Primary Non-Traumatic Intracranial Hemorrhage
A Municipal Emergency Hospital Viewpoint

P. Yarnell, M.D., and M. P. Earnest, M.D.

SUMMARY The devastating natural history of 138 consecutive admissions for non-traumatic intracranial hemorrhage to a major emergency care municipal hospital is reviewed. Sixty-four percent of the patients had demonstrable intracranial hematomas while 36% had mainly subarachnoid hemorrhage. Hypertension was a related condition in 43% of the parenchymal hematoma patients, while proved aneurysms accounted for 74% of the subarachnoid hemorrhage patients. There was only a 14% survivorship for patients requiring emergent surgery. All operated hematoma patients survived delayed surgery with improved level of responsiveness. The overall mortality was 74% for intracranial hematoma patients and 58% for aneurysm-caused subarachnoid hemorrhage patients.

The spectrum of conditions associated with primary non-traumatic intracranial hemorrhage patients often depends on the institution reporting them. These patients most frequently present as acute emergencies, and as such they are initially cared for by those hospitals with active emergency retrieval systems. During the 53-month period encompassed in this report, the Denver General Hospital was the major provider of its city's emergency ambulance service. When primary intracranial hemorrhage was suspected in an emergency patient, the neurology-neurosurgery service assumed responsibility for the diagnosis and therapy. This report details the devastating natural history of those patients with non-traumatic intracranial hemorrhage presenting for emergency admission.

Methods
From September 1971 through January 1976, 138 patients with intracranial hemorrhage were admitted as neurological emergencies. Of this group, 88 patients had primary non-traumatic intracranial hematomas while 50 had subarachnoid hemorrhage. One of the intracranial hematomas was an acute subdural from an aneurysm rupture, while all the rest were intraparenchymal hematomas. The related conditions in these patients are detailed in Table 1.

In the parenchymal hematoma category, excluding the congenital aneurysm-related hematomas, the patients were clustered in the 50-year to 69-year age group. There were 63 hemispheric hematomas, eight pontine hematomas, three cerebellar hematomas, and two primary intraventricular hemorrhages. Forty percent of the hemispheric hematomas were primarily localized in the basal ganglia.

In the congenital aneurysm cases, the peak age grouping was 40 to 49 years, and there was a 3:2 female-to-male ratio. The aneurysms were mainly supratentorial, divided in thirds between the anterior cerebral-anterior communicating complex (15 cases), the internal carotid-posterior communicating complex (15 cases), and the middle cerebral artery trifurcation (14 cases). There were two posterior fossa aneurysm cases and three patients with multiple aneurysms. The congenital aneurysm-related intracranial hematomas were all hemispheric except for one which was both infratentorial and supratentorial and one which was subdural.

The subarachnoid hemorrhage group (Table 1) included the 37 aneurysm patients without angiographically demonstrable intracranial hematomas, six arteriovenous malformations, divided equally between the supratentorial and infratentorial compartments, and seven unknown cases where a cause for the subarachnoid hemorrhage was not demonstrable either on repeated pan-cerebral angiography (five cases) or at postmortem (two cases).

Of the 88 patients with intracranial hematoma, 35 were operated on and 53 were treated medically. Of the 35 surgical patients, six were operated on after a delay of at least eight days. Table 2 details the outcome in terms of survival for the entire intracranial hematoma group. Table 3 details the patient profile and therapy data for the six cases of delayed surgery for hemispheric hematoma. Table 4 details the outcome for the congenital aneurysm subarachnoid hemorrhage patients. All six arteriovenous malformation subarachnoid hemorrhage patients survived, one of whom had elective surgery. Of the seven unknown category subarachnoid hemorrhage patients, two died and five did well.

Results and Discussion
During the 53 months of this study, 64% of 138 intracranial hemorrhage patients had demonstrable significant hematomas, while 36% had mainly subarachnoid bleeding. Of the intraparenchymal hematoma patients, 43% were related to hypertension, followed in frequency by congenital aneurysm rupture (13%), alcoholic bleeding diathesis (6%), complications of anticoagulation therapy (6%), mycotic aneurysmal rupture (3%), and arteriovenous malformations (3%). A large group of patients (21%) was found to have no known condition related to their intracranial hematoma. This high incidence of hypertension and alcoholism in our population and the relative paucity of primary hematological disease patients reflects the relative incidence of these conditions in the population served by our municipal hospital. Other series have shown a similar distribution of etiologic conditions, with hypertension invariably the most common association followed, in most, by aneurysms and vascular malformations. One of these studies had a relatively high percentage of cases (13%) with blood dyscrasias. This probably reflects the concentration of such patients in the large university medical center reporting that series. We can find no other series that comments on an association with alcoholism and bleeding which we saw in five cases. The bleeding defect in those cases was not easily explained. Frequently the prothrombin time and partial thromboplastin times were markedly prolonged, but...
platelet counts and fibrin split products were normal. Another category of unusual cause of intracranial hemorrhage in our series was acute bacterial endocarditis with rupture of a mycotic aneurysm. We had three of these cases, which are discussed in more detail elsewhere. Such cases are frequently diagnosed only retrospectively.

The subarachnoid hemorrhage (SAH) cases were caused by aneurysms in 74% and arteriovenous malformation in 12%, while 14% had undetermined cause despite exhaustive investigation. The distribution of causes is in approximate agreement with other series. Pakarinen, in a review of 589 cases of primary SAH, found aneurysms in 67.9%, arteriovenous malformations in 3.8% and no obvious lesion in 26.7%. However, his review of the literature on the findings in pan-cerebral vertebral angiography showed aneurysm as the cause in 53%, arteriovenous malformation in 7%, and negative studies in 40%. The Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage found aneurysms in 51%, hypertensive and/or arteriosclerotic vascular disease in 15%, arteriovenous malformation in 6% and unknown cause in 22%. The higher percentages of success in finding a cause in that study and ours compared with older literature may be due to improved radiological techniques.

Outcome was difficult to evaluate in conventional terms because of the extremely grave condition of many of our patients on presentation. Indeed, prior to the availability of highly organized city-wide ambulance systems, and prior to immediate neuroradiological diagnosis, many of the patients either would have been dead on arrival or would have died without an antemortem diagnosis. Tables 2 and 4 define outcome in the widest latitude in terms of survival and death. Many of the survivors had severe neurological residue as a complication of their illness.

Therapy included 21 early operations for primary parenchymal hematomas, seven emergent operations for congenital aneurysm-related parenchymal hematomas, and one operation for the aneurysm-related subdural hematoma. There was a combined survivorship of only four of these 29 patients (14%). This reflects the extremely grave condition of those patients requiring emergency intracranial clot removal and decompression. In the non-aneurysm parenchymal hematoma total group of 63 patients, there were six instances where surgery was delayed from 8 to 28 days

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**Table 1** Non-Traumatic Intracranial Hemorrhage in 138 Patients from September 1971 through January 1976

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Clinical course</th>
<th>Surgery day</th>
<th>Outcome</th>
<th>Associated conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>L thalamus, posterior temporal hemorrhage</td>
<td>R hemiplegia and aphasia defect, drowsiness</td>
<td>28</td>
<td>More awake, persistent deficit, nursing home</td>
<td>Diabetic, oral agents</td>
</tr>
<tr>
<td>R centrosylvian hemorrhage, 4 mm ICV shift initially, 10 mm ICV shift</td>
<td>Lethargic, dense R hemiplegia</td>
<td>26</td>
<td>More awake, persistent R hemiplegia; fatal pulmonary embolus at rehabilitation center</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>R centrosylvian hemorrhage, 6 mm ICV shift</td>
<td>Stabilized, dense L hemiplegia and ignoral of L body</td>
<td>11</td>
<td>Persistent L hemiplegia, moderately improved ignoral, wheelchair existence</td>
<td>Diabetic, oral agents; hypertensive</td>
</tr>
<tr>
<td>Large L retrosylvian parietal hemorrhage</td>
<td>Drowsiness with dense R hemiplegia and global aphasia</td>
<td>8</td>
<td>More awake, persistent R hemiplegia and aphasia; fatal pulmonary embolus at rehabilitation center</td>
<td>Hypertensive</td>
</tr>
<tr>
<td>Large R supra- and centrosylvian mass</td>
<td>Persistent drowsiness, moderate L hemiparesis and ignoral of L body</td>
<td>9</td>
<td>Awake, mild L paresis, wheelchair at home</td>
<td>Coumadin overdose, small vessel vasculitis, alcoholism</td>
</tr>
<tr>
<td>Large R centrosylvian mass, 10 mm ICV shift initially, 17 mm ICV shift Day 20</td>
<td>Somnolent, L hemiplegia and ignoral of L body</td>
<td>21</td>
<td>Awake, dense L hemiplegia, less L ignoral; went to rehabilitation center</td>
<td>Hypertensive</td>
</tr>
</tbody>
</table>

SA = Spanish American; B = Black; O = Oriental; C = Caucasian; L = left; R = right; M = male; ICV = internal cerebral vein.
TABLE 4

Aneurysmal Subarachnoid Hemorrhage Outcome in
36 Patients

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Died</th>
<th>Survived</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery</td>
<td>2</td>
<td>&gt; 58%</td>
</tr>
<tr>
<td>Medical</td>
<td>19</td>
<td>&gt; 42%</td>
</tr>
</tbody>
</table>

Table 4 shows the outcomes of 36 patients with aneurysmal subarachnoid hemorrhage treated with surgery and medical therapy. The table indicates that 11 patients died after surgery, with an survival rate of > 58%, and 19 patients survived, with a survival rate of > 42%.

Posthemorrhage (Table 3). These patients were operated on because of unsatisfactory neurological plateaus in the level of consciousness or focal deficit or both. They all became more alert and better rehabilitation candidates postoperatively; however, there was relatively little, if any, change in their focal deficits.

There were 11 survivors of 13 aneurysm elective surgery patients. The best outcomes were in those with relatively mild signs and symptoms as presentation of their subarachnoid hemorrhage.

Only one arteriovenous malformation subarachnoid hemorrhage was selected for elective malformation removal, and that patient did well. The two emergent arteriovenous malformation operations for parenchymal hematoma are included in the primary parenchymal hematoma fatalities. There was one other parenchymal arteriovenous malformation rupture in a patient who died shortly after angiographical diagnosis. Of the total 17 patients with intracranial arteriovenous malformations urgently admitted during this period, nine had intracranial hemorrhage: six were subarachnoid with a benign outcome, while three who died were intracerebral.

The seven patients with undetermined source of subarachnoid hemorrhage were divided between the two presenting in extremis, ending fatally, and the five presenting with mild signs, leaving the hospital completely intact.

Intracranial hemorrhage is a devastating event with a 74% mortality in our intracranial hematoma group and a 58% mortality in the aneurysm-related subarachnoid hemorrhage. These are our two major categories of non-traumatic intracranial hemorrhage. The high mortality of intraparenchymal hemorrhage, regardless of etiology (i.e., hypertension, aneurysm, etc.), is most forbidding. It has become an important guide for our decisions concerning therapy. In those patients presenting in extremis but who are otherwise physiologically intact, and who have accessible clots, we frequently recommend early surgery. However, we realize the futility of this effort in most instances. In those patients in stable condition we favor aggressive medical management hoping for either improvement or stabilization for delayed surgery. We recommend late surgery, i.e., beyond one week, when the patient stabilizes with a poor level of consciousness. In this way we hope to improve rehabilitation potential. Operating on patients who are alert but with marked focal deficits has not been helpful in our experience. A similar approach has been favored by others.

With the high mortality and morbidity of intraparenchymal hemorrhage and the known association with hypertension, the most effective therapy is prevention. Active treatment of hypertension has been shown to reduce the occurrence of intraparenchymal hemorrhage in a large population. Therefore, detection and correction of hypertension must be the primary therapy for this disease. Treatment of hypertension possibly might also reduce the occurrence of congenital aneurysm rupture, but there are no data available concerning this.

References

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