PRIMARY INTRACEREBRAL AND SUBARACHNOID HEMORRHAGE

AN APPROACH TO DIAGNOSIS AND THERAPY

MARC FISHER *

SUMMARY — The diagnosis of primary intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH) has become easier with the advent of modern imaging techniques. The incidence of ICH has declined, while SAH has remained relatively constant. The prognosis for both disorders remains dismal and the mortality rate is substantially higher than that observed with ischemic stroke. Early imaging with CT or MRI is important for rapid and accurate diagnosis. General medical management in a skilled nursing facility should be available for patients who are not moribund. Therapy for ICH is predominantly supportive and effective medical and surgical intervention remains elusive. For SAH, calcium channel blockers may reduce cerebral ischemic complications related to vasospasm, but effective medical therapy to prevent rebleeding has not been established. Early surgery after SAH should be considered in clinically stable patients. Many challenges remain regarding the prevention and treatment of both these cerebral hemorrhage subtypes.

Hemorragia intracerebral primária e subaracnóidea: uma avaliação do diagnóstico e da terapêutica.

RESUMO — O diagnóstico da hemorragia intracerebral primária (HIP), bem como o da hemorragia subaracnóidea (HSA), ficou mais fácil com o advento das modernas técnicas de imagem. A incidência da HIC tem declinado, ao passo que a da HSA tem permanecido relativamente constante. O prognóstico de ambas ainda é desanimador e a taxa de mortalidade substancialmente maior que a observada nas afecções isquêmicas. A indicação precoce da TC ou da RNM do crânio é importante para um diagnóstico rápido e preciso. Pacientes que não estejam moribundos devem receber cuidados médicos gerais em instalações com equipes de enfermagem especializada. O tratamento da HIC é predominantemente de apoio e intervenções médicas e cirúrgicas continuam indefinidas. Quanto a HSA, bloqueadores dos canais de cálcio podem reduzir as complicações cerebrais isquêmicas relacionadas ao vasoespasmo, mas não se estabeleceram tratamentos médicos eficazes para prevenir novos sangramentos. Naqueles pacientes clinicamente estáveis, deve-se considerar a antecipação da cirurgia. Muitos desafios permanecem no tocante à prevenção e ao tratamento desses dois subtipos de hemorragia cerebral.

PRIMARY INTRACEREBRAL HEMORRHAGE

Primary Intracerebral Hemorrhage (ICH) is a condition of bleeding into the brain parenchyma without an underlying source such as a vascular malformation, aneurysm, brain tumor or bleeding disorder. The incidence of ICH has declined over the past several decades, but this category of stroke still accounts for approximately five to ten percent of all stroke cases and 15 to 25 percent of stroke related deaths 44. **The percentage of ICH cases which are fatal has declined, but this may relate to more accurate diagnosis in the CT scanning era**[38]. **ICH typically occurs in patients somewhat younger than the age associated with ischemic stroke.**

Hypertension is commonly associated with ICH, although in recent case series less than 50 percent of the cases have had prior hypertension io. Cigarette smoking and alcohol consumption are also risk factors which are strongly associated with the

* MD, Department of Neurology, The Medical Center of Central Massachusetts and The University of Massachusetts Medical School.

Marc Fisher, MD — The Medical Center of Central Massachusetts - 119 Belmont Street - Worcester, MA, 01605 - USA.

development of ICH. Caplan has recently suggested that three factors are important for the development of ICH u. The first factor is an acute rise in blood pressure such as may be induced by cold, sympathomimetic drug use and stimulation of the trigeminal nerve. The second factor is an acute increase in cerebral blood flow, either diffusely or focally. Lastly, ICH can be seen in association with reperfusion of ischemic or injured cerebral tissue such as after relieving of a local arterial blockade.

Pathogenesis — Spontaneous ICH has been associated with the rupture of cerebral aneurysms associated with small intracerebral blood vessels. This concepts dates to the work of Charcot and Bouchard over a century ago[13]. These so called Charcot and Bouchard aneurysms are 100 to 500 microns in diameter, which develop along deep small penetrating arteries such as the lenticulostriate arteries. These arteries typically develop at arterial branch points where pulsatile stress on the artery is maximal. These arteries are typically devoid of connective tissue layers and therefore represent regions of weakening within the arterial system. The formation of these aneurysms is associated with lipohyalinosis and fibrinoid necrosis, processes which favor the weakening of arterial walls **!8.** Microaneurysms are much more common in patients with a history of hypertension, although they may be seen in normotensive patients as well. As previously noted, in the suggestions made by Caplan, not all ICH patients have these chronic changes. He suggests that more acute processes such as an acute rise of blood pressure and blood flow may abruptly lead to arterial rupture and intracerebral hemorrhage. Dissection of a section of small intracerebral arteries is another proposed mechanism for the development of ICH[18]. Finally, it has been suggested that small angiomas can rupture and become obliterated, again leading to the development of ICH **6.**

Clinical Manifestation — Primary ICH has a stereotyped pattern of presentation, although exceptions may arise **31.** Typically ICH begins during the awake state and uncommonly during a time of exertion. The patient usually complains of a headache which in many cases is lateralized towards the side of ICH. The patients may develop focal neurological deficits in association with the area of ICH within the brain parenchyma. The neurological deficits may reach their maximum level within minutes, but in many cases there is a smooth and gradual progression over hours. Nuchal rigidity is common. Patients with ICH may rapidly become obtunded or comatose in contradistinction to patients with ischemic stroke, who rarely have clouding of consciousness early in the course. Seizures are seen in a distinct minority of cases, but they are more common than in patients with ischemic stroke **7.** The course of primary ICH is typically monophasic, but cases have been reported in which delayed deterioration has occurred[14]. Clinical-radiologic studies have demonstrated delayed enlargement of the initial hematoma, hours or even days after the initial event. These delayed enlargements are typically associated with clinical worsening. The risk of delayed worsening may be higher in patients with putaminal hemorrhages and in patients in whom hypertension is poorly controlled.

Diagnosis — Prior to the advent of CT scanning, the diagnosis of ICH was made by correlating the neurological history and examination with the examination of cerebral spinal fluid (CSF). Lumbar puncture, however, carries a significant risk for neurological deterioration in patients with ICH and therefore is no longer recommended. CT scanning allows for rapid and accurated diagnosis of ICH as well as providing information about it's site and size. CT scanning is highly specific and sensitive for the diagnosis of ICH (Fig. 1). Contrast enhanced CT scanning should be obtained in addition to non-contrast studies to assess the presence of abnormal vasculature such as may be seen with an arterial venous malformation. Cerebral angiography was employed in the past to make the diagnosis of ICH, typically indirectly based upon the presence of an avascular mass with shift of intracranial arteries. Angiography is only rarely employed at present in cases with an atypical location of iCrt to assess tue presence of vascular malformations or aneurysms, especially in younger patients.

MKI scanning which has significant advantages for the early diagnosis of ischemic stroke may not be as useful in ICH **16.** Acutely, ICH will appear as an area of low density on T2 weighted images. Surrounding areas of edema demonstrate hyperintensity. Mass effect will be readily apparent as well. Subacutelly, 3 to 10 days after ICH. the area of increased signal on Tl weighted images becomes apparent and the

T2 weighted images remain hypointense (Fig. 2). In the chronic state the Tl images are not very helpful but the T2 weighted images remain hypointense. These changes in signal intensity relate to the presence of various hemoglobin degradation products which appear at different time point after the ICH.

Differential Diagnosis — The majority of ICH patients relate to primary rupture of a small intracranial artery, usually in a setting of chronic hypertension or an acute rise in blood pressure n**.3i.** However, other conditions can lead secondarily to the development of ICH. Hemorrhage into an area of infarction is not rare. Hemorrhagic infarction typically takes on a punctate character, but on occasion globular hemorrhages which mimic primary ICH may be present. This occurrence is more likely in patients with large embolic infarcts, who receive anticoagulants **12.** Hemorrhagic infarction is thought to occur because reperfused arteries have a weakened character, which may then lead to rupture of the vessel and extravasation of blood. Small deep ICH can present with signs and symptoms which mimic lacunar infarcts and the prognosis does not appear to differ between the two conditions.

Oral anticoagulation without the presence of ischemic stroke can be associated with development of ICH and ICH accounts for 0.5 to 1.5 percent of bleeding events associated with anticoagulation **23.** The risk of ICH in patients receiving oral anticoagulants is 8 to 10 times that in non-anticoagulated patients. ICH associated with anticoagulants is typically larger and more lethal than the ICH in patients not maintained on anticoagulants. The acute development of focal neurologic deficits in an anticoagulated patient necessitates a rapid evaluation for the presence of ICH.

The recent widespread application of clot specific and non-clot specific thrombolytic agents in patients with acute MI has lead to the development of ICH in association with these medications. There does not appear to be any substantial difference in risk for the development of ICH between these two different types of thrombolytic agents **17.** With tissue plasminogen activator *(t-pa)* a dose response relationship for the development of ICH has been shown in that higher doses of t-pa have a higher risk for ICH. One study has suggested that it is the use of concomitant heparin sodium therapy with t-pa which leads to the development of ICH **22.** Platelet anti-ag*-gregants effects have also demonstrated in association with t-pa and this may play a role in the development of ICH as well. Thrombolytic therapy for ischemic stroke is presently under evaluation and we can anticipate ICH complications in these patients. Bleeding into cranial neoplasms may also lead to the development of ICH **9.** Approximately 1 to 2 percent of ICH patients can be anticipated to have an underlying primary or metastatic tumor as the source for their event. High grade glioblastomas are a tumor subtype with an increased risk for ICH. The diagnosis of bleeding into a tumor can usually only be established by the performance of appropriate imaging studies. Blood dyscrasias such as leukemia and hemophilia area commonly complicated by ICH **3i**.One-third of patients with these disorders may die from direct result of ICH. ICH in these disorders is typically multifocal and may occur in atypical sites. Thrombocytopenia and clotting factor deficiencies lead to a tendency for hemorrhage in these disorders.

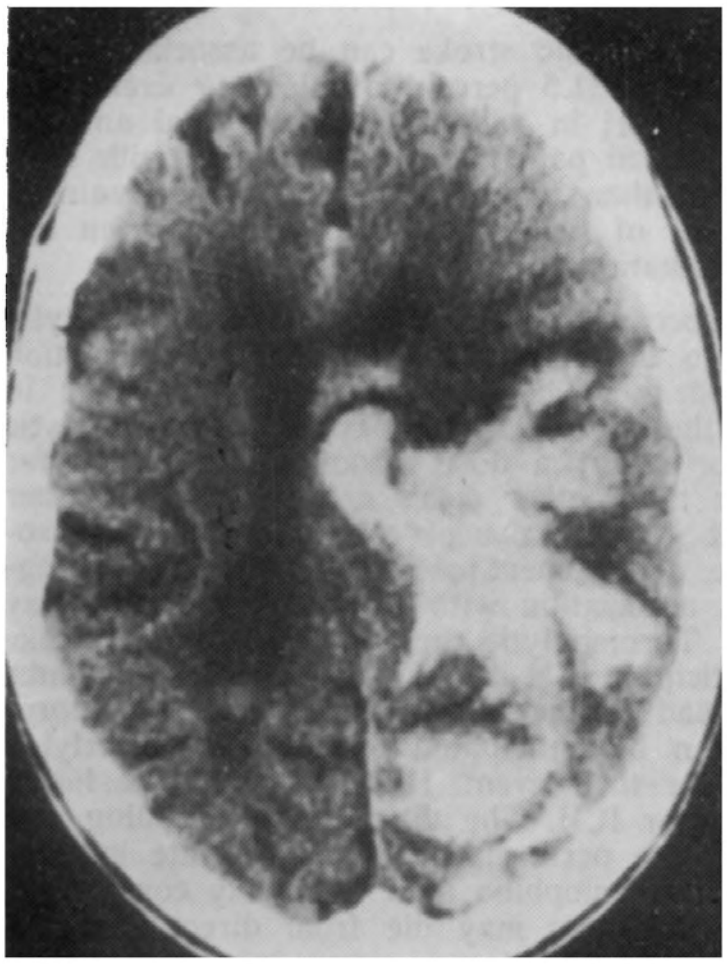
Disseminated Intravascular Coagulation (DIC) may also be associated with ICH as well as widespread intravascular thrombosis. DIC may occur after severe trauma, in patients with underlying neoplasms or in association with disseminated infection **27.** Platelet dysfunction and deficiencies such as those seen in patients with idiopathic thrombocytopenic purpura and in patients who have received chemotherapeutic drugs may also be associated with the development of ICH.

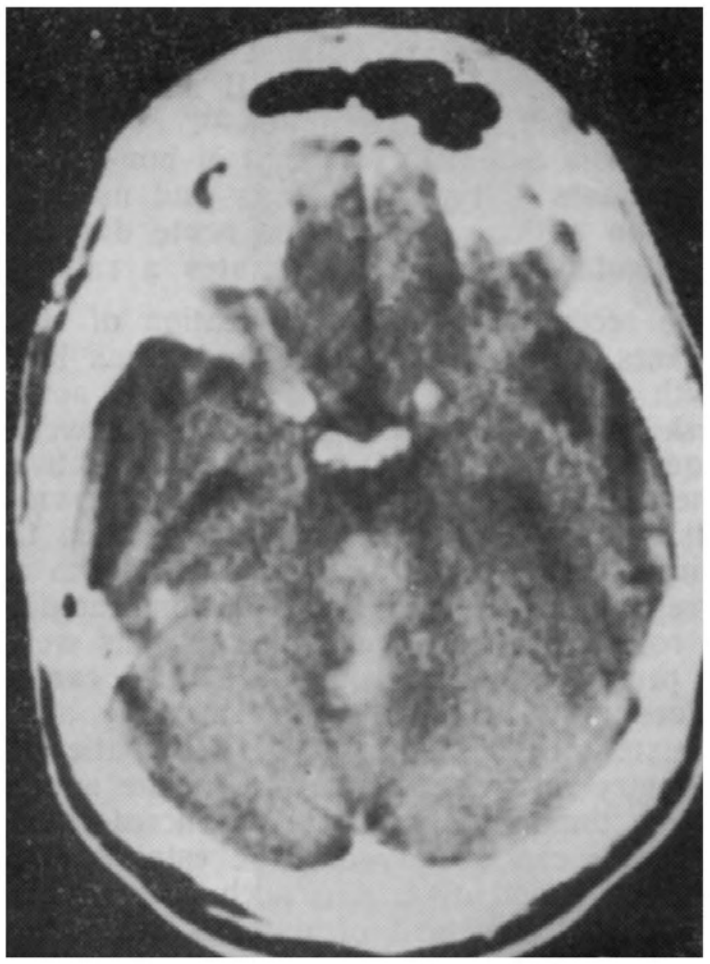
Drugs, both legal and illicit, may lead to the development of ICH. Sympathomimetic drugs such as methamphetamine pseudoephedrine and phenylpropanolamine have been associated with the development of ICH, usually lobar in location **6.** These drugs can cause acute elevation in blood pressure and also a vasculitis, both conditions predisposing to ICH. Cocaine is an illicit drug which has been associated with the development of ICH **28.** Cocaine can cause cerebral vasospasm and also hypertension, leading to the development of ICH.

Clinical Syndromes of ICH — ICH develops is stereotyped anatomical localizations within the brain, leading to well recognized clinical-anatomical syndromes **21,** The most common sites for ICH are the putamen *(30 to 50 percent of cases),* lobar *(15 to 20 percent)* thalamus *(10 percent),* pons *(10 percent)* and cerebellum *(10 per-*

cent). Putaminal intracerebral hemorrhage is the most common site and is thought to occur secondary to rupture of lateral branches of the lenticulostriate arteries. The typical clinical syndrome associated with putaminal ICH is the rapid development of contralateral hemiplegia and hemisensory loss, homonymous hemianopsia and paralysis of conjugate gaze contralateral to the side of hemorrhage **30.** Medium to large size putaminal hemorrhages are associated with a rapid decline in the level of consciousness and this may progress to coma at an early stage. Aphasic syndromes may be seen with hematomas in the dominant hemisphere and a neglect syndrome may be observed in patient with non-dominant hemisphere putaminal ICH. The prognosis in these patients is worse when intraventricular extension occurs or there is upward extension of the hematoma.

ICH into subcortical white matter, so called lobar hemorrhage, has been observed with increasing frequency with the advent of more advanced imaging techniques **24.** The





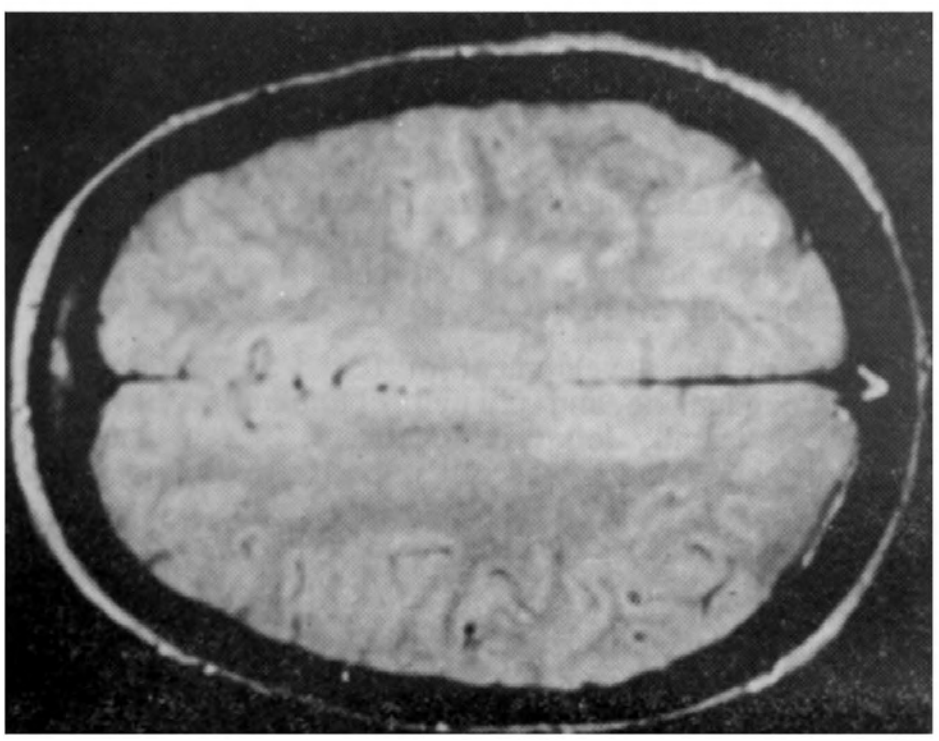
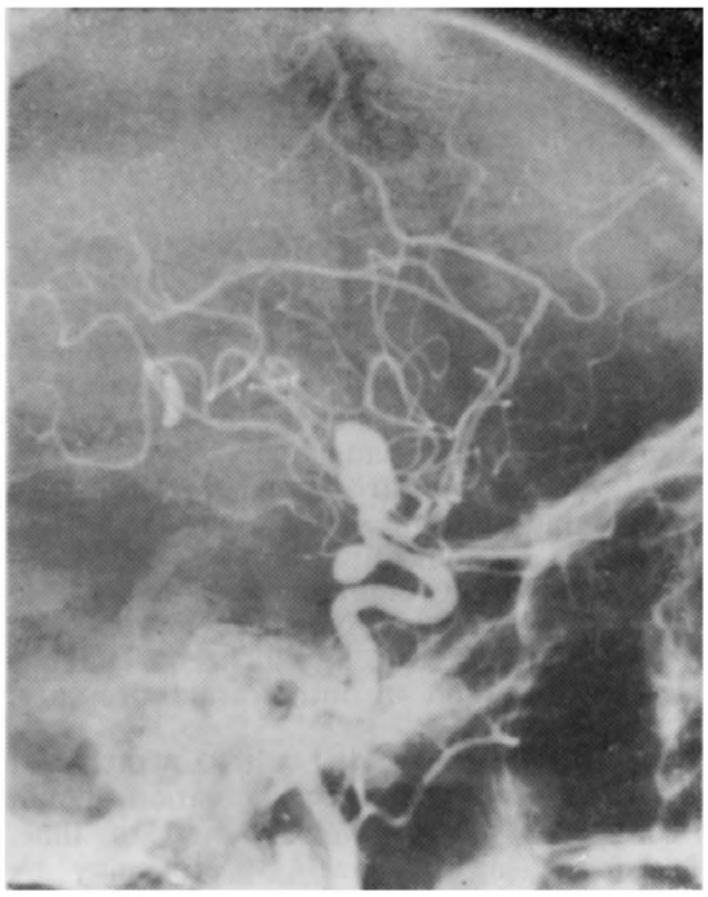


Fig. 1 (above, left) — A large lobar hemorrhage with ventricular extension is easily diagnosed on non-contrast CT scan.

Fig. 2 (below) — An MRI scan demonstrating a subacute intracerebral hemorrhage.

Fig. 3 (above, right) — A midline cerebellar hemorrhage is observed on a non-contrast CT scan.



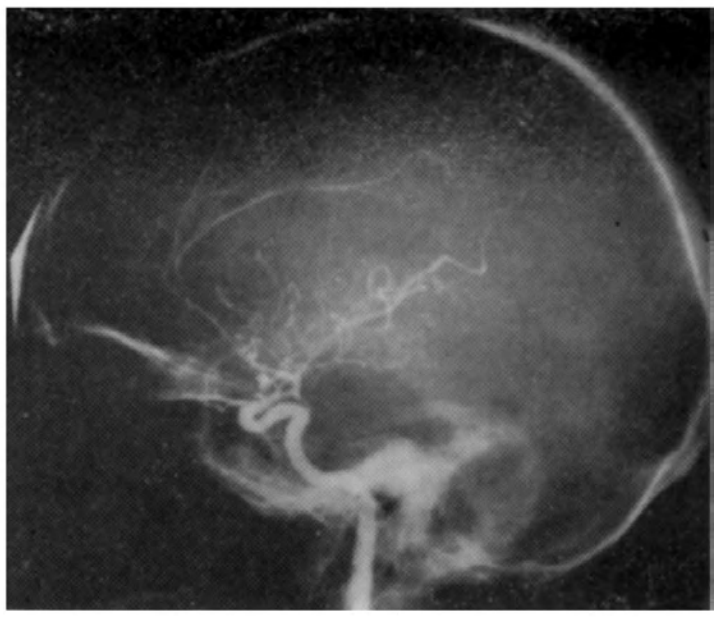


Fig. 4 (left) — This lateral cerebral angiogram demonstrates a large saccular aneurysm.

Fig. 5 (right) — Vasospasm of the middle cerebral artery and an associated aneurysm are demonstrated by this cerebral angiogram.

prognosis in these patients is more favorable than in patients with deeper hemorrhage and therefore it probably was underdiagnosed in strictly autopsy series. The symptoms and signs of lobar hemorrhage will depend on the location involved. Lobar hemorrhages may occur in the deep white matter, in the occipital lobe, the temporal lobe, the parietal or frontal lobes. Lobar ICH has a much lower association with hypertension than ICH in other locations. Lobar ICH is more commonly associated with amyloid angiopathy which weakens vessels walls in a widespread fashion and may cause multiple ICH foci and recurrent ICH is Five to ten percent of ICH cases result from amyloid angiopathy and this is the second most common predisposing factor to the development of ICH, trailing only chronic hypertension.

Thalamic ICH manifest a typical clinical syndrome in which sensory-motor or only sensory deficits predominate **41.** Development of motor symptoms and signs witn thalamic hemorrhage relates to extension of the hemorrhage or pressure effects on the internal capsule. Oculomotor findings with thalamic hemorrhage are characteristic and include downward deviation of the eyes, skew deviation, incomplete paralysis of vertical gaze and pupilary inequality. In addition, conjugate eye deviation either toward the involved side or contralateral^ may occur. Thalamic hemorrhage may rupture into the ventricular system and this development is associated with a poor prognosis. A delayed consequence is intractable pain known as the «Thalamic Pain Syndrome»**3i.** An unusual restless hand posturing may also develop chronically after thalamic hemorrhage. An aphasic syndrome with mutism initially and subsequent paraphasic and anomic speech may be seen in association with thalamic hemorrhage.

Pontine hemorrhage has the highest mortality of all ICH syndromes **19.** These patients typically develop rapid decline in consciousness with early coma. They also demonstrate quadriplegia, multiple cranial nerve abnormalities and small unreactive pupils. Patients with unilateral pontine ICH have neurological deficits which reflect the area ot the pons involved with the ICH and the prognosis is not as dismal as that seen with large paramedian pontine ICH.

Cerebellar ICH usually presents in a stereotyped fashion and must be diagnosed early to allow for rapid intervention and salvage. The patients will seek medical attention because of the rapid development of headache, vertigo, nausea and vomiting, drowsiness and gait difficulties **2°.** Neurological examination typically demonstrates paralysis of ipsilateral conjugate gaze with horizontal nystagmus. Peripheral facial palsy is common as is ipsilateral limb ataxia, gait ataxia and slurred speech. The course is complicated by a progressive decline in consciousness over several hours. The

early recognition of this syndrome is important because rapid surgical intervention may be life saving and the rapid performance of a CT scan *(Fig. 3)* will usually be diagnostic.

Therapy — General medical management of ICH patients is important and in some cases critical. Many of these patients require management in specialized stroke units or intensive care units **45.** Adequate oxygenation and maintenance of an airway may be critical, especially in patients with a depressed level of consciousness. Artificial ventilation may be required in some patients, as they deteriorate. Hypertension may be an underlying problem in some patients with acute exacerbation in relationship to the cerebral hemorrhage. Treatment of acute hypertension in ICH patients engenders controversy. Substantial elevations in blood pressure may abate without treatment, aside from bedrest. If the blood pressure remains above 200mmHg for more than 6 to 12 hours, then lowering of the blood pressure is probably indicated. The patient's blood pressure should be lowered to pre-morbid levels, if they are known or the upper end to the normotensive range. If hypotension is induced, there may be deleterious effects, because of impaired cerebral autoregulation, chronically and acutely. Beta blocking agents are the preferred medication for the lowering of blood pressure in ICH patients, but diuretics may also be employed. Calcium channel blockers, nipride and hydralazine should be avoided because of their vaso-dilatory effects which can enhance the formation of cerebral edema and also lead to elevation of intracranial pressure.

The treatment of cerebral edema associated with ICH can be difficult. Intracranial pressure monitoring may be required in some patients. Initially, hyperventilation should be tried to lower ICP but this may be effective for only a short period of time. Chronic diuresis with medications such as mannitol, sorbitol and glycerol may be effective in selected cases, when they are employed serially over several days. Care should be exercised because these osmotic diuretics can induce systemic dehydration, electrolyte imbalances and pulmonary edema. The use of corticosteroids for the treatment of cerebral edema associated with ICH is not routinely recommended. Dexamethasone has been shown to worsen outcome in ICH patients and therefore should probably be avoided **33.** if patients develop acute hydrocephalus in association with ICH and cerebral edema, emergency ventricular shunting may be beneficial and should be considered.

Surgical treatment to evacuate hematomas in ICH patients has been attempted for decades. Conclusive evidence that surgery is beneficial remains lacking and has not been adequately studied in a randomized and controlled fashion, therefore scientific validity is questionable. In a recent study of patients with putaminal hemorrhage, a prospective randomized trial demonstrated that there was no difference in outcome in patients randomized to surgery, as compared to best medical therapy **5.** At this time, recommendations for surgical intervention in ICH are tenuous, but the following are suggested by some authorities **31,45.** Patients with cerebellar hemorhage who are deteriorating, should probably have prompt evacuation of the hematoma, if they have not become comatose. Patients with small cerebellar hemorrhages, who are clinically stable and do not have impaired consciousness, should probably be managed conservatively. Patients with lobar ICH should be considered for surgery, if there is progression of the neurologic deficit and continued decline in the level of consciousness. Stable patients can probably be managed medically. Surgery does not appear to be helpful in patients with putaminal, thalamic or pontine ICH and is not recommended in these conditions.

SUBARACHNOID HEMORRHAGE

Most cases of subarachnoid hemorrhage (SAH) occur secondary to rupture of saccular aneurysms. Other causes include rupture of arterial venous malformations, complications of anticoagulant therapy, bleeding into meningeal metastases and as a complication of coagulopathies. This review will focus on SAH secondary to ruptured aneurysms. The incidence of SAH approximates 4 to 10 per 100,000 per year and up to 1 percent of the autopsy population is noted to have an incidental, unruptured aneurysm 35. The majority *(85 percent)* of aneurysms occur within the anterior circulation and the most common locations are at the junction of the internal carotid artery and posterior communicating artery, along the course of the anterior communi-

eating artery and at the bifurcation of the middle cerebral artery **37.** Approximately 15 percent of aneurysms occur in the posterior circulation, typically at the bifurcation of the basilar artery or at the origin of it's more proximal branches. Multiple aneurysms are found in 15 to 20 percent of cases. Systemic disorders such as polycystic kidney, Marfan's syndrome, Ehrlers-Danlos syndrome and coarctation of the aorta are other medical conditions associated with an increased incidence of intracerebral aneurysms.

Unruptured, incidental cerebral aneurysms are being discovered with increasing frequency, as non-invasive imaging technology as high resolution MRI scanning becomes more widely available The risk for subsequent rupture of these anuerysms appears to be size related in that aneurysms larger than 1 centimeter in diameter have a substantial risk for rupture, while smaller aneurysms do not appear to be as dangerous **43.** The best management approach for these patients is uncertain, but it has been suggested that aneurysms larger than 8 millimeters be considered for prophylactic surgical clipping, if the patient is a good medical risk.

Risk factors for the development of saccular aneurysms and subsequent SAH are not as well characterized as those for ischemic stroke. Hypertension apears to be a risk factor in some, but not all studies **29.** Cigarette smoking and oral contraceptives have also been suggested as possible risks.

The pathogenesis of saccular aneurysms remains controversial. It is uncertain whether they represent a congenital malformation or an acquired defect of the muscular media and internal elastic lamina of large to medium size intracranial arteries **36.** The congenital hypothesis is favored by the frequency of multiple aneurysms, the familial occurrence of aneurysms on occasion and the association of aneurysms with other systemic diseases. The degenerative hypothesis receives support because of the relatively late development of aneurysms in most patients, the association with hypertension and the pathological observations of interval changes, calcification and thrombosis, suggesting a dynamic process.

Clinical Manifestations — Saccular aneurysms remain asymptomatic in many patients until a catastrophic event occurs. Some patients report a warning headache before SAH occurs. These warning headaches may represent minor leaks and should be investigated, if clinical suspicion warrants it **40** Aneurysm patients may develop focal neurological symptoms, as the aneurysms enlarge and exert pressure effects on vital intracerebral structures. A typical presentation would be the development of a third nerve palsy with pupilary involvement in association with an enlarging posterior communicating artery aneurysm **4.** Occasionally, embolization of thrombus within aneurysms may cause transient ischemic attack or stroke, as the presenting manifestation for the aneurysm **34.** Almost all patients with SAH present with a severe headache, usually the worst headache ever experienced by the patient. This is commonly associated with nausea, vomiting, photophobia and neck stiffness. Alteration of consciousness occurs early in the course in many patients. Focal neurological signs will develop in association with dissection of the SAH into the brain parenchyma or as a consequence of blood within the subarachnoid space interfering with function of cranial nerves or more distal nerve roots. Neurological examination will reflect these abnormalities and also will commonly demonstrate a sixth nerve palsy, as an indirect pressure effect. Examination of the optic fundi may demonstrate subhyaloid retinal hemorrhage or papilledema. Clinical grading scales such as that of Hunt and Hess may be useful in evaluating and triaging patients **8.** The clinical grade relates primarily to the level of consciousness and patients who are graded I or II at presentation have a relatively good prognosis, whereas grade IV or V patients *(moribund)* have a very poor prognosis. Grade III patients who are obtunded have an intermediate prognosis.

Laboratory Studies — If a CT scan can be obtained, this should be the initial laboratory study to confirm the presence of bleeding within the subarachnoid space and perhaps a large aneurysm with the use of contrasts. If a CT scan is not available or is initially negative, then a lumbar puncture should be performed to look for the presence of blood in the CSF. If the CSF is examined at least 2 hours after the initial hemorrhage, it will be grossly bloody in most cases and when spun down should demonstrate xanthochromia in the supernatant. There may be a reduction in CSF glu-

cose and the opening pressure of the lumbar puncture will reflect elevations of intracranial pressure. The diagnosis of SAH initially obtained by these procedures should prompt the early performance of 4 vessel cerebral angiography to determine the presence and location of intracranial aneurysms *(Fig. 4).* Cerebral vasospasm may also be seen and may impact upon the timing of surgical intervention. The initial angiography may be normal in up to 10 to 20 percent of cases and many authorities would suggest a second study in 10 to 14 days to definitively exclude the presence of an aneurysm[4] **2.**

General Medical Therapy — Patients with SAH should be admitted into an intensive care unit or a unit with skilled nursing capability[8]. They should be kept at absolute bedrest and elevation of the head of the bed is suggested to approximately 30 degrees. Careful pulmonary therapy to avoid atelectasis and pneumonia is helpful. Immobilized patients, who are at risk for the development of deep vein thrombosis, should be given pneumatic compression boots or stockings. Stool softeners should be used to prevent constipation and nausea should be treated with an antiemetic. Headache or other pain should be managed with appropriate analgesia such as acetaminophen, codeine or meperidine. Sedation is appropriate in agitated patients and diazepam or alternatively phenobarbital may be used, especially if prophylactic seizure control is indicated. Blood pressure should be carefully lowered, if the systolic pressure remains above 160 to 180mmHg. The blood pressure should not be lowered substantially, as this might enhance the development of cerebral vasospasm and delayed ischemia. Hyponatremia should be screened for and may develop, as a consequence of the syndrome of inappropriate ADH. Salt wasting may occur because of increased circulating levels of atrial natriuretic peptide levels. Therefore, serum electrolytes, fluid intake and output and serum osmolarity should be carefully assessed and treated if abnormalities develop.

Neurologic Complications — The three major neurologic complications associated with SAH are rebleeding, cerebral vasospasm with ischemia and the development of hydrocephalus **8.** Rebleeding of a ruptured saccular aneurysm is a major cause of deterioration in SAH patients. Rebleeding most commonly occurs within the first 24 hours after the initial rupture and approximately two-thirds of the patients who rebieed will die because of it **25.** Treatment to prevent rebleeding remains lacking. Bedrest and controlled hypotension do not appear to be effective. Antifibrinolytic agents have been used to prevent clot lysis and agents such epsilon aminocaproic acid or tranexamic acid have been employed i. Studies with these agents suggest that they may reduce the rebleeding rate, but they appear to enhance the development of the delayed cerebral ischemia and therefore the overall mortality rate with this therapeutic approach does not change. Antifibrinolytic drugs should not be used routinely, but some authorities suggest using these drugs for several days after the initial SAH, if delayed surgery is contemplated.

Cerebral vasospasm occurs, commonly after SAH and the maximal time for developing this complication appears to be during the second week after the initial SAH **8.** Vasospasm may induce cerebral ischemia and the development of infarction. This most commonly occurs in the distribution of the anterior cerebral artery. Vasospasm may be identified by cerebral angiography *(Fig. 5)* or alternatively transcranial doppler techniques. The amount of blood demonstrated on initial CT scanning appears to correlate with the risk for the development of delayed vasospasm[2]. It has been hypothesized that breakdown products of red blood cells initiate the vasospasm. There is no proven therapy to reduce the development of vasospasm in SAH patients. Hypervolemic and hypertensive therapy has been employed to augment cerebral perfusion pressure. The expansion of intravascular volume with agents such as hetastarch or plasmanate has also been employed. These therapies can lead to the development of complications such as myocardial infarction, congestive heart failure, arrhythmias, rebleeding and hyponatremia. Calcium channel blockers, typified by nimodipine, have been used to ameliorate vasospasm **32.** They do not appear to prevent vasospasm, but do reduce the development of ischemic events after SAH. The overall outcome appears to be improved by nimodipine. It is recommended that 60mg be given every six hours for up to 21 days after SAH.

Hydrocephalus may develop after SAH because of obstruction at the outlets of the tourth ventricles **39.** Hydrocephalus typically occurs during the second week after SAH

and may be associated with clinical deterioration such as change in mental status, incontinence and gait difficulties. This complication is easily diagnosed by the performance of imaging studies such as CT or MRI. The patients can be managed with steroids, diuretics or mannitol. If the deterioration continues, the placement of a ventricular shunt may be necessary.

Surgical Therapy — Surgical management, usually the placement of a clip at the base of the aneurysm, is indicated in patients who are stable. The clip ligation reduces the risk of subsequent rerupture. Controversy remains concerning the most appropriate timing for surgery. Some authorities advocate early surgery within 1 to **2** days after presentation in patients who are in the better clinical grading group. Others recommend late surgery, 7 to 14 days after SAH. The presence of cerebral vasospasm would favor later surgery. The advantages of early surgery include reduction in risk of rebleeding, cisternal lavage to impede arterial constriction and the safer use of hypertensive and volume expansion therapy, if cerebral ischemia develops. The performance of surgery early may be more technically demanding, but improved operating techniques and anesthesia have solved some of these difficulties. Delayed surgery appears to have a lower complication rate, but many patient may deteriorate before reaching the operating room and be denied potential surgical benefits. A recent study comparing early versus late surgery in SAH patients failed to demonstrate a clear cut advantage for one time versus the other **26.** Following surgery, angiography should be performed within several days to evaluate the positioning of the clip ligation and to confirm that it was successful. Patients after aneurysm clipping are typically managed in an intensive care unit for several days. Postoperative cerebral vasospasm may require the continuing use of hypervolemic arid hypertensive therapy.

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