SPONTANEOUS BRAIN HEMORRHAGE accounts for about 10% of all strokes and is associated with a high morbidity and mortality rate. While the incidence of stroke due to occlusive cerebrovascular disease has declined the incidence of intracerebral hemorrhage has remained relatively stable. The syndromes that result from brain hemorrhage are sufficiently characterized to permit their clinical recognition in many patients. Computed tomography (CT) has revolutionized the management of brain hemorrhage by demonstrating these lesions with clarity and detail. The clinical and CT criteria for decisions regarding medical and surgical management continue to undergo change.

Pathophysiology

Hypertensive brain hemorrhage tends to occur in specific sites: putamen, thalamus, cerebellum and pons. Lobar hemorrhage may be related to hypertension but it can also occur in association with normal blood pressure and no radiographic evidence of a specified etiologic factor. Spontaneous nonhypertensive brain hemorrhage may be associated with aneurysm, arteriovenous malformation (AVM), primary or metastatic brain tumor, infarction, anticoagulation, diseases associated with clotting disorders such as leukemia, thrombotic thrombocytopenic purpura, sickle cell disease and cerebral arteritis such as occurs with collagen vascular disease, amyloid angiopathy and methamphetamine abuse.

In hypertensive hemorrhage, the most common sources of bleeding are the small penetrating arteries in the base of the brain (the lenticulostriate and thalamoperforating arteries) and the paramedian branches of the basilar artery. Though these vessels normally can withstand extremely high pressure without rupture, pathological changes lead to weakness. It has been suggested that microaneurysms could be the cause of the hemorrhage but careful histological studies indicate that fibrinoid necrosis is usually the etiology of the weakness in the arterial wall. The final triggering mechanism for the rupture is not known, although it has been speculated that a sudden increase in blood pressure coincident with factors such as exertion or emotion stress may exceed the tolerance of the vessel wall. This notion is supported by the finding that the setting for the hemorrhage is usually during activity and infrequently during sleep.

Pathological studies demonstrate tracking of blood along tissue planes with displacement of tissue. This latter effect, which is often more evident than gross tissue destruction, provides the basis for hope that the ultimate outcome may be considerably better than indicated by the acute deficit. No correlation has been found between the size of the hematoma and blood pressure.

The mechanism of later clinical deterioration is less certain. Rebleeding is rare. In a study with chromium-labeled red cells injected at the time of admission for hypertensive hemorrhage, it was found that patients who died had virtually no evidence of labeling in the original hemorrhage although the Duret hemorrhages reflected the post-admission fatal cerebral herniation were easily labeled. Occasionally the CT scan will document enlargement of the hematoma and extravasation of dye has been seen in a few arteriograms performed immediately after admission. The chief mechanism for subsequent worsening is the development of edema and ischemic necrosis around the lesion.

Reduction in the hematoma size is accomplished by reparative mechanisms over several months. The process is slow because macrophage activity must occur along the rim of the mass to reabsorb the hematoma. Within a year the hematoma site is converted to a slit-like cavity with orange-stained walls representing hemosiderin-laden macrophages surrounded by tissue which appears more or less normal.

Diagnostic Studies

Any patient suspected of having a brain hemorrhage should have an immediate CT scan. The scan gives a precise localization of the hemorrhage, outlines its size and configuration, and shows the degree of edema in adjacent brain tissue. The scan can also be repeated as needed for evaluation of the subsequent clinical course.

Over several weeks, the high density seen on CT scan gradually becomes isodense, and then changes to a low density appearance. This change in the scan appearance is due to an alteration in photon absorption rather than actual resorption of the hematoma as shown by CT-autopsy correlation. From a few days to some months after hemorrhage, the CT scan with contrast administration will often show a ring-like enhancement around the hemorrhage which presumably represents the area of edema or local ischemic infarction.

There is almost no indication for lumbar puncture in

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a patient suspected of having brain hemorrhage. It should not be used as a diagnostic study because large hemorrhages can lead to transtentorial herniation, and in small hemorrhages, the spinal fluid may be clear.

When the clinical syndrome and CT scan indicate a typical hypertensive hemorrhage, particularly in the putamen, thalamus, cerebellum, or pons, angiography is rarely indicated. In cases where hypertension is not the likely cause and in lobar hemorrhages, angiography is needed to help decide whether there is a vascular malformation or tumor. However, the procedure may fail to show either of these lesions, particularly in the acute phase. We recommend that if the initial angiogram is negative and no other cause for the hematoma is apparent, the study be repeated in 2–3 months when pressure from the hematoma has subsided. If no abnormality is seen at that time, we would continue to follow the patient with a CT scan at 4–6 month intervals to be certain that an underlying tumor is not being overlooked as a cause for hemorrhage.

Every patient with brain hemorrhage should have coagulation parameters checked. These should include prothrombin time (PT), partial thromboplastin time (PPT), and platelet count. In patients known to be receiving aspirin, bleeding time should be measured.

Medical Treatment

When a diagnosis of brain hemorrhage has been established by CT scan, measures are taken to normalize blood pressure, prevent recurrent hemorrhage, reduce mass effect, control edema, and prevent seizures. Since most hemorrhages appear to have stopped before the patient arrives in the hospital, it has been difficult to assess efforts to stop hemorrhage in the occasional case of continued bleeding. Recurrence of hemorrhage is rare, except when the etiology is an aneurysm. Aminocaproic acid has been recommended only in cases of brain hemorrhage due to ruptured aneurysm.

Hypertension is controlled with drug therapy. We do not recommend lowering the blood pressure below normotensive levels since autoregulation is probably impaired in the brain tissue around the hemorrhage and a significant decrease in cerebral perfusion pressure may produce secondary ischemic damage.

When increased intracranial pressure (ICP) is suspected it should be treated vigorously. Steroids may be helpful. We have noted temporary worsening after premature taper of steroid therapy with improvement on restoration of high dosage. However, the benefit of this therapy in a large series of cases has not been established. One controlled study showed no benefit, but the majority of patients were in coma or deep stupor. Intravenous mannitol is an effective and safe therapy for increased ICP. Furosemide can be used alone or with mannitol to potentiate its effect. The stuporous or comatose patient must be intubated to insure adequate ventilation and the maintenance of a normal or preferably moderately reduced pCO2.

Careful management of fluid and electrolytes is required in all patients. One must watch for inappropriate antidiuretic hormone secretion which is not uncommon in these cases. All patients are given anticonvulsant medication.

The best guide to prognosis has been the state of consciousness. Patients with small hematomas will generally respond to medical therapy as will most patients with moderate size hematomas. These patients are awake and can be followed by clinical examination and monitoring of ICP is usually not necessary. The use of continuous monitoring of ICP in the management of patients with large hematomas has been reported. A subarachnoid bolt or ventricular catheter may be used. The effects of hyperventilation, mannitol and furosemide can then be closely followed. When these measures are insufficient to control increased ICP, consideration is given to surgical removal of the hematoma or to treatment with large doses of barbiturates. The latter treatment undoubtedly lowers ICP in some patients where all other measures have failed but it is unknown whether it actually alters ultimate outcome.

Surgical Treatment

The indications for surgical therapy for brain hemorrhage continue to be modified. Although clear-cut indications are not yet available for all patients, clinical and CT guidelines for therapy are emerging.

Several reports have dealt with the timing of operation in patients with hypertensive hemorrhage. Benefit of delayed operation has been assessed by some and of early operation by others. Surgery can be life-saving in the deteriorating patient and of early operation by others. It has not been established whether morbidity can be lessened by immediate or delayed removal of a hematoma in a patient with a stable moderate or severe neurological deficit. Reports of single cases and small series suggest that surgery may diminish late morbidity.

When a hematoma in any location is associated with a history and finding of severely increased intracranial pressure and brain stem compression, emergency surgery is considered depending on the findings on CT scan and physical examination and the initial response to medical therapy. Surgery is generally not undertaken if there is massive hemorrhage with loss of pupillary reaction and brain stem function and no response to the medical therapy.

There is little to be gained by direct surgery in cases of thalamic and pontine hemorrhage. In patients with cerebellar hemorrhage there are distinct indications for surgery and in patients with putaminal and lobar hemorrhages surgery may be considered. This is discussed in the next section.

With the advent of microsurgical techniques there has been more interest in immediate surgery to evacuate the hematoma and obliterate the bleeding vessel. It is claimed that this not only prevents further bleeding but also reduces neurological morbidity by preventing secondary changes such as edema formation and ischemic damage from the persisting mass effect of the hematoma. Further experience is necessary before immediate surgery can be recommended in patients who are stable neurologically.
The operation usually performed is a craniotomy with careful removal of the hematoma, avoiding damage to the surrounding walls. When tumor is suspected the walls of the hematoma must be biopsied carefully. The removal of a hematoma both in the acute and in the chronic stage by CT-guided stereotactic techniques has been reported. The development of specialized instrumentation has made it possible to subtotally remove deep hematomas in a few patients where the lesion could not be approached by conventional operation without disrupting normal brain tissue. Whether this new approach will be safe and effective remains to be proven in a larger series.

Postoperative care must be meticulous in order to avoid recurrence. Blood pressure must be controlled as the anesthesia wears off. Continuous monitoring of blood pressure must be done particularly during the transfer of the patient from operating table to bed and to the recovery room area. Rapidly acting intravenous anti-hypertensive agents are administered as necessary to keep the blood pressure in the normal range. Steroid medication is continued. In supratentorial lesions, an anticonvulsant is recommended. Electrolytes are checked regularly. If the patient’s state of consciousness if reduced, intubation must be continued. The place of continuous intracranial pressure monitoring in postoperative management has not been established.

Any evidence of worsening should lead to a CT scan for evaluation. Postoperative complications include recurrence of bleeding, hydrocephalus, infection and seizures.

Special Clinical Problems and Guidelines For Treatment

Putaminal Hemorrhage

The most common site for hypertensive hemorrhages is the putamen. The hemorrhage may remain localized or it can track into the white matter, into the frontal or temporal lobe, involve the internal capsule or rupture into the ventricle. The larger the lesion, the greater the deficit and the worse the prognosis.

The clinical syndrome is well-described. Patients are characteristically up and active when they become aware that something is wrong. Then a hemiparesis emerges smoothly and steadily. This may progress to a hemiplegia and in some cases it is accompanied by hemisensory loss, hemianopia, dysphasia if the dominant hemisphere is affected and unawareness of the deficit if the non-dominant hemisphere is involved. There is frequently conjugate deviation of the eyes to the side of the hemorrhage. The syndrome may cease at any point or continue to coma and death within a few hours. In 27 consecutive cases, a smooth onset characterized 62% while 30% developed symptoms so rapidly that observers felt that deficit was nearly maximal at onset. None of the patients experienced fluctuation of the deficit. Headache affected only 14% at onset and only 28% at any time, leaving nearly 72% free of headache even in the presence of substantial focal neurological deficit. On examination, none showed papilledema or subhyaloid pre-retinal hemorrhages. Some form of motor deficit affected all cases, varying from mild to complete paralysis. Sensory disorder was not fully evaluated in some cases but approximately 65% of the patients tested showed some alteration in response to pin prick.

Most small and many moderate-sized hematomas in the putamen make a good recovery either spontaneously or with medical treatment. With hematomas larger than 3 cm in diameter, the initial treatment is usually medical but if the patient shows signs of increasing neurological deficit or decreasing state of consciousness in spite of vigorous medical therapy, surgical removal of the hematoma is considered.

In an evaluation of the CT scans in 24 patients with putaminal hemorrhage, three groups were defined. In the first group, patients comatose on admission were found to have massive hemorrhages and a poor prognosis. The second group were alert with substantial neurological deficit and moderate-sized hematomas. A few made acceptable recoveries, but the majority were left with a significant deficit. The third group had only mild deficits, were found to have small hemorrhages on the CT scan and they generally made a good recovery. Whether surgery would have improved the outcome in the first two groups is not known.

Thalamic Hemorrhage

The classic features include as initial deficit a hemisensory loss and, when the internal capsule becomes involved, hemiparesis. Extension into the upper brain stem is common and can cause vertical gaze palsy, retraction nystagmus, skew deviation, loss of convergence, ptosis and miosis, anisocoria, or unreactive pupils. Dysphasia may occur in patients with left-sided hemorrhages. If the hematoma is large, deep coma may be present from the onset. Headache is rare. Compression of the cerebrospinal fluid pathways may cause hydrocephalus.

In 18 patients with hypertensive thalamic hemorrhage, the diagnostic clinical features were limitation of vertical gaze, downward eye deviation, and small but reactive or sluggish pupils. All had a contralateral sensory-motor deficit. Headache was present in only 20–40% of these patients. The motor deficit was similar to that of putaminal hemorrhage. The sensory deficit was often of striking severity and widely distributed over the limbs, head, face and trunk on the affected side. These findings were confirmed in another report of 23 patients.

In two reports of 41 patients with thalamic hemorrhage, it was found that all died when the hematoma was greater than 3.3 cm on the CT scan. Patients with smaller hematomas recovered but often with disability. There is no evidence to indicate whether direct surgery would be of benefit to the patients with larger hematomas. In general we have not operated on patients with thalamic hemorrhage except to treat hydrocephalus. This may develop acutely and requires emergency ventricular drainage. In some patients placement of a permanent internal shunt is required.
Lobar Hemorrhage

Ropper and Davis have characterized the syndromes associated with lobar cerebral hemorrhages in 26 cases. Occipital hemorrhage (11 cases) caused severe pain around the ipsilateral eye and dense hemianopia. Left temporal hemorrhage (seven cases) began with mild pain in or just anterior to the ear, fluent dysphasia with poor auditory comprehension but relatively good repetition and a partial hemianopic visual deficit. Frontal hemorrhage (four cases) caused a distinctive syndrome beginning with severe contralateral arm weakness, minimal leg and face weakness, and frontal headache. Parietal hemorrhage (three cases) began with anterior temporal headache and hemisensory deficit, sometimes involving the trunk to the midline. A right temporal hemorrhage (one case) arrived in coma and no clinical syndrome could be assessed. The authors concluded that spontaneously lobar hemorrhage and branch artery embolization in the same region produced similar clinical syndromes. When there is hemorrhage, headache is usually the first prominent symptom. Its onset is rapid but not instantaneous and, when combined with one of the typical syndromes, suggests lobar hemorrhage rather than another type of stroke. In these 26 patients 8 (31%) were known to have had hypertension prior to the hemorrhage, 14 (54%) had documentation of normal blood pressure, 2 (8%) were on anticoagulants, 2 (8%) were found to have an AVM and 1 (4%) had a metastatic tumor. In another report of 22 patients with lobar hemorrhage, 45% were hypertensive and bleeding had occurred from a metastatic tumor in 14%, an AVM in 9%, a blood dyscrasia in 5% and in 27% the cause was unknown.

Most of the spontaneous or hypertensive lobar hematomas can be treated medically and will make a good recovery without surgical treatment. However, if the patient is showing signs of increasing neurological deficit in spite of medical therapy, surgical removal of the hematoma is indicated.

Cerebellar Hemorrhage

Hemorrhage in the cerebellum causes a life-threatening syndrome which can be reversed by prompt surgical treatment. Classically, the onset of this hemorrhage is sudden, with nausea, vomiting, and inability to stand or walk. In a series of 56 patients with cerebellar hemorrhage, headache was present in 75%, dizziness in 55%, and loss of consciousness at onset in 14%. Examination showed appendicular ataxia in 78%, facial palsy in 60%, and ipsilateral gaze palsy in 54%. No distinctive clinical feature could be delineated in the acute state in non-comatose patients to predict those who would survive with minimal or mild disability and those who might progress to brain stem compression and coma.

The cerebellar hematoma represents a special situation regarding treatment. Deterioration due to brain stem compression is unpredictable, and often irreversible once set in motion. It is critically important to treat the patient before compression causes alteration in the state of consciousness and an unstable clinical situation. In our experience, 10 of 12 patients who were alert or drowsy preoperatively survived operation, while only four of 16 patients who were stuporous or comatose before surgery lived. The relationship of the level of consciousness to prognosis and the importance of not delaying surgery in patients with acute cerebellar hematomas has been stressed in other reviews. It should be pointed out, however, that even for a patient in deep coma emergency evacuation of the hematoma can result in good recovery, especially if the time interval between the development of the comatose state and surgery is short. Therefore, operation should not be denied to a patient who is in coma as a result of cerebellar hemorrhage unless there is clinical or CT evidence that the brain stem has been destroyed.

In a report of 10 patients with cerebellar hematoma, six had a progressive course with early brain stem compression and all had hematomas 3 cm or greater on CT scan. We generally recommend removal of hematomas that are greater than 3 cm in diameter if the patient is seen within the first week of the onset of symptoms. Patients seen later who have a stable neurological course may be treated with medical therapy but are closely observed.

Those patients with smaller lesions and no signs of brain stem compression are monitored carefully in the intensive care unit. Many patients with small hematomas have a benign course and may be treated medically with good results. However, once the patient shows signs of brain stem compression deterioration can proceed at an unpredictable rate and therefore operation is indicated even if the hematoma appears smaller than 3 cm.

The hematoma is removed under direct vision via a suboccipital craniectomy. Undoubtedly, a few patients can be treated satisfactorily by ventricular drainage, but this therapy alone does not relieve brain stem compression and may be followed by late deterioration. However, ventricular drainage may be necessary after evacuation of the hematoma to treat persisting hydrocephalus.

Pontine Hemorrhage

This is one of the most dramatic and least treatable of all brain hemorrhages. A small hematoma often leads to immediate coma, rapid quadriplegia, decerebrate rigidity, pin-point pupils which may be barely reactive to light, and a variety of ocular motility disturbances. The uncommon smaller hemorrhage may cause the patient to be paralyzed but able to communicate by ocular movements ("lock-in" state). Most patients do not survive the acute phase.

A few cases of successful removal of a pontine hematoma have been reported. Sano and Ochiai reviewed 24 patients with pontine hematoma due to hypertension. Six with hematomas less than 1.0 cm in diameter as shown by CT survived but only one patient is working. All patients with hematomas larger than 1.0 cm died except for one. Four had suboccipital craniectomy for removal of the hematoma and several had ventricular drainage. It was concluded that direct operation was of doubtful value.
Intraventricular Hemorrhage

With the use of the CT scan, the degree of intraventricular hemorrhage can be readily recognized. Most intraventricular hematomas result from rupture of a parenchymal hematoma into the ventricles but occasionally primary intraventricular hemorrhage can occur. Although traditionally thought to have a poor prognosis, intraventricular hemorrhage can be associated with a relatively benign clinical course. The spontaneous resolution of intraventricular hemorrhage was originally documented by pneumoencephalogram and has been well demonstrated with the CT scan. A review of 54 patients with intraventricular hemorrhage on the CT scan revealed an association with a large number of disorders including hypertension, saccular and mycotic aneurysm, AVM, tumor, and coagulation disorders. In this series 78% of the cases were associated with intraparenchymal hematoma. Hypertensive hemorrhage in any of the common sites can rupture into the ventricle. In a report of 32 patients with hypertensive intracerebral hemorrhage, 62% had intraventricular rupture. Intraventricular hemorrhage also occurs from rupture of an aneurysm, and these hemorrhages are generally more extensive than those due to other causes.

The guidelines for surgical treatment are generally the same as those outlined for the parenchymal site of the hemorrhage. Ventricular drainage has usually not been helpful because the catheters frequently become obstructed. However, in an occasional patient emergency ventriculostomy can be life-saving and is indicated when neurological deterioration appears to be secondary to acute hydrocephalus from intraventricular hemorrhage. The less severe cases who survive the acute phase commonly require placement of a shunt.

Hemorrhage due to Aneurysms, Arteriovenous Malformations and Tumors

About one-fifth of patients with aneurysmal hemorrhage and one half of those with hemorrhage from an AVM will have an associated intracerebral hematoma. When either aneurysm or AVM is suspected in a patient with an intracerebral hematoma, angiography is indicated. The indications for surgical removal of the hematoma are essentially the same as in other forms of spontaneous intracerebral hemorrhage.

In the aneurysm patient the timing of operation to repair the aneurysm should not be influenced by the intracerebral hemorrhage unless the patient is deteriorating as a result of the mass effect. In patients who require early operation to remove the hematoma every effort should be made to repair the aneurysm at the same operation to prevent postoperative rebleeding.

In patients with a ruptured AVM the incidence of early rebleeding is low. If the hematoma has to be removed urgently and the AVM is not readily accessible or would require extensive surgery, excision can be delayed for several weeks until brain swelling has subsided. Occasionally a "cryptic" AVM will be discovered at surgery in a patient with an intracerebral hemorrhage and negative angiogram.

Brain tumor is responsible for about 10% of all spontaneous intracerebral hemorrhages. Metastatic tumor, particularly melanoma and glioma, are responsible for the majority of these cases. Pituitary adenomas bleed frequently but the hemorrhage is usually within the tumor and rarely results in intracerebral hemorrhage. Whenever a tumor is suspected as the cause of intracerebral hemorrhage because of clinical, CT or angiographic findings the hematoma should be removed with thorough exploration and biopsy of the wall of the hematoma cavity. If a diagnosis is not established and the suspicion is still high, follow-up CT scan and angiography are indicated.

Hemorrhage Due to Amyloid Angiopathy

Amyloid angiopathy is an infrequent cause of spontaneous lobar brain hemorrhage. It usually occurs in patients over 60 years of age and may be associated with multiple hemorrhages. When lobar intracerebral hemorrhage is found in a normotensive elderly patient, this diagnosis must be considered. If such a patient has had two or more hemorrhages in different sites this diagnosis becomes the primary consideration. Whenever amyloid angiopathy is a prominent diagnostic possibility every effort should be made to treat the patient medically since surgical evacuation is frequently attended by uncontrollable hemorrhage and postoperative recurrent bleeding. If surgery becomes necessary because of continuing neurological deterioration despite vigorous medical therapy, care should be taken to disturb the walls of the hematoma cavity as little as possible to avoid further bleeding from the fragile amyloid laden parenchymal vessels.

Hemorrhages Due to Coagulation Disorders

Anticoagulant Therapy

With the widespread use of anticoagulants for treatment of a variety of disorders, the number of patients with brain hemorrhage due to this cause has increased. The majority of patients who develop this complication are found to have either a prothrombin time longer than the therapeutic range or a focal lesion such as infarction to account for bleeding.

In a patient on anticoagulants an isolated intracerebral hemorrhage may occur in the absence of bleeding in other areas of the body.

The initial evaluation and treatment is the same as described for hypertensive brain hemorrhage. Immediate transfusion of fresh frozen plasma reverses anticoagulation. To maintain hemostasis, administration of Vitamin K1 (phytonadione) preparation usually restores normal coagulation within 6 hours except in patients with significant liver disease. With these measures, operation can usually be safely performed. Prothrombin time should be rechecked postoperatively and corrected as necessary.

Thrombocytopenia

The normal blood platelet count is 100,000 to 400,000 per mm³. Thrombocytopenia is diagnosed when the platelet count is less than 80,000/mm³.
is a common cause of clotting deficiency. Intracerebral hemorrhage due to thrombocytopenia has been reported in idiopathic thrombocytopenia purpura and in a variety of disease states in which there is secondary thrombocytopenia. The initial symptoms of spontaneous intracranial bleeding in these patients is usually headache followed by deterioration in the level of consciousness. The onset is often insidious, coming on over several days. Usually the hemorrhage is intracerebral, but subdural hemotoma can occur.12

Surgery is hazardous if the platelet count is below 50,000 and is of concern when the count is 50,000–100,000. It is usually possible to achieve a hemostatic level by a combination of platelet transfusions and administration of corticosteroid drugs which often have effects on the hemostatic mechanisms as well as on the underlying disease process. Guidelines for surgery on the hematoma are the same as previously stated. In a report on intracranial hemorrhage in children with idiopathic thrombocytopenia purpura, it was recommended that emergency splenectomy be done prior to neurosurgical intervention.83,84

Hemophilia

The vast majority of hemophilic patients are deficient in factor VIII, a small number in factor IX, and an occasional patient in factor XI. In all patients there is a prolonged partial thromboplastin time. The problem has been summarized in several reports.84,85

Brain hemorrhage usually is associated with mild trauma but it can occur spontaneously. Any patient with hemophilia who complains of persistent headache should have a CT scan. When hemorrhage is confirmed, appropriate replacement treatment should be started immediately. To prevent further spontaneous, intra-operative or postoperative hemorrhage, it is necessary to maintain a minimum level of at least 20% of the deficient factor with transfusions of the appropriate concentrate. If an operation is done, replacement needs to be given until the incision is healed.86 The indications for operation are the same as those previously outlined.

Summary

Spontaneous brain hemorrhage accounts for about 10% of all strokes and is fatal in about 50% of the cases. Its incidence, in contrast to other types of strokes, has not declined. Hypertension accounts for about half of these hemorrhages; the rest are due to tumors, aneurysms and vascular malformations, inflammatory and degenerative vasculopathies and hematologic and iatrogenic disorders of coagulation. In some patients no cause is ever found. Hypertensive brain hemorrhage occurs in the deep gray nuclei of the hemispheres, the cerebellum, and the pons and results in specific clinical syndromes depending on the location.

Computerized tomography has revolutionized the diagnosis of brain hemorrhage and is resulting in the development of rational criteria for medical and surgical management of these lesions. Intensive medical therapy guided by clinical status and continuous monitoring of ICP may improve outcome. Surgical removal of the hematoma is indicated in lobar and putaminal hemorrhages when the patient is deteriorating in spite of vigorous medical therapy. In addition most large (>3 cm) cerebellar hemorrhages, as well as smaller cerebellar hemorrhages that result in significant brain stem compression should be evaluated. The roles of intensive medical therapy, elective late surgery and of immediate operation in improving eventual functional outcome need to be investigated further.

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Recent Progress in the Role of Platelets in Occlusive Vascular Disease

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Platelets are derived from bone marrow megakaryocytes by extension of cytoplasmic processes which undergo attenuation, develop constrictions at the distal ends, and then rupture, thereby releasing platelets. During its life span of approximately 10 days the unstimulated platelet functions in some unknown manner to maintain vascular integrity. The platelet is also the only blood cell component involved in the uptake and transport of serotonin (5-HT). If blood vessel continuity is interrupted, the vessel contracts and concomitantly platelets at the site are activated. Activation can also take place intravascularly by pathological stimuli such as endotoxin and immune complexes generated in certain disease states. Adhesion of platelets in proximity to the subendothelium occurs and this is accompanied by aggregation (cohesion) of additional platelets which have been 'recruited' into the microenvironment. This sequence (primary hemostasis) is modulated by adhesion of platelets to subendothelial collagen, formation of thromboxane A_2 (TXA_2), mobilization of intraplatelet calcium, release of adenosine diphosphate (ADP) and 5-HT (fig. 1). Platelet activation also results in exposure of specific surface receptors which bind fibrinogen and this bound fibrinogen is a cofactor for aggregation. Such exposure is inhibited when platelet cAMP levels are elevated by agents such as prostacyclin (PGL_2).

The stimulated platelet undergoes a unique morphological change from that of a disc to a spiny sphere and the surface membrane phospholipoprotein develops the capacity to catalyze interactions between activated coagulation proteins, culminating in thrombin formation and fibrinogen polymerization (secondary hemostasis). The process of clot retraction is initiated when platelets form pseudopodia which adhere to fibrin strands at points where the strands cross one another. The platelet pseudopodia then contract and draw the sides of injured vessels together. Clot retraction requires ATP, glucose, calcium and normal fibrin formation. Formation of the hemostatic platelet plug is then complete.

Stimulated platelets also secrete proteins which were originally synthesized in the megakaryocyte. Among these are platelet factor 4 (PF-4), which has anti-heparin properties and can react with heparan sulfate in the vessel wall. The platelet-derived growth factor (PDGF) which stimulates smooth muscle cell proliferation is also released. PDGF has been implicated in the atherosclerotic process.

Platelets and Thrombosis

Arterial thrombi resemble hemostatic plugs in that they form via an interaction of platelets with an injured vascular surface. Morphologically such thrombi contain mainly adherent platelets at the interface and a mixture of leukocytes, fibrin and erythrocytes in the distal portion. These observations prompted the use of pharmacologic agents capable of suppressing platelet aggregation, release and adhesion. In the early 1970's clinical trials were initiated with the overall goal of attempting to prevent or reduce platelet accumulation in diseased vessels of the heart, brain, extremities and on vascular prostheses. To date analyses of almost every clinical trial have been fraught with interpretive difficulties. Attempts were initially made to prolong platelet survival with platelet inhibiting drugs plus anticoagulants in patients with valve prostheses and with aortocoronary bypass grafts, since shortened platelet survival as measured by isotopic techniques correlated with malfunctioning prostheses and thromboembolic phenomena. In arterial thromboembolism, increased con-
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