Surgical Treatment of Primary Intracerebral Hemorrhage. Part 1: New Angiographical Classification

MASAHIRO MIZUKAMI, M.D.* HIROSHI KIN, M.D.* GORO ARAKI, M.D.† HIROSHI MIHARA, M.D.† AND YOJI YOSHIDA, M.D.‡

SUMMARY  A new angiographical classification of primary intracerebral hemorrhage is presented.

We have clarified the predilection sites of intracerebral hemorrhage and the advancing direction of the hematoma by studying autopsy cases. Furthermore, we tried to detect the presence or absence of destruction of the internal capsule and ventricular classification system based on clinicopathological studies which, along with the patient's level of consciousness, is felt to be the most important indication for operation.

Introduction

CEREBROVASCULAR DISEASE has been the most frequent cause of death in Japan since 1952. About half of the patients (90,000 every year) die from primary intracerebral hemorrhage, which usually occurs in elderly persons with arteriosclerosis and/or hypertension. However, very little progress has been made in the treatment of this disease in spite of the rapid advances in medicine during the last two decades.

The time has come for us to scrutinize this disease process and attempt to understand its pathology and pathophysiology. With the hope that surgical treatment may offer an effective approach to this illness, we began extensive studies on this subject at the Institute of Brain and Blood Vessels, Mihara Memorial Hospital, 366 Otamachi, Isesaki, Gunma, Japan.

We analyzed 100 cases of primary intracerebral hemorrhage and the arteries most commonly involved and the direction of the ruptured artery by means of angiography. Our classification, introducing the idea of dynamic changes of hematoma advancement from localized to advanced type, can be applied to clinical practice. This classification, along with the patient's level of consciousness, is felt to be the most important indication for operation.

Methods

Our study is based on 60 autopsy cases which had primary intracerebral hemorrhage in which ruptured arteries were examined by microangiography, the cleared specimens immersed in tetrahydronaphthalene, and serial histological examinations performed between 1966 and 1968. We tried to find the arteries most commonly involved and the direction in which the hematoma advanced.

We analyzed 100 cases of primary intracerebral hemorrhage in which carotid angiography was carried out within two weeks after the onset and the diagnosis was confirmed at operation or autopsy during the same period. Hemorrhages due to other causes and verified by angiography, operation and autopsy were excluded.

Results

PATHOLOGICAL FINDINGS

Ruptured Arteries (Figs. 1a and b)

Figures 1a and b show normal microangiograms of perforating arteries supplying the putamen and thalamus, which are the most frequent sites of hemorrhage. There are three to six lateral lenticulostriate arteries that leave the trunk of the middle cerebral artery and enter the base of the
FIGURE 1a. Microangiogram of the lenticulostriate arteries. The most lateral branch of the lenticulostriate distributes the lateral margin of the putamen. The subcortical branches of the insular artery distribute to the external capsule. The watershed (1) between the distribution of these arteries is least resistant to disruptive mechanical forces. Hemorrhages arising from the most lateral branch of the lenticulostriate arteries progress through this area. Hence, the distance between the most lateral point of the lenticulostriate artery and the insular artery (—) always is enlarged. The distal portion of the lateral branch passes through the posterior limb (II).

FIGURE 1b. Microangiogram of the arteries which distribute the thalamus. (1) The thalamogeniculate arteries which mainly distribute the lateral nuclei. (2) The thalamoperforate arteries which mainly distribute the medial nuclei. (3) The medial branches of the posterior choroidal arteries which mainly distribute the anterior nuclei. The thalamic hemorrhage arises from one branch of the thalamogeniculate arteries or the thalamoperforate arteries.

FIGURE 2a. The autopsy finding and microangiogram of the putaminal hemorrhage. The distance between the lateral branch of the lateral lenticulostriate arteries and the insular artery is always enlarged. The ruptured lenticulostriate artery is not seen on this slice.

Advancing Direction of Hematoma (Figs. 3a and b)

We analyzed the advancing direction of the hematoma by tracing the hematoma on both horizontal and coronal brain sections.

In putaminal hemorrhage arising from rupture of the most lateral branch of the lenticulostriate artery, the hematoma advanced along the lateral margin of the posterior choroidal arteries, which are mainly distributed to the anterior nuclei. In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.

In 12 of the 44 cases of putaminal hemorrhage, the rupture was found in the lateral lenticulostriate arteries (fig. 2a). In four of the 16 thalamic hemorrhages, the rupture was found in the thalamogeniculate and thalamoperforate arteries (fig. 2b). In addition, small ruptured arteries were found around the hematoma in most cases. In other cases the ruptured arteries could not be identified due to destruction from the hematoma or due to incomplete histological examinations. However, from analysis of the location and extension of the hematoma, the involved ruptured arteries were suspected to be the lateral lenticulostriate, thalamogeniculate or thalamoperforate arteries.
FIGURE 2b The autopsy finding and microangiogram of the thalamic hemorrhage. The distance between the lateral branch of the lateral lenticulostriate arteries and the insular artery is never enlarged. The ruptured thalamogeniculate artery (1).

The putamen. This margin corresponds to the watershed area between the distribution of the lateral lenticulostriate arteries and the subcortical branches of the middle cerebral artery. When the bleeding further advanced and expanded, it progressed superiorly in an anteroposterior direction and destroyed the internal capsule, and perforated into the frontal horn or trigone of the lateral ventricle between the caudate nucleus and thalamus.

In thalamic hemorrhage, there were two predilecting sites of hemorrhage. One was hemorrhage in the lateral nuclei by rupture of thalamogeniculate arteries, and the other was hemorrhage in the medial nuclei by rupture of thalamoperforate arteries, as mentioned previously. Hemorrhage from thalamogeniculate arteries easily extended to the posterior limb of the internal capsule and advanced through the region of least resistance in this white matter. Advancing further, the hematoma perforated into the trigone and the body of the lateral ventricle.

**TABLE 1** Classification of Primary Intracerebral Hemorrhage

<table>
<thead>
<tr>
<th>Hemorrhage</th>
<th>Type</th>
<th>Location</th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Putaminal</td>
<td>Localized</td>
<td>Internal capsule</td>
<td>13</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Advanced</td>
<td>Ventricles</td>
<td>2</td>
<td>15</td>
</tr>
<tr>
<td>Thalamic</td>
<td>Localized</td>
<td>Lateral nucleus</td>
<td>8</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Advanced</td>
<td>Medial nucleus</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Subcortical</td>
<td>Advanced</td>
<td>Whole thalamus</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

S.P.: sylvian point.
Hemorrhage from the thalamoperforate arteries easily perforated into the third ventricle and developed within the thalamus. When the bleeding advanced further, it directed itself toward the hypothalamus and cerebral peduncle.

ANGIOGRAPHICAL FINDINGS

Table 1 shows our angiographical classification divided into three groups according to the ruptured artery, and further subdivided into subtypes according to the destruction of the internal capsule and ventricular perforation. Our classification introduces the idea of dynamic changes as the hematoma advances from localized to advanced type, and this classification can be applied to clinical practice.

The characteristic angiographical findings of each type are listed below.

Putaminal Hemorrhage

Localized Type (Fig. 4a). Hemorrhages arising from the most lateral branch of the lateral lenticulostriate artery progress through the watershed area between the distribution of the lateral lenticulostriate arteries and the subcortical branches of the insular artery, as we have previously reported. Hence, the lateral branch is deviated medially and the distance between the most lateral point of the lateral branch and the insular artery is always enlarged in the AP view (normal: 15 mm) (fig. 3a). When the hematoma does not extend to the posterior limb of the internal capsule and is localized in the above-mentioned area, the distal portion of the lateral lenticulostriate artery, which passes through the posterior limb, is not influenced by compression from the hematoma and remains almost in its original position. Consequently, the lateral branch is deviated medially, but its distal portion remains in its normal position and points laterally to the superior sagittal sinus (sign of sparing of the internal capsule). When seen in a lateral view, the lenticulostriate arteries exhibit a fanlike pattern extending almost through the entire anterior-posterior length of the internal capsule. Consequently, advancement along the anterior-posterior direction can be interpreted by displacement of these arteries and of the sylvian group from the middle cerebral artery. In the localized type, the anterior-posterior deviation of the lenticulostriate arteries is slight and their pattern is almost normal due to the small size of the hematoma.

The shift of the anterior cerebral artery and the anterior part of the internal cerebral vein is usually less than 5 mm due to the small size of the hematoma.

Advanced Type. Putamen–Internal Capsule (Fig. 4b). The lateral lenticulostriate artery is deviated medially including its distal portion, pointing to the superior sagittal sinus. This
finding indicates that the portion which passes through the posterior limb of the internal capsule is deviated medially. Hence, the hematoma is advancing to the posterior limb from the putamen and is influencing this portion by compression. When seen in the lateral view, the lenticulostriate arteries lose their normal fanlike pattern and deviate anteriorly or posteriorly according to the advancement of the hematoma.

Both the anterior cerebral artery and the internal cerebral vein are displaced in most cases.

**Putamen-Internal Capsule-Ventricle.** The lateral lenticulostriate artery is largely deviated medially, and it points in a direction between the superior sagittal sinus and the inferior margin of falx. In other cases, the lateral lenticulostriate artery is straightened and upright, and its normal “S” curve is lost or not visualized. It can be strongly suspected that the hematoma has perforated into the ventricle when the displacement of the internal cerebral vein is more than 6 mm and the displacement of the anterior cerebral artery is small. When the hematoma perforates into the ventricle and causes internal hydrocephalus, the angiogram shows unrolling of the pericallosal artery and straightening or poor filling of the subependymal veins.6

### Thalamic Hemorrhage

**Localized Type (Figs. 5a and b). Hemorrhage in Lateral Nuclei.** A hemorrhage arising from the thalamogeniculate artery never progresses into a watershed area as previously mentioned. Hence, the distance between the lateral lenticulostriate artery and the insular artery is normal or shortened (fig. 3b). When seen in a lateral view, the lenticulostriate arteries are deviated anteriorly.

The anterior cerebral artery is usually not deviated because of its location far anteriorly from the thalamus. The elevation of the sylvian point is very characteristic in the lateral view. Since the distance from the most lateral part of the lateral nuclei to the sylvian point is about 15 mm on the normal brain, the sylvian point is easily deviated by compression from the hematoma superiorly and laterally.

The midportion of the internal cerebral vein is usually deviated toward the opposite side due to this anatomical position.

**Hemorrhage in Medial Nuclei.** A small hematoma in the medial nuclei is very difficult to diagnose by angiography. Some cases had a slight deviation of the internal cerebral vein alone. Ventriculography will eventually establish the diagnosis.

### Advanced Type

**Whole Thalamic Hemorrhage (Figs. 5c and d).** The whole thalamic hemorrhage in which the hemorrhage advances from the lateral nuclei demonstrates the same characteristic angiographical findings as the hemorrhage in the lateral nuclei and, moreover, it shows hydrocephalus. However, the whole thalamic hemorrhage, which extends from the medial nuclei, shows hydrocephalic findings only. In such cases the definite diagnosis can be made by ventriculography.

### Subcortical Hemorrhage

Three subcortical hemorrhages were found among our 100 cases. We excluded those in which the microangioma was revealed by serial angiography or at operation.
FIGURE 5C Anteroposterior view of carotid arteriogram of thalamic hemorrhage (whole thalamic hemorrhage). The distance between the lenticulostriate artery and the insular artery is shortened (—). The anterior cerebral artery is generally in midline.

Discussion

The predominant theory concerning the pathogenesis of primary intracerebral hemorrhage is that of arterial rupture due to so-called angionecrosis or the microaneurysm resulting from it. These microaneurysms are usually found beneath the cerebral cortex, in the putamen and the thalamus, and rarely found in the internal capsule or globus pallidus.

In our autopsy series, large hematomas were found to be caused by rupture of the lateral lenticulostriate, thalamogeniculate and thalamoperforate arteries. This fact may be due to the proximity of the large-caliber high pressure arteries, such as the main trunk of the middle cerebral artery or the posterior cerebral artery as Russell has said.

As we have already reported, when a microaneurysm ruptures, bleeding continues and develops in the direction of least resistance as shown in figures 2a and b. The development of the hemorrhage then is dependent on both the place of origin and the direction in which the fascicles are separated. In catastrophic cases, the bleeding from the original ruptured artery seems to continue for a few hours and the acute angionecrosis may occur in the small vessels around a hematoma which was often observed histologically. Bleeding from these vessels may accelerate further formation of the larger hematoma. When the bleeding continues, it may cause ventricular rupture and tamponade. This promotes the intracranial hypertension which leads to secondary brain stem compression. In our opinion it is of clinical importance to stop the bleeding and remove the compressive mass lesion as soon as possible.

Certainly, in some cases of primary intracerebral hemorrhage, whose onset is catastrophic with immediate and maximal involvement of thalamus or hypothalamus, we cannot offer anything surgically. However, surgical intervention might save patients with hemorrhages due to rupture of the lateral lenticulostriate artery developing laterally and anteroposteriorly. Clinically they show a fairly slow onset with progressive signs of increased intracranial pressure and brain stem compression.

The prognosis of primary intracerebral hemorrhage varies according to the site and extension of the hemorrhage, the rapidity of the clot formation, and the direction of the hematoma advancement. Therefore, an early angiogram provides information which allows the neurosurgeon to make rational decisions regarding prognosis and therapy.

Study of the literature reveals many differences concerning the classification of hemorrhage within the cerebral hemisphere. The variations arise from the many authors using different criteria, various source materials and methods of study (e.g., pathological, clinical, radiological).

We have tried to clarify the main sites of intracerebral hemorrhage and the advancing direction of the hematoma by studying autopsy cases and angiograms. Furthermore, we
have tried to detect the presence of destruction of the internal capsule and ventricular rupture in patients with putaminal hemorrhage who might benefit from surgical intervention. We have concluded that an analysis of the direction of displacement of vessels, e.g., the main cerebral arteries, deep perforating arteries, especially the lenticulostrate artery, and deep cerebral veins, permits an accurate estimate of the location, extent and depth of the hematoma.

We are well aware that our angiographical classification will not always correspond with the autopsy findings in detail, especially the degree of destruction of the internal capsule or ventricular perforation. From a practical point of view, however, our classification has proved to be useful in 185 operated cases and will become the first step in the reappraisal of surgical treatment of primary intracerebral hemorrhage.

We will report the operative indications, techniques and results in a separate paper.

References

Effects of Ischemia on the Mg++ Requiring Adenosine Triphosphatase Associated With Neuronal Synaptic Vesicles in Gerbil Brain

ELIZABETH S. QUAYLE, M.D.,* SAMUEL T. CHRISTIAN, PH.D.,† AND JAMES H. HALSEY, JR., M.D.‡

SUMMARY The Mg++ requiring adenosine triphosphatase (ATPase) associated with neuronal synaptic vesicles is extremely vulnerable to ischemia. After five minutes of ischemia both the maximum velocity and the substrate binding capacity of the enzyme were decreased. Results also indicate that these changes are related to the rapid intraneuronal lactic acidosis accompanying ischemia. Ischemia was simulated by 37°C incubation of gerbil brain after decapitation. According to a recent hypothesis this enzyme plays a key role in exocytotic neurotransmitter release. Therefore, any inhibition of the ability of this enzyme to function would result in a drastically reduced capacity for transsynaptic impulse propagation.

Introduction

OF THE MANY EVENTS that occur at the synaptic junction, the method by which transmitter material is released in response to neuronal stimulation is one of the most important but least understood. Berl et al.1 have postulated that an actin-like protein (neurin), present in the axolemmal mem-

*Instructor in Neurology, †Associate Professor of Neurobiology in Psychiatry, Neurosciences Program, and ‡Professor and Chairman, Department of Neurology, the University of Alabama, Birmingham, Alabama 35294.

This work was supported by Grant #NS-08802 from the National Institute of Neurological Diseases and Stroke.
M Mizukami, H Kin, G Araki, H Mihara and Y Yoshida

Stroke. 1976;7:30-36
doi: 10.1161/01.STR.7.1.30

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/7/1/30

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/