SPECIAL ARTICLE

INTRACEREBRAL HEMORRHAGE ASSOCIATED WITH HYPERTENSION AND ARTERIOSCLEROSIS*

LEO M. DAVIDOFF, M.D.†

During a symposium on "Cerebrovascular Disease with Aging" recently conducted by Wright, Adams, Covalt, Fazekas, and Merritt¹ at the New York Academy of Medicine, some pertinent statistics were cited concerning the increasing problem of cerebrovascular accidents in our population. In 1952 about 170,000 people, more than 44,000 of them under the age of 65, died of cerebral vascular disease in the United States. Moreover, an estimated 1,800,000 still alive are known to have suffered some manifestation of cerebral vascular disease. Many of these are sufficiently incapacitated to require the full-time or part-time services of from one to three other persons. Many thousands are occupying beds in nursing homes, in private, municipal, state or federal veterans' hospitals for prolonged periods of time, and thousands more represent heavy burdens to their families at home. While spontaneous intracerebral hemorrhage represents only a portion (about 25 per cent) of this serious problem, it nevertheless proved to be, according to a Metropolitan Life Insurance Company Bulletin in 1946, the fourth most common cause of death in the United States of America. Indeed, the mortality from spontaneous intracerebral hemorrhage, without surgical interference, is appallingly high. Thus Aring and Merritt¹ found a mortality of 98 per cent in 116 cases. Rose¹⁰ reported on 205 patients, of which 80 per cent had died within 24 hours. Zimmerman,¹⁴ who reviewed 107 cases, described a 94 per cent mortality with the first attack, and in the remainder death occurred within 5 weeks after the onset.

There are many conditions that may be responsible for intracerebral bleeding. Among these are hypertension, arteriosclerosis, cerebral aneurysm, cerebral vascular anomalies, septicemia, subacute bacterial endocarditis, brain tumors, periartheritis nodosa, lupus erythematosus, tuberculosis, syphilis, eclampsia, trauma, blood dyscrasias, and scurvy. Hemorrhage may also occur when heparin or other anticoagulants are used in the treatment of thromboses. It is clear that in each of these conditions the cerebral hemorrhage is an incident in an underlying disease, and the success or failure of treatment of the hemorrhage depends often upon the prognosis in the disease responsible. On the other hand, the catastrophic occurrence of a massive hemorrhage in a chronic condition like hypertension or arteriosclerosis, or a silent condition like a berry aneurysm or vascular anomaly, is frequently

* Prepared at the request of the Editorial Board of the Journal of Neurosurgery.
† Professor and Chairman of the Department of Surgery, Albert Einstein College of Medicine, Yeshiva University, New York, New York.
the precipitating cause of death, unless, by surgical evacuation of the clot, the immediate danger can be allayed.

Very few clinics are able to accumulate a sufficient number of surgically treated cases with any one type of etiologically based hemorrhage, and the majority of reports in the literature include the author's experience with all his cases of hemorrhage of whatever etiology. The result is likely to be confusing, since material reported under similar titles often deals with widely dissimilar situations. Since in the majority of cases cerebral hemorrhage is caused by hypertension and arteriosclerosis, I propose, therefore, to discuss this type alone in order to help crystallize our thinking with regard to this major segment of our problem.

Jewesbury,7 in a paper on "Atypical Intracerebral Haemorrhage," in defining the typical case with which he did not intend to deal, inadvertently succinctly epitomized the very situation that I wish to discuss here:

"The hypertensive, rather elderly man, while at work, suddenly develops headache which is often associated with vomiting. Consciousness is quickly lost and there are signs of hemiplegia. Blood may be found at lumbar puncture and death occurs in the course of a few hours or days. At autopsy arteriosclerosis is commonly found and the internal capsule and basal ganglia are the site of a haemorrhage from rupture of a basal branch of the middle cerebral artery."

Let us see how this definition conforms to the combined experience of the many authors in their publications on this subject. The number of pertinent reports in the world literature is voluminous, but fortunately there is now available the detailed summary of all of these contributions in the monograph by Guy Lazorthes on L'hémorragie cérébrale vue par le neuro-chirurgien.

In this we learn that the patient is, indeed, in the older age group, ranging between 40 and 60 years; those of 65 years of age are more likely to have cerebral softening rather than hemorrhage. Of 21 patients operated upon by me only 1 was under 40 years old, and 4 were over 60. Patients younger than 40, without hypertension, who suffer from intracranial hemorrhage, do so more often as a result either of vascular malformation or of unknown cause. And as for sex, the proportion of males to females is two to one, as reported in the literature. Among the 21 patients in my own series, however, 8 were males and 13 females.

The immediately precipitating cause may be physical exertion, emotional shock, or extremes of temperature, such as severe cold in midwinter or extreme heat in midsummer.

In the majority of instances the vessels involved are the lateral striate

---

* Dorothy Russell6 in 1954 found that out of 461 cases of intracerebral hemorrhage coming to autopsy at the London Hospital between 1912 and 1932, 292 were in patients who had suffered from vascular hypertension (54 per cent). Lazorthes8 reported that out of 345 cases of hematoma treated by surgery, in the literature, on which figures were available, arterial hypertension was present in 28 per cent of the cases. Since surgical reports are likely to be on selected cases, the proportion of hemorrhage in patients with hypertension in Russell's figures is probably more representative of the actual situation. In Lazorthes' personal series there were 17 patients with vascular hypertension out of 35 cases, and in mine7 there were 21 out of 33 cases.
branches of the middle cerebral artery. Modern anatomists find it impossible to distinguish among these branches any individual artery like the "lenticulostriate" artery of Duret or the "artery of cerebral hemorrhage" of Charcot. In rare instances intracerebellar hemorrhages may also occur.

There is considerable disagreement about the pathogenesis of cerebral hemorrhage in hypertensive individuals, and probably the mechanism may vary from case to case. Thin vessels may rupture as a result of vasospasm if, indeed, such a condition ever occurs. Necrosis of the wall of the vessel, tiny aneurysms or angiomas, or physicochemical changes in the brain surrounding a vessel may give rise to diapedesis of red blood cells.

Unlike the hemorrhages of the brain seen with aneurysms, those associated with hypertension usually begin deeply in the basal ganglia and may dissect their way peripherally. The hemorrhage may remain small and confined to the region of the internal capsule, in which case the clinical course may be like that of a cerebral thrombosis. It may be violent, involve vital centers quickly, or rupture into a ventricle when it is rapidly fatal. Finally it may dissect its way into the white matter of the occipital, temporal, or frontal lobes and then stop bleeding as a result of tamponade not only by the mass of the hemorrhage itself but by the edema of the surrounding brain. It is the latter case in which surgical evacuation of the clot may be expected to be successful in saving the patient's life, and reducing the neurological deficits that may have resulted, had he survived and recovered spontaneously.

Clinically the onset is usually abrupt, although premonitory symptoms such as headache, paresthesias, and aphasia may be elicited in the history. The patient usually complains of sudden severe headache, vomits, and rapidly passes into coma. Occasionally convulsions occur. As the coma deepens, the face becomes flushed, the pulse quickens and breathing becomes stertorous and may become Cheyne-Stokes in character. If the hemorrhage ruptures into the ventricles, the temperature may become very high, but may also fall to subnormal levels. The pupils fail to react to light and are often unequal. The eyes may be found deviated, usually toward the side of lesion. All deep and superficial reflexes, except for Babinski's sign, which may be bilateral, are usually abolished and the limbs are flaccid. If the coma is not too deep and the patient may be made to respond to painful stimuli, the failure of one side to respond then indicates the side of the hemiplegia.

In contrast to this, the patient who suffers a cerebral thrombosis is usually older. The onset is more gradual. Headache is either absent, or mild in degree. There is seldom any loss of consciousness. Vascular hypertension is not a prominent sign in the majority of cases. However, the differential diagnosis between hemorrhage and thrombosis is sometimes quite difficult.

Since there has also been an upsurge of interest in more active treatment of thrombosis of cerebral vessels producing strokes, by the use of anticoagulants, stellate ganglion blocks, vasodilatory drugs, etc., it must be clearly understood that these therapeutic measures which are being tried for cerebral artery thrombosis are dangerous when applied to cases of cerebral
hemorrhage. Conversely, craniotomy is of no earthly use in cases of thrombosis. This points up the importance of accurate differential diagnosis in these cases. It is no longer sufficient for a physician to make a diagnosis of a "cerebral vascular accident." He may now wish to institute specific treatment, and unless he can be sure of the diagnosis, he may treat a patient with hemorrhage with anticoagulants and increase the bleeding, or subject a patient, already debilitated by cerebral thrombosis, to a major operation unnecessarily.

In the nonfatal cases of hemorrhage, the clinical picture becomes stabilized and the patient may actually improve. Pulse, respirations, and temperature may approach normal, and coma may lessen. The patient shows some spontaneous movements, groans, and responds to painful stimulation. The hemiplegia may now be more evident. Hemisensory deficits may become demonstrable. Visual-field defects may be picked up, and disturbance of speech can be demonstrated when the dominant hemisphere is involved. The improvement, however, may be only transient, lasting perhaps a week or two. The vascular hypertension, which may not have been apparent in the acute stage, may reappear. Papilledema may develop. Additional bleeding, reactive cerebral edema, or hypostatic pneumonia may supervene and lead to a fatal outcome.

The laboratory aids to diagnosis of cerebral hemorrhage have little to offer in the routine examination of the blood and urine, except for a leucocytosis in the peripheral blood varying between 12,000 and 22,000.

Lumbar puncture may reveal clear, colorless fluid, with or without microscopic evidence of red cells, xanthochromic fluid, or grossly bloody fluid. However, in most instances the initial pressure is elevated (90 per cent according to Scott). This again is in contrast to what is found in cerebral thrombosis, in which the fluid is usually clear, colorless, and under normal pressure.

Plain roentgenograms of the skull may show displacement of the pineal shadow (in 4 of my 21 cases) toward the side opposite the hemorrhage, and occasionally (as in 2 of my cases) skull fractures sustained during a fall caused by the sudden onset of the hemorrhage.*

The electroencephalogram is usually abnormal in cases of cerebral hemorrhage. In those with deep-seated lesions the abnormal electrical activity may be diffuse, with the greater abnormalities lateralized to the side of the lesion. In the hemorrhages which reach closer to the cortex a sharp focus of slow-wave, high-voltage activity is often demonstrable.

Cerebral angiography has been probably the greatest single stimulus to the reappraisal of these cases from the neurosurgical point of view. Dangerous side effects or sequelae from the procedure have been largely eliminated. Even the patients in serious condition may be examined safely. This is especially true when an apparatus employing dual roentgen-ray tubes is used, so that only a single injection of opaque substance need be made to ob-

* Intracerebral hemorrhage secondary to trauma is not here under discussion.
tain serial angiograms in both anteroposterior and lateral projections. Cerebral angiography has multiple advantages. In cases of cerebral hemorrhage not here under discussion, namely those resulting from ruptured aneurysm or vascular anomaly, the source of the bleeding may itself be demonstrable. But even in the cases in point—associated with hypertension and arteriosclerosis—the presence of a mass and its exact localization is usually determinable from these studies. Except in the relatively rare instances of cerebellar hemorrhage, ventriculography, which is usually much less well tolerated, may usually be excluded.

The primary concern of the present essay is the surgical treatment of these patients in so far as they can be treated at all. Nonsurgical treatment is primarily supportive: To keep the air passages clear, to alleviate marked vascular hypertension, to try to reduce excessive intracranial pressure, to try to stop the bleeding by favoring coagulation. The accumulated blood, that is, the lesion itself, can be evacuated only by surgical means. The purpose of the operation should be, first, to interrupt the progressively increasing intracranial pressure which could be fatal if allowed to continue, and second, to minimize the sequelae. The finest surgical judgment, however, must be exercised in determining when the lesion is so small that nature will take care of it without interference, or when the hemorrhage is so massive and its effects so overwhelming that nothing can save the patient from a fatal outcome. In all other cases the surgeon must be prepared to operate.

In the more favorable cases the usual course of events is the primary period of shock and catastrophe, followed in a day or two by stabilization or even amelioration of the clinical picture, and after 8 to 14 days a secondary relapse. If there were a way of determining during the dramatic acute phase whether in a given case the stabilization or even amelioration were going to take place, surgical interference could be delayed until this period had been reached and before the downhill progress resumed. Unfortunately this is not always possible, and such authors as Hamby,6 Guillaume,4,5 Fazio,3 and Riishede9 advocate early operation, especially in cases of hematomas of the temporo-occipital region, on the assumption that some who might otherwise die can thus be saved, while in the others nothing is lost anyway.

Lazorthes, after evaluating all the available literature and based on his own experience, believed that the operation is best carried out, and with greatest hope of success, if it can be delayed until the end of the first week after the acute episode. My personal experience leads me to feel that the prognosis as to the operative results depends more upon the condition of the patient at the time surgery is undertaken than upon the time interval after the ictus. These two points of view are actually not very far apart. By the time a patient reaches the neurosurgeon’s hands, an interval of 4 or 5 days has usually intervened since the acute episode, and no time should be lost to complete the necessary work-up, including angiography, and to proceed with the operation if one is to be done. I believe the attitude, expressed particularly by Riishede, that all patients should be operated upon since there is nothing to be lost, is forfeiting the surgeon’s power of discretion, which
should, on the contrary, be exercised increasingly with his experience and skill. Obviously no good can come of operating upon patients who are completely comatose, with high fever, rapid pulse, Cheyne-Stokes respirations, densely bloody cerebrospinal fluid under high pressure, and a high degree of vascular hypertension. If, on the other hand, after a few days of supportive therapy, these signs improve, operation may then be indicated and may have some chance for success.

The method of operation should be by cranioplasty, with evacuation of the clot under direct vision, and occlusion of bleeding vessels if identified. The probing of the hemorrhagic cavity through drill holes, burr holes, or trephine holes, I believe, should be abandoned, except perhaps in a patient in extremis whose lesion is well localized, who fails to rally, and in whom a major craniotomy would surely be fatal. At best it is a job half done, since usually the lesion consists as much or more of solid clot than of liquid blood. There is, moreover, danger of additional brain injury, or even of opening a pathway for blood to enter the ventricles, if it has not already done so spontaneously.

Concerning the results of operation in cases of intracerebral hematoma, the difficulty in evaluation is attributable to the marked variation of factors in the various reports. Obviously when cases of varying etiology are lumped together, the good results in young people without hypertension leave the bad results in cases of older patients with hypertension and arteriosclerosis. In my own series there were 14 cases* of young patients without hypertension, all of whom have survived, as against 21 older patients with hypertension, of whom only 11 survived. This would give me an over-all mortality of about 28 per cent. However, the mortality in the 21 cases of patients with hypertension is 47 per cent, or even if we left out 2 cases in which death occurred after attempted ventriculography and before actual operation, the mortality would still be 8 out of 19 cases or 42 per cent.

Lazorthes collected reports on 446 patients from the literature, of which 124 died following operation; that is a mortality again of 28 per cent. But these, too, are cases of hemorrhage based on varying etiological factors. As far as his own cases go, out of a total of 35 patients with 34 operations, 17 of which were in elderly hypertensives, there were 6 operative mortalities, of which 5 belong to the hypertensive group, or 29 per cent of the 17 cases.

What condition the survivors are in some years after operation is quite another matter. The majority of younger patients without hypertension are fully restored to health. Seven out of 10 of my 14 patients who were followed for from 3 to 19 years, reported themselves to be in good health. Three were partially restricted by residual hemiparesis. Three of the 10 reported one or more convulsions, controlled by medication.

In the hypertensive group, however, of the 11 who survived only 8 could be followed for from 2 to 23 years. Five of them were restored to full activity (one in spite of residual hemiparesis). One was partially restricted because

* Two additional cases have been added since my report in 1957.
of hemiplegia. Two are invalids because of hemiplegia and hypertension.

Of the mixed group of 446 cases from the literature referred to above, there were 159 patients surviving operation, of whom 69 (35 per cent) were cured, and 90 (65 per cent) were suffering from sequelae.

Of Lazorthes’ 17 patients with hypertension, the 12 survivors included 6 who were fully restored and 6 who were suffering from sequelae.

In summary, then, the increasing longevity of our population is producing an increasing number of patients suffering from cerebral arteriosclerosis and vascular hypertension, giving rise to an increasing number of cases of intracerebral hemorrhage.

The diagnosis of this condition, based on clinical and laboratory examinations including cerebral angiography, can be made quite accurately in the majority of instances.

Treatment by supportive measures is indicated as the only therapy in some cases, and as adjunct to surgical treatment in others.

Surgery is indicated in those cases in which massive hemorrhage occurs, and in which the patient is not obviously in extremis.

The results of surgical treatment are still not brilliant, either in terms of mortality or morbidity, but are so vast an improvement over the almost 100 per cent mortality in the nonoperative cases that surgeons should persist in their efforts to acquire experience, and perfect technique in this field, in order to increase their effectiveness in the treatment of these patients.

REFERENCES