suctioning are critically important. If pneumonia develops, culture-specific antibiotics should be administered expeditiously.

Neurogenic pulmonary edema (NPE) is a complication that occurs when a patient has sustained significant neurologic insult. It is characterized by a rapid egress of protein-rich fluid from pulmonary capillaries into alveoli and is believed to result from a loss of endothelial integrity in response to massive sympathetic discharge. Less likely is the so-called blast theory put forward by Theodore and Robin (1976) whereby a marked increase in systemic vascular resistance leads to excessive shunting of blood flow into the pulmonary circulation and subsequent alteration in capillary permeability. Management of NPE necessitates intubation and mechanical ventilation with positive end-expiratory pressure (PEEP). Pulmonary artery pressure should be kept as low as possible while maintaining adequate cardiac output. This can be a particular challenge in a patient with NPE who is in symptomatic vasospasm since triple H therapy poses risk of exacerbation of pulmonary compromise.

Volume overload, with or without cardiac complications, can lead to pulmonary edema. One study documented a 17% incidence of pulmonary edema among patients treated with triple H therapy. Often, this is an iatrogenic complication that can be avoided by placement of a Swan-Ganz catheter and meticulous management of pulmonary artery pressure. Pulmonary embolism (PE) is another potential adverse event that can affect a patient who is bedridden for a prolonged period. Intermittent pneumatic compression stockings and early mobilization help reduce the risk of PE. In high-risk patients, subcutaneous heparin may be added after exclusion of the aneurysm from the circulation (clipping or coiling). Prophylaxis against PE by placement of an inferior vena cava filter is recommended in patients who have documented deep vein thrombosis.

Infections. Close surveillance for possible sites of infection should include regular inspection of vascular catheter insertion sites, daily chest radiographs in intubated patients, frequent temperature checks, and monitoring of the urine and white blood cell (WBC) counts. Postoperatively, patients should have their wounds scrutinized. In any patient who manifests significant temperature elevation, cultures should be obtained and consideration given to changing indwelling lines or catheters.

Particular care should be paid to patients who have ventriculostomy catheters. Prophylactic intravenous antibiotics are generally used, and CSF is regularly checked with Gram stain, culture, cell counts, glucose, and protein. If infection is present, every attempt should be made to identify the organism and its sensitivity to antibiotics. Noninfectious sources of fever are medications, particularly phenytoin and H2 antagonists, deep venous thrombosis, and central («neurogenic») fever.

Gastrointestinal. Gastrointestinal (GI) bleeding is an uncommon but potentially devastating complication after SAH. Patients who have sustained severe cerebral injuries are at increased risk of developing gastroduodenal erosions («Gushing ulcers») from hypersecretion of gastric acid. Therefore, all patients with SAH are treated with antacids and H2 blockers. Aspirin, nonsteroidal anti-inflammatory drugs (NSAIDS) and steroids are generally avoided because they increase the risk of mucosal injury.

For this reason, Hct level should be determined daily. Stool specimens should be checked for occult blood if GI bleeding is suspected. Treatment of upper GI bleeding consists of nasogas-tric saline lavage, fluid replacement and, if necessary, transfusion. More serious cases may require endoscopic cauterization of bleeding sites or laparotomy.

Electrolytes/Endocrine Levels. Ten percent to 34% of patients have hyponatremia after aneurysmal SAH (Mayberg M.R. et al., 1994). Hyponatremia appears to be more common among patients of poor neurologic grade and it may be an independent risk factor for poor outcome. Although early studies focused on the syn drome of inappropriate antidiuretic hormone secretion (SIADH) as the primary reason for hyponatremia in these patients, more recent work implicates excessive natriuresis, or «cerebral salt-wasting syndrome.» Most patients are actually volume contracted as a result.

Determination of electrolytes/endocrine levels is critical since SIADH and cerebral salt-wasting syndrome are managed very differently. Fluid restriction is the treatment of choice for patients with SIADH. Studies have shown that fluid restriction is potentially harmful when applied to SAH patients with

hyponatremia. Current recommendations are to avoid fluid restriction in this group of patients and to provide maintenance fluid requirements with hypertonic crystalloid solutions—D5 1.5% NaCl or D5 3% NaCl— depending oh the patient's volume status and degree of hyponatremia.

Hypernatremia may also occur. Among SAH patients, it occurs most often in association with anterior cerebral artery aneurysm surgery. Compromise of anterior hypothalamic function may lead to diabetes insip-idus, which is characterized by polyuria, polydipsia, and hypernatremia. Treatment involves fluid replacement and antidiuretic hormone (vasopressin). Persistent diabetes insipidus is generally manifest in patients who have sustained severe neurologic injury and is a poor prognostic sign.

SPONTANEOUS INTRACEREBRAL HEMATOMAS

Spontaneous intracerebral hematoma (SICH) is a hemorrhage within the substance of the brain whose origin is unrelated to immediately preceding trauma. **Primary** forms are not directly caused by another disease process and include hypertensive hemorrhages. If the bleeding is due to an acquired or congenital process, such as tumor, aneurysm, arteriovenous malformation (AVM), or blood dyscrasia, it is considered to be **secondary.**

EPIDEMIOLOGY

The determination of epidemiologic features of SICH is difficult due to relatively small numbers of cases, misdiagnoses, and inaccuracies in death certification. Careful studies in defined populations have yielded significant insight, however (Broderick J.P., Brott T, Tomsick T., Miller R., Huster G., 1993). The incidence of stroke, including SICH, has experienced a downward trend over the past few decades, but it remains an important cause of morbidity and mortality. Stroke is the third most frequent cause of death in the United States, being responsible for 2% to 4% of all deaths. Eight to 13% of strokes and 14% to 20% of stroke deaths are due to SICH.

The incidence increases with advancing age and is higher in men than women. SICH is 1.5 to 2 times as common as subarachnoid hemorrhage, with an equivalent mortality rate (Broderick J.P., Brott T., Tomsick T., Miller R., HusterG., 1993). Preexisting hypertension is found in 50% of SICH cases and is the most common predisposing condition. Its importance as a contributing cause has decreased in association with improved treatment. Other risk factors include prior cerebral infarction, coronary artery disease, diabetes mellitus, anticoagulant treatment, aspirin use, low serum cholesterol, alcohol or drug use, and cold weather exposure. A patient's increasing age does raise the overall risk of intracerebral hemorrhage, because amyloid angiopathy produces a higher percentage of SICHs in the elderly (Juvela S., Hillbom M., Palomaki H., 1995; Wolf P.A., 1994).

SICH also occurs more frequently in Oriental countries. In Japan a hemorrhage rate of 120 in 100,000 population has been reported. SICH produces 17% of all strokes, and 22% of those in Japanese men. Hypertensive intracerebral hemorrhage accounts for 35% of all strokes in Taiwan (Chen ST., Chiang C.Y., Hsu C.Y., Lee T.H., Tang L.M., 1995), 31% in Hong Kong, and 32% in Korea.

The occurrence of SICH has been declining. In the Rochester study, the average age-adjusted incidence fell from 15.7 in 100,000 in 1945 to 1952, to 7.3 in 100,000 in 1969 to 1976. The downward trend began between 1953 and 1960, which coincides with the introduction of effective antihypertensive medications, and as a result hypertension has become less frequently associated with SICH, as noted above. The trend reversed and increased to 14 in 100,000 for the decade 1975 to 1984. The most recent data for the 1985 to 1989 period reveal that the incidence is holding steady at 13.5 in 100,000 (Brown R.D. et al., 1996). There has been a coincident drop in 30-day mortality. This recent increased incidence and decreased mortality are both felt to be due to the availability of computed tomography (CT), producing an increase in detection of small SICHs after its introduction in the 1970s. An estimated 24% of these hemorrhages were previously mis-identified as cerebral infarcts in the pre-CT era.

ETIOLOGIES

A general theory for the cause of SICH has been advanced, in which two principal etiologies often coexist: acute increases in cerebral blood flow (CBF) in areas of normal or ischemic arterioles and capillaries and damage to penetrating blood vessels by chronic arterial hypertension. In addition, larger arteries may become weakened, or small acute vascular injuries may hemorrhage in the presence of

bleeding diathesis. The various etiologies of SICH are listed in **Table.** Some categories overlap. Impaired hemostasis from disseminated intravascular coagulation (DIG) contributes to delayed traumatic intracerebral hemorrhage (DTTCH), and bleeding following cardiac surgery is related to anticoagulant use and emboli. Aneurysms can be caused by infection or trauma, and many in the «unknown» group may hemorrhage due to an acute rise in blood pressure. The etiologies listed below amyloid angiopathy are uncommon.

ETIOLOGY

Hypertensive
Chronic (acute) Non hypertensive Congenital vascular anomalies
Aneurysms
Arteriovenous malformations Coagulopathy Tumors Vascuiopathy, vasculitis
Cerebral amyloid angiopathy
Moyamoya disease
Vasculitis Drug related
Sympathomimetics
Anticoagulants
Fibrinolytics Postoperative
Intracranial
Carotid
Cardiac Poststroke
Arterial infarction
Venous occlusion Delayed posttraumatic
Parenchymal
Aneurysmal
Postoperative Infectious aneurysm Pregnancy
Neonatal intraventricular Unknown Secondary brainstem

Chronic arterial hypertension remains the most frequent antecedent of SICH, and the incidence is related to the degree and duration of blood pressure elevation. As mentioned previously, its importance in the etiology of SICH has been decreasing, but it continues to play a role in 50% of cases. The future trends of selected etiologies are also indicated in Table 1. The incidence of aneurysms and AVMs is fixed, although their detection may increase slightly in the future. Amyloid angiopathy will increase as our population ages. Anticoagulants may be used less frequently with better monitoring, and illicit drug use may become more common. Fibrinolytic therapy is increasing as more uses are found, and better detection and treatment of DIC may prevent DTICH.

PATHOPHYSIOLOGY

Chronic arterial hypertension is the most common cause of SICH and is associated with a vasculopathy affecting the 100- to 400-jum cerebral perforating arteries. They arise at right angles to their parent vessels, and as end arteries without collaterals for run off they are not protected from blood pressure increases. As a result of hypertension, Charcot-Bouchard (miliary) aneurysms develop on these perforating vessels and are found in association with SICH. Fisher (1972) divided microaneurysms into four groups: miliary saccular aneurysms, miliary aneurysms in lipohyalinosis, asymmetric fusiform miliary aneurysms, and bleeding globes. Also, Fisher has demonstrated lipohyalinosis in these small arteries. This subintimal accumulation of lipid and proteinaceous material is thought to contribute to rupture and SICH formation. Affected vessels include the lenti-culostriate arteries, the thalamoperforating arteries, the paramedian basilar artery branches, and branches of the superior cerebellar and posterior inferior cerebellar arteries. Regions irrigated by these vessels correspond to common sites of hypertensive hemorrhages. Hypertensive changes are more common in the proximal portion of the perforators, and thus putaminal hemorrhages are more frequent than caudate forms. In an ultrastructural study of hypertensive patients, Takebayashi (1985) found medial damage at bifurcations of distal lenticulo-striate arteries. He thought that these areas are sites of potential rupture producing cerebral hemorrhage. Microaneurysms were considered to represent areas of reabsorption and recanalization of small subclinical hemorrhages. The precise mechanism producing SICH is still uncertain. There are additional factors contributing to hemorrhage. Parenchymal or vascular damage such as with trauma or infection weakens support to vessels. In amyloid angiopathy, thickening of arterioles and venules occurs due to intramural amyloid deposition. Areas of fibrinoid necrosis and microaneurysms are seen. These brittle, amyloid-laden vessels may be excessively susceptible to trauma or blood pressure changes. Impairment of autoregulation, as seen with chronic hypertension or trauma, may allow excess vascular

stresses. Since SICH usually occurs during the morning or early afternoon when patients are likely to be active, changes in blood pressure with activity or on a diurnal basis may contribute. Hemostatic compromise plays a role in hematomas associated with DTICH, bleeding diatheses, and anticoagulant intake.

Although generally considered to be an acute, shortlived process, the bleeding in hypertensive SICH has occasionally been demonstrated by angiography (Hornig C.R. et al., 1993) and CT (Wijdicks E.F.M., Fulgham J.R., 1995) to continue for hours or even days from onset. Prolonged hemorrhage is more common with thalamic clots, is associated with persistent hypertension, and has a poor prognosis. Delayed deterioration from continued or recurrent bleeding at an average of 40 hours following onset was seen in 3% of hypertensive SICH patients in one series. In most cases the hemorrhage is completed within 6 hours, but if later expansion develops it is more likely for clots larger than 5 cm. The size increase during both the early formation and any later expansion of the hematoma is considered to be from secondary hemorrhages produced by tearing of adjacent vessels as the clot forms. Torn blood vessels and edematous parenchyma with petechiae are seen around hematomas studied at autopsy. The ultimate size of the clot is determined by the blood pressure, the size and rigidity of the ruptured vessel, the state of autoregulation, the adequacy of hemostatic mechanisms, the structural condition of the adjacent parenchyma, and the proximity to the ventricular system.

Experimental studies have demonstrated an ischemic penumbra surrounding the hematoma related to decreased CBF (Camarata P.J., Heros R.C., Latchaw R.D., 1994). A generalized increase in intracranial pressure (ICP) also lowers CBF. In addition, vasoactive substances such as kinins, histamine, and serotonin may be released from the clot into surrounding neural parenchyma with exacerbation of ischemia. Ischemia-induced depletion of intracellular energy stores then leads to disturbance of calcium homeostasis, release of excitatory aminoacids, formation of arachidonic acid and free radicals, and ultimately neuronal death. These effects are potentially pre ventable, and improved CBF following stereotactic removal of putaminal hemorrhages has been shown in humans.

Initially, intracerebral hemorrhages appear as focal collections of soft, dark red blood surrounded by a rim of edematous neural parenchyma containing petechial hemorrhages. Typical appearances and patterns of spread for common SICH locations have been described. A small hemorrhage may dissect along white matter tracts, splitting rather than disrupting them. Little functional deficit may result following clot resorption. Large hemorrhages destroy extensive amounts of cerebral tissue and can produce increased ICP and herniation. Ventricular extension occurs more commonly with caudate, thalamic, cerebellar, and pontine bleeds and often leads to obstructive hydrocephalus. Blood can also leak into the subarachnoid space. Death results from brainstem compression, brainstem distortion with secondary hemorrhages, or direct extension of the clot into the brainstem. Basal ganglia hematomas larger than 85 ml or more than 6% of the brain volume, and cerebellar clots larger than 3 cm in diameter have a poor prognosis without treatment.

If the patient survives, the hematoma is gradually broken down. In a few days, the clot changes to brown or tan in color and is surrounded by yellow or green hemoglobin breakdown products (Garcia J.H., Ho K.L., Caccamo D.V., 1994). The parenchymal margins are infiltrated initially by mononuclear cells (2 to 4 days), and later by fibroblasts (5 to 20 days), and then hemosiderin-laden macrophages (more than 20 days). Eventually all that remains is a slit-like cavity that is much smaller than the original hematoma. The adjacent brain is gliotic, hemosiderin-stained, and may contain axonal spheroids. There have been reports of chronic expanding intracerebral hematomas. These are lobar clots in normotensive patients that simulate a tumor by slowly enlarging and producing progressive neurologic deficits. The hematoma is surrounded by a thick fibrous capsule that enhances with contrasted CT scanning. Enlargement may be due to repeated bleeding from newly formed vessels on the inner capsular surface.

DIAGNOSTIC STUDIES

A patient presenting with SICH often requires an extensive laboratory evaluation. The large range of possible etiologies may necessitate screening for hematologic and clotting derangements, infectious processes, vasculitis, and illicit drug use. An elevation of body temperature and the peripheral white blood cell count occurs in some cases as a stress reaction or from irritation by intraventricular blood (Suzuki S. et al., 1995). The cause of hypertension should be determined if not already known. Evaluation of the cardiac, renal, and peripheral vascular systems can be helpful. Ophthalmoscopic examination may re veal the presence of subhyaloid hemorrhage from subarachnoid hemorrhage (SAH), hypertensive retinal changes, or papilledema from increased TCP. When studied, it will be bloody or xanthochromic in 70% to 90% of patients.

Definitive diagnosis of SICH is made radiographically, most commonly with CT, which is very sensitive for hematomas only a few millimeters in diameter due to the high density of acute blood. The white appearance of the clot is caused by high attenuation of the x-ray beam by the protein component of

hemoglobin. Since the introduction of CT over 20 years ago, an increase in SICH incidence has come about related to the correct diagnosis of patients previously thought to have had infarctions. Valuable information concerning hematoma size, location, associated mass effect, ventricular or subarachnoid extension, and secondary hydrocephalus can be obtained. This assists in medical and surgical management as well as in predicting outcome. **The administration of intravenous contrast** should be considered in patients (a) under age 40; (b) with no history of hypertension; (c) with a neurologic deficit increasing for more than 4 hours; (d) with a history of neoplasm, blood dyscrasia, vasculitis, or bacterial endocarditis; or (e) with subarachnoid blood or an atypical location or appearance of the hematoma. The clot becomes denser within a few hours, and the density reaches a peak by 1 to 2 days due to clot retraction and serum resorption. Low-density edema surrounds the clot and peaks by 4 to 5 days. With time, liquefaction and resorption occur, and the clot becomes isodense to the brain substance. The time course of this change is related to the size of the hematoma, taking 2 to 3 weeks for small hemorrhages, and up to 2 months for large clots. Eventually only a narrow cavity remains. Magnetic resonance imaging (MRI) has more recently been used in evaluating SICH patients. The multiplanar demonstration of the hematoma is advantageous. Complex time-dependent changes occur within and around the clot, modifying its appearance and allowing estimates of its age**.** These include gradual chemical changes in the hemoglobin molecule and erythrocyte lysis, which alter the MR characteristics of the clot. Oxyhemoglobin is present within the clot at 0 to 12 hours, deoxyhemoglobin at 1 to 7 days, methemoglobin at 5 days to several months, and hemosiderin at 1 week to several years. Gadolinium enhancement of the hematoma periphery is seen early (3 to 64 days) due to breakdown of the blood-brain barrier, and later (48 to 84 days) due to vascular granulation tissue. Contrast administration improves the visualization of causative lesions such as AVMs or neoplasms.

Within the first few hours after the bleed, hyperacute clots contain mostly oxyhemoglobin, which is isointense to brain on T_r and T_2-weighted images, so CT will be more sensitive than MRI. Acute hematomas imaged after approximately 24 hours will have a hypointense center on T_2-weighted images caused by deoxyhemoglobin formation in the most hypoxemic portion of the clot. A hyperintense ring of edema develops around the clot and becomes maximal at 4 to 5 days. In the early subacute stage, the clot will have high signal intensity. Those with ventricular bleeding often have little parenchymal involvement and no motor or sensory findings. Headache, vomiting, drowsiness, confusion, and meningismus are typical symptoms, mimicking a SAH. In other cases, the hemorrhage dissects laterally toward the internal capsule. Hemiparesis is present, although usually less severe than with putaminal bleeds. Sensory loss or conjugate gaze paresis may also develop.

Hemisensory deficits are generally found with thalamic hemorrhages. Hemiparesis, vertical gaze paresis, pupillary disturbances, dysphasia, hemineglect, or thalamic pain syndromes can be present in patients with large hematomas. Progression into the ventricular system is associated with a poor prognosis. CT scanning has enabled the recognition of small thalamic hematomas, and clinical syndromes associated with various locations have been described. Posterolateral bleeds are the most common. They often involve the internal capsule, resulting in significant motor and sensory loss. Aphasia, neglect, or lateral gaze paresis may also occur. An anterior or anterolateral site results in mild sensorimotor deficit, often with behavioral change, memory loss, and abulia. Medial hemorrhages frequently extend into the third ventricle. Minimal sensory and motor change is seen, but drowsiness, confusion, abulia, and amnesia develop. The least common site is posterodorsal. These produce mild, transient motor and sensory signs, but are associated with an unusual aphasia if the dominant side is affected (Kumral E. et al,, 1995).
Symptoms and signs are dependent on the Ideation and size of the clot and progress more gradually than with deep lesions. Severe headache at onset of bleeding is more common than with deep hematomas, and the location of the headache may correlate with the hemorrhage site. Frontal lobe clots produce bifrontal headache with contralateral arm weakness, mild lateral gaze paresis, and behavioral changes. Temporal hemorrhage results in a headache near the ear with a visual field defect, and dysphasia is also present if the dominant side is affected. An anterior temporal headache is seen in parietal bleeds, with con tralateral sensorimotor and visual field loss, as well as dysphasia or neglect. An occipital lobe hematoma causes ipsilateral eye pain and contralateral hemianopia. Seizures and headaches are more common, coma is less common, and prognosis is better than for deep hematomas.

Natural History. One-third of patients have a maximal deficit at onset; over half progressively deteriorate during subsequent minutes to hours. Sixty percent have altered levels of consciousness on admission, and two-thirds of these are comatose. If death occurs it is usually within a few days. Mortality and prognosis for recovery are strongly correlated with the hematoma volume and the patient's Glasgow Coma Scale (GCS) score on admission. Broderick et **al.** (1993) found that these two factors can accurately

predict outcome. **If the GCS score is 8 or less and clot volume 60 cm^3 or more, the 30-day mortality is 91%;** it is only 19% if the GCS score is 9 or more and the volume under 30 cm^3. Other important prognostic indicators include age, deep versus superficial location, intraventricular extension, and midline shift. Thalamic hematomas have the worst prognosis. Comatose patients with large clots generally die, while those with moderate-sized clots and focal signs survive with residual deficits. Many with mild deficits and small clots recover completely. Another hemorrhage develops in 3% to 6% of survivors, with onset from 13 to 28 months after the initial bleed. The new hematoma is generally in a different location, and the prognosis is worse, especially for those with bilateral lesions. Uncontrolled hypertension is a major risk factor for subsequent bleeding.

MEDICAL TREATMENT

Management in an intensive care unit (ICU) is necessary for critically ill patients to prevent cardiac and pulmonary complications. Those with significantly altered levels of consciousness are endotracheally intubated for airway protection and the maintenance of good oxygenation. Hypotonic intravenous fluids should not be given, as they may exacerbate cerebral edema.

Hypertension is frequently present following SICH, both from preexisting disease and as a response to elevated ICP. Persistent hypertension has been associated with increased vasogenic edema, early rebleeding, and increased morbidity and mortality. In patients with chronic hypertension, however, there is an upward shift of autoregulation. Also, the hematoma raises local tissue pressure, producing an area of ischemia in adjacent parenchyma, and increased ICP can alter CBF at distant sites. Thus hypertension following SICH may produce complications, while hypotension may increase ischemia. It is reasonable to keep the blood pressure in a middle range, perhaps with the mean arterial pressure just under 125 mm Hg (Dandapani B.K. et al., 1995). Patients with lobar hematomas are at risk for seizure development, and prophylactic anticonvulsant treatment is frequently begun. The need for long-term therapy is less clear. In one study, only 29% of patients with early (less than 2 weeks) seizures following SICH developed epilepsy, as opposed to 93% of those with later onset of seizures. No benefit was found with corticosteroid administration, and complications were increased. Clotting abnormalities should be identified and corrected, and anticoagulant medications should be stopped and their effects reversed.

Intracranial Pressure Monitoring and Treatment. A correlation exists between ICP and the neurologic status of the patient. ICP monitoring aids in medical management and is used to guide surgical decisions. Head elevation and hyperventilation to lower pCO_2 can decrease elevated pressures. Mannitol is also helpful in reducing ICP and may be combined with furosemide. A randomized trial found no improvement in outcome with the use of intravenous glycerol, however. Ventricular drainage is another adjunct for ICP management and is additionally used to treat hydrocephalus caused by ventricular extension.

Stereotactic Aspiration. When medical treatment alone is ineffective, hematoma removal may be needed to preserve the patient's life and increase the chances for neurologic recovery. Ideally, the method for accomplishing this would be rapid, simple, low risk, inexpensive, and have a high success rate. Stereotactic aspiration may combine many of these characteristics. Early attempts to aspirate recent hematomas encountered difficulty in removing the more solid portions, limiting their success. The center of the hematoma is firmer than the periphery, and the clot is more difficult to aspirate between 24 hours and 14 days after formation. A variety of devices have been devised to improve aspiration, including an Archimedes screw, a high-pressure fluid irrigator, an ultrasonic aspirator, and a modified Nucleotome (Surgical Dynamics, San Leandro, CA). Endoscopic evacuation was employed in a randomized series of 100 patients with improvement in outcome and lower mortality for subcortical hemorrhages. The development of thrombolytic drugs has led to their use in liquefying clots to improve the volume aspirated. Several Japanese groups have a large experience with the use of urokinase and have found it to be helpful. A catheter is inserted into the hematoma, and aspiration is performed. Urokinase is then instilled, and several hours later aspiration is repeated. Urokinase injection and aspiration are continued until sufficient clot is evacuated. Most of the hematoma can be removed by this method, and the rebleeding rate is 4% to 7%. Since evacuation takes hours to days with this method, it is not advisable in patients with signs of herniation. A similar technique employing tissue plasminogen activator has also been used with encouraging results.

SURGICAL EVACUATION

Open surgical evacuation is not indicated in the presence of irreversible neurologic injury evidenced by markedly depressed consciousness, rapid clinical deterioration, loss of brainstem function, or massive hemorrhage (Crowell R.M., Ojemann R.G., Ogilvy C.S., 1995). It is probably not necessary for alert patients with clots less than 2 cm in diameter The critical size for gangliobasal hematomas may be 85 ml. It has been suggested that the need for surgery can be based on the percentage of intracranial space occupied by the clots: surgerv is not needed if the clot occupies less than 4% of the intracranial space; should be based on the patients clinical status if the clot occupies 4% to 8%; should be performed if the clod occupies 8% to 12%; and will not help if the clod is more than 12%. However, one must consider that small hematomas in certain areas such as the temporal lobe may be more dangerous than larger clots elsewhere, and that cerebral atrophy can improve the tolerance for these masses. Evacuation should be considered for the patient who initially retains reasonably preserved neurologic function, and later deteriorates or has uncontrollable ICP despite medical treatment. Surgical treatment may also be contemplated if deficits do not improve after a period of time (Crowell R.M., Ojemann R.G.,OgilvyC.S., 1995).

There is conflicting information concerning the ability of surgical clot removal to improve outcome. Several randomized trials did not find any benefit over conservative treatment. Others have demonstrated improved results when surgery is performed within 6 to 7 hours of onset. The better outcome with early surgery may be due to limitation of edema formation or prevention of irreversible deterioration. A large cooperative study in Japan involved 339 centers and 7,010 patients with pu-taminal hemorrhage. They concluded that alert or drowsy patients with small hematomas are best treated conservatively, drowsy or stuporous patients with larger clots are candidates for aspiration and urokinase, patients in «semicoma» with or without herniation signs should have open surgery, and deeply comatose patients do poorly regardless of treatment.

Surgical approaches are made through the site of pial extension or adjacent noneloquent cortical areas. Transtemporal or transsylvian routes can be used for deep hematomas (10). Intraoperative ultrasonography aids in localizing the clot. Standard neurosurgical principles regarding gentle tissue handling, use of magnification and good illumination, and meticulous hemostasis should be followed. It is commonly thought that small remnants of adherent clot need not be removed. Any tissue that appears unusual should be biop-sied, and if a tumor is discovered an attempt at resection should be made. If an aneurysm is responsible for the hematoma, it should be clipped. On the other hand, AVM removal can be delayed until cerebral edema has resolved (Crowell R.M., Ojemann R.G., Ogilvy C.S., 1995).

Intraventricular Hemorrhage. Intraventricular hemorrhage (IVH) has been classified into primary and secondary forms. **Primary IVH** is completely contained within the ventricles or arises within 15 mm of the ventricular wall. Secondary **IVH** has a parenchyma! component originating more than 15 mm from the wall. Primary IVH is uncommon, accounting for 3% to 22% of intracranial bleeds. Both primary and secondary forms may be caused by hypertension, aneurysm, AVM, tumor, or coagulopathy. Eighty percent of IVHs are related to an intracerebral hematoma. Ventricular dilation is a frequent accompaniment. Ventricular extension is seen in one-third of cases of SICH, and the mortality rate is higher if it occurs. Prognosis is particularly poor if diffuse IVH with hemorrhagic dilation of the fourth ventricle is present. Headache, vomiting, nuchal rigidity, confusion, and altered consciousness are typical symptoms. Focal deficits such as hemiparesis will be found in cases of secondary bleeding. Ventricular drainage may be needed to control symptomatic hydrocephalus, but catheter occlusion can be a problem. Sometimes bilateral ventriculostomies may be needed. Clots will usually disappear in about 2 to 3 weeks. Intraventricular thrombolytic therapy has been successfully used to clear the hemorrhage in 3 to 4 days (Findlay J.M., Grace M.G.A., Weir B.K.A., 1993).

Intratentorial Hemorrahage| Cerebellar Hemorrahage. Cerebellar hematomas make up 10% of all SICHs. The highest incidence is in the sixth to eighth decades, with a male preponderance. Two-thirds are due to hypertension, and these originate in the Cerebellar hemisphere in the area of the dentate nucleus from distal superior Cerebellar or posterior inferior cerebellar artery branches. Extension to the fourth ventricle may occur, and 75% develop secondary hydrocephalus. Vermian clots are uncommon and may be massive. Hemorrhages in younger patients are often related to a vascular malformation. The cerebellum is also a frequent site of SICH in relation to anticoagulant use. Death is caused by brainstem compression and tonsillar herniation. Of those treated medically, 60% to 80% die, including almost all in stupor or coma.

A classic presentation is of a patient with sudden occipital headache, vomiting, dizziness, and an inability to stand or walk. However, many patients with cerebellar hemorrhage do not have typical symptoms or signs, and the diagnosis is more difficult in those with an altered level of consciousness. Symptom onset can be acute or subacute. Headache, nausea, vomiting, depressed level of consciousness,

dizziness or vertigo, small and reactive pupils, conjugate gaze palsy, nystagmus, peripheral facial palsy, dysarthria, ataxia, and pyramidal tract signs can be found. A triad of appendicular ataxia, ipsilateral gaze palsy, and peripheral facial weakness has been described. Two of three of these signs are found in 73% of patients. The diagnosis can be made rapidly with CT, and the degree of quadrigeminal cistern effacement correlates with outcome. Angiography is indicated if a vascular malformation is suspected.

The surgical indications are better defined in this group than at any other location. Rapid and irreversible deterioration can occur with little warning. Those who progress to coma generally do so within a few hours, but delayed worsening from several days to 1 month after onset can occur. The level of consciousness before surgery is the most important factor determining outcome. Mortality is 17% if the patient is alert or drowsy. If the patient is comatose, the mortality rate is 75%; a favorable outcome is more likely the more rapidly the clot is evacuated after the onset of symptoms with emergency surgery. Many recommend posterior fossa craniectomy and clot evacuation for all lesions 3 cm in diameter or larger (Camarata P.J., Heros R.C., Latchaw R.D., 1994). Removal of clots less than 3 cm in size should be considered if the level of consciousness is altered, if other signs of brainstem compression are present, or if there is deterioration. Conservative management is reasonable for the patient with a small hematoma who is alert, clinically stable, and without signs of brainstem compression or hydrocephalus (Camarata P.J., Heros R.C., Latchaw R.D., 1994). Patients in similar condition with clots larger than 3 cm who are initially seen 1 week or more after symptom onset may also be followed with close observation. Kobayashi et al. (1994) recommend conservative treatment for all patients with GCS scores of 14 or 15 with clots smaller than 4 cm in diameter. Hydrocephalus generally resolves following evacuation of the hematoma. The use of ventricular drainage prior to hematoma removal can lead to upward herniation. Stereotactic aspiration with urokinase has also been successfully used in treating this condition.

Brainstem Hemorrhage. Brainstem hemorrhages occur primarily in the pons, and make up 3% to 13% of SICHs. Hypertension is the cause of 90%, and there is no sex predominance. Bleeding is due to hypertensive effects on paramedian perforating branches of the basilar artery and usually begins near the midline at the junction of the tegmentum and the basis pontis. The peak incidence is in the fourth and fifth decades. A cryptic vascu malformation is likely in a young, nonhypertensive patient. Upward extension to the midbrain, hypothalamus, or thalamus may occur, and fourth ventricular rupture is present in 70%. Downward extension is rare. Hemorrhage restricted to the tegmentum is occasionally seen with a better prognosis.

A sudden onset of symptoms with rapid development of coma is seen in over one-half of cases. When the progression is slower, findings include a posterior headache, depressed consciousness, alterations in pulse, blood pressure, and respiration, posturing, paralysis including flaccid quadriplegia, ocular bobbing, pinpoint pupils, vertigo, vomiting, dysarthria, and hyperthermia. Lateral tegmental bleeds produce a crossed hemisensory loss, ataxia, oculomotor palsy with the one-and-a-half syndrome, and ipsilateral cranial nerve deficits. The diagnosis is made correctly in only one-fourth of cases. Treatment is determined by the patient's condition. Most have catastrophic neurologic impairment, and over 80% die within 48 hours. If the patient survives the ictus and is thought to be salvageable, supportive ICU care with respiratory and blood pressure management is instituted. Many patients will need ventricular drainage. Some smaller clots will resorb and the patient will have neurologic improvement. Several cases have been successfully treated surgically with stereotactic aspiration or open evacuation. For the latter, a surgical approach through the fourth ventricle or under the temporal lobe is used based on the CT or MRI appearance.