Initial and Recurrent Bleeding Are the Major Causes of Death Following Subarachnoid Hemorrhage

Joseph P. Broderick, MD; Thomas G. Brott, MD; John E. Duldner, MD; Thomas Tomsick, MD; Alan Leach, MS

Background and Purpose The goal of this study was to determine the causes of mortality and morbidity after subarachnoid hemorrhage.

Methods We identified all first-ever spontaneous subarachnoid hemorrhages that occurred in the nearly 1.3 million population of greater Cincinnati during 1988.

Results Thirty-day mortality for subarachnoid hemorrhage was 45% (36 of 80 cases). Of the 36 deaths, 22 (61%) died within 2 days of onset; 21 of these deaths were due to the initial hemorrhage, and one death was due to rebleeding documented by computed tomography. Nineteen of the remaining 14 deaths after day 2 were caused by the initial hemorrhage (2 cases) or rebleeding (7 cases). Volume of subarachnoid hemorrhage was a powerful predictor of 30-day mortality (P=.0001). Only 3 of the 29 patients with a volume of subarachnoid hemorrhage of 15 cm³ or less died before 30 days. Two of these 3 patients died from documented rebleeding; the third had 87 cm³ of additional intraventricular hemorrhage. Delayed arterial vasospasm contributed to only 2 of all 36 deaths.

Conclusions Most deaths after subarachnoid hemorrhage occur very rapidly and are due to the initial hemorrhage. Rebleeding is the most important preventable cause of death in hospitalized patients. In a large representative metropolitan population, delayed arterial vasospasm plays a very minor role in mortality caused by subarachnoid hemorrhage. (Stroke. 1994;25:1342-1347.)

Key Words subarachnoid hemorrhage • mortality

Surgical and medical treatment studies of subarachnoid hemorrhage have reported that vasospasm and rebleeding are the major causes of 30-day mortality and morbidity after subarachnoid hemorrhage.1,2 These treatment studies almost always exclude the most moribund patients and patients who die before reaching the hospital. Yet, only studies that examine the outcome of all patients in a large, well-defined population can accurately determine the most important causes of early death and morbidity after subarachnoid hemorrhage. This information is necessary to determine the most effective public health strategies for decreasing the morbidity and mortality associated with this deadly disease.

The present study examines the causes of morbidity and mortality associated with all cases of subarachnoid hemorrhage that occurred in the nearly 1.3 million population of greater Cincinnati during 1988.

Subjects and Methods

All first-ever spontaneous subarachnoid hemorrhages that occurred in greater Cincinnati during 1988 were identified as previously reported.6 Inclusion in the study required that at the onset of hemorrhage the patient resided in the five-county region, as determined by zip code, that the onset of the subarachnoid hemorrhage occurred during 1988, and that the patient met the criteria for subarachnoid hemorrhage. Subarachnoid hemorrhage was defined as blood in the subarachnoid spaces, not caused by trauma, detected by computed tomography (CT) scanning or at autopsy, or as a clinical history and examination consistent with subarachnoid hemorrhage (sudden onset of severe headache or change in level of consciousness) with xanthochromia and many red cells in the cerebrospinal fluid.

The medical record systems reviewed included those of all 20 acute care hospitals and five coroners' offices in the five-county region. Three of these hospitals can be characterized as referral centers for aneurysmal subarachnoid hemorrhage, 15 are community hospitals, 1 is a Veterans Hospital, and 1 is a tertiary-referral children's hospital. Patients with subarachnoid hemorrhage who were referred from outside of the five-county region were excluded.

The abstracted clinical data and all available CT and magnetic resonance imaging films for each case were evaluated by a neurologist. Selected films were also evaluated by a neuroradiologist. We also reviewed case report forms of patients treated as part of experimental studies of nimodipine and nicardipine for subarachnoid hemorrhage in greater Cincinnati hospitals during 1988. Two additional cases of subarachnoid hemorrhage that occurred during 1988 were identified. One patient's diagnosis had been coded improperly by medical record personnel during 1988. The second patient's chart was the only chart with a discharge diagnosis of subarachnoid hemorrhage that could not be located on the initial screening of medical records at the 20 hospitals.

Volume of subarachnoid hemorrhage was measured from the original CT films in the following manner. First, each CT image was placed on a light box, above which was fixed a video camera connected to a Joyce-Loebl Magiscan M2A Image Analysis computer. After obtaining an appropriate degree of clarity and brightness, an individual image was captured by the
camera, digitalized, and reproduced on the video monitor. Regions of subarachnoid hemorrhage were identified by a neurologist, and the borders of the regions were then roughly approximated onto the screen using a light pen. The circumscribed area then was segmented according to a gray scale ranging from 0 (black) to 100 (white). Upper and lower limits of the scale were set manually for each image, ensuring that only the area of subarachnoid hemorrhage was highlighted and that the low-density brain tissue and other tissues surrounding the hemorrhage were excluded. The number of pixels constituting the subarachnoid hemorrhage was determined using a linear centimeter scale on each CT image, a calibration square was constructed, and the number of pixels within a calibration square was used to determine the calibration factor (pixels per square centimeter). The number of pixels of hemorrhage in an individual CT slice was then divided by the calibration factor to obtain real surface area measurements in square centimeters. The surface area was multiplied by the image slice thickness (1 cm) to obtain a slice volume. Slice volumes were added to obtain the total volume of subarachnoid hemorrhage. The same procedure was also used to calculate separately the total volume of intraventricular hemorrhage and intraparenchymal hemorrhage. The volume of subarachnoid hemorrhage as determined by this method depends on the volume of existing cerebrospinal fluid spaces at the time of hemorrhage, since blood diffuses into adjacent subarachnoid fluid spaces. Thus, for a given volume of bleeding, persons with enlarged subarachnoid fluid spaces will tend to have a larger measured volume of subarachnoid hemorrhage than those patients with smaller subarachnoid spaces.

The amount of subarachnoid blood was also estimated directly from the CT film using the classification method of Fisher and colleagues: grade 1, no blood detected; grade 2, diffuse deposition or thin layer of blood with all vertical layers of blood less than 1 mm thick; and grade 3, localized clots of blood and/or vertical layers of blood of 1-mm thickness or greater. The amount of blood in the ventricular system was categorized as per Petrak and colleagues: grade 0, no intraventricular hemorrhage; grade 1, small amount of blood layering in the occipital horns; grade 2, blood occupying one full lateral ventricle with or without blood in the third or fourth ventricles; and grade 3, major intraventricular hemorrhage with blood packed into all ventricles and possible distension of the ventricular system.

Neurological function at presentation was measured by the World Federation of Neurological Surgeons Scale: grade 1, Glasgow Coma Scale score of 15 with no headache or focal signs; grade 2, Glasgow Coma Scale score of 15 with headache and nuchal rigidity but no focal signs; grade 3, Glasgow Coma Scale score of 13 to 14 with headache and/or nuchal rigidity but no focal signs; grade 4a, Glasgow Coma Scale score of 13 to 14 with headache, nuchal rigidity, or focal signs; grade 4b, Glasgow Coma Scale score of 9 to 12 with headache, nuchal rigidity, or focal signs; and grade 5, Glasgow Coma Scale score of 8 or less and can have headache, nuchal rigidity, or focal signs. The Glasgow Coma Scale score, which is the main basis for the World Federation of Neurological Surgeons Scale, was often recorded on the life squad or emergency department record. For those cases in which a specific Glasgow Coma Scale score was not recorded in the medical record, the score was estimated using the records of the neurological examination made by the physician and nurses in the emergency department.

Other data abstracted from the medical record for this study included age, sex, race; admission blood pressure, pulse, and respiration; date and time of stroke onset; the presence of vasospasm and the location of aneurysms as determined by cerebral angiography; hydrocephalus on CT; hyponatremia (serum sodium less than 130 mEq/mL); operative clipping of ruptured aneurysm and time from symptom onset to operation; placement of an intraventricular drain; intubation; and antihypertensive therapy. For the 10 patients in whom the date but not the time of stroke onset could be accurately determined from the medical record, we assigned noon as the time of onset unless a patient arrived at the hospital before that time.

Rebleeding was defined as acute clinical deterioration that was accompanied by evidence of rebleeding in the subarachnoid space, ventricular system, or brain parenchyma by follow-up CT or autopsy. Increased seepage of blood into the ventricular system on subsequent CT scans was not included as recurrent bleeding. The diagnosis of clinical vasospasm was made using the following criteria: (1) classic symptoms of vasospasm that included onset 3 to 12 days after subarachnoid hemorrhage, worsening of headache, stiff neck, or low-grade fever, insidious onset of confusion, disorientation, or drowsiness, and new focal deficits that often fluctuated; (2) negative CT findings to exclude rebleeding and hydrocephalus; (3) no other identifiable cause of neurological worsening; and (4) confirmatory evidence on cerebral angiography when available.

Clinical outcome was graded from the medical records using a modified Oxford Handicap Scale as follows: 0, no symptoms; 1, minor symptoms that do not interfere with lifestyle; 2, minor handicap with symptoms that lead to some restriction in lifestyle but do not interfere with the patient's capacity to look after himself; 3, moderate handicap with symptoms that significantly restrict lifestyle and prevent totally independent existence; 4, moderately severe handicap with symptoms that clearly prevent independent existence though not needing constant attention; 5, severe handicap with totally dependent patient requiring constant attention night and day; and 6, death.

Kaplan-Meier 30-day survival curves were calculated. Survival among men and women as well as whites and blacks was compared by log-rank test. Using 30-day mortality as the dependent variable, univariate logistic regression analysis was performed on the variables that were available at the initial medical evaluation as follows: age, race, sex, initial systolic blood pressure, volume of subarachnoid hemorrhage as measured by image analysis, volume of intraventricular hemorrhage, volume of parenchymal hemorrhage, and World Federation of Neurological Surgeons Scale score. Multivariate logistic regression analyses were then performed (including forward, backward, and stepwise modeling procedures) to determine the model that best predicted outcome at 30 days.

One multivariate analysis used death at 30 days as the dependent variable; another analysis used death or total dependency (Oxford Handicap Scale score of 5) at 30 days as the dependent variable. Comparisons between operated and nonoperated patients were made by $\chi^2$ and unpaired $t$ tests. Values of $P<.05$ (two-tailed) were considered significant. Data are presented as mean±standard deviations except as indicated.

**Results**

There were 82 cases of spontaneous subarachnoid hemorrhage during 1988. Sixty-nine cases of subarachnoid hemorrhage were identified by CT, 11 by autopsy, and 2 by clinical evaluation and lumbar puncture. Of 71 hospitalized patients, 27 were admitted to the three tertiary-referral hospitals, and 44 patients were admitted to the remaining community hospitals.

Two causes of the 82 cases of subarachnoid hemorrhage were verifiable: ruptured aneurysm, as documented by angiography (34 patients) at autopsy (11 patients) or during the operative removal of an associated parenchymal hemorrhage (2 patients), and arteriovenous malformation (2 patients, both cases documented at autopsy). Of the remaining 33 hospitalized patients with subarachnoid hemorrhage, 11 had angio-
TABLE 1. Cause and Timing of Deaths After Onset of Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Category</th>
<th>No. of Cases</th>
<th>No. of Deaths</th>
<th>Initial Bleed</th>
<th>Rebleed</th>
<th>Vasospasm</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proven aneurysm</td>
<td>47</td>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospitalized</td>
<td>38</td>
<td>7</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Operation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 1-3</td>
<td>7</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 4-7</td>
<td>5</td>
<td>0</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 8 or more</td>
<td>19</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No operation</td>
<td>7</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonhospitalized/ autopsy cases</td>
<td>9</td>
<td>9</td>
<td>D-1,1,1,1,1,1,1,1,1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Probable aneurysm (no angiogram or autopsy)</td>
<td>20</td>
<td>18</td>
<td>D-1,1,1,1,1,1,1,1,1,2,1,2,4,5</td>
<td>D-5,11</td>
<td>D-8,14</td>
<td>D-16,24</td>
</tr>
<tr>
<td>No bleeding source</td>
<td>13</td>
<td>2</td>
<td>D-1,1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>80</td>
<td>36</td>
<td>23</td>
<td>8</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

grams that showed no source of the subarachnoid hemorrhage. Two other patients had an autopsy that indicated no clear source of bleeding. The poor clinical condition of the rest precluded cerebral angiography. The median time from onset of subarachnoid hemorrhage to first medical evaluation was 50 minutes. The 2 cases of subarachnoid hemorrhage due to rupture of an arteriovenous malformation are excluded from the remaining analyses.

The mean age of the 80 patients with subarachnoid hemorrhage thought due to aneurysmal rupture was 54 ± 13 years (31 men, 49 women; 61 white, 19 black). Of the 80 patients, 36 (45%) died within 30 days of onset. The 30-day mortality of patients admitted to one of the three referral hospitals (33%) was not significantly lower than the mortality of patients admitted to the remaining hospitals (41%). However, as noted in “Methods,” patients with subarachnoid hemorrhage that were referred from outside of the five-county region were excluded from analysis. These excluded patients would be expected to have a higher survival rate. The 67 patients with a documented or probable aneurysm had a 30-day mortality of 51% (Table 1).

13 patients who had no evidence of a ruptured aneurysm by angiogram or autopsy had a 30-day mortality of 15% (Table 1).

Twenty-two deaths (61%) occurred within the first 2 days of onset, and 9 of 21 patients died before reaching the hospital (Figs 1 and 2, Table 1). One of the 22 very early deaths was caused by CT-documented rebleeding. Various causes of death in the remaining 14 patients who died after day 2 included rebleeding (7 patients), mass effect and herniation from a large intracerebral hematoma accompanying the subarachnoid hemorrhage (1), the initial subarachnoid hemorrhage (1, day 4), myocardial infarction (1), pneumonia (1), cardiac arrhythmia (1), hydrocephalus and clinical evidence of vasospasm (1), and brain damage secondary to cardiac arrest on day 1 and subsequent hydrocephalus, hypotension, and possible vasospasm (1).

Original CT films that were used to measure the volume of subarachnoid, intraventricular, and intraparenchymal hemorrhage were available for 59 of the 71 hospitalized patients. The mean volume of subarachnoid hemorrhage for the 59 patients was 21 ± 22 cm³. Volume of subarachnoid hemorrhage was a powerful predictor of subsequent 30-day mortality in a univariate logistic regression analysis ($P = .0001$). Only 3 of the 29 patients with a volume of subarachnoid hemorrhage of 15 cm³ or less died before 30 days (Fig 3). Two of these 3 patients had documented rebleeding as the cause of death, and the third had 87 cm³ of intraventricular hemorrhage in addition to 6 cm³ of subarachnoid hem-

![Number of Deaths](Fig 1) Bar graph showing timing of deaths after onset of subarachnoid hemorrhage. Day of death (x axis) is in calendar days from stroke onset. Thus, a patient who had onset of subarachnoid hemorrhage on 3/4/88 and died on 3/5/88 was classified as dying on calendar day 1. Patients who died on calendar day 0 are included under calendar day 1 in the graph.

![Proportional graphic of causes of death among 80 patients with subarachnoid hemorrhage](Fig 2)
Bleeding After Subarachnoid Hemorrhage

Fig 3. Scatterplot showing relation of age and volume of subarachnoid hemorrhage (SAH) to 30-day mortality (59 cases with computed tomographic measurements).

orrhage. Only 1 of the 8 patients with a volume of subarachnoid hemorrhage of 50 cm³ or more survived. Using Fisher's system of grading the amount of subarachnoid hemorrhage on the CT film, a similar relation between the amount of subarachnoid hemorrhage and mortality was seen. None of the 17 patients with grade 1 or grade 2 subarachnoid hemorrhage were dead at 30 days, whereas 24 (57%) of the 42 patients with a grade 3 hemorrhage died ($P=.001$, $\chi^2$ test). Intraventricular hemorrhage was present on the initial CT scan in 21 (36%) of 59 patients (mean volume for the 21 patients, 30±36 cm³) and was a significant predictor of 30-day mortality in the univariate analysis ($P=.008$). Classification of intraventricular hemorrhage by the Petruk Scale also significantly predicted 30-day mortality ($P=.01$, $\chi^2$ test). Intraparenchymal hemorrhage was present on the initial CT scan in 8 (14%) of the 59 patients (mean volume for the 8 patients, 27±26 cm³) but was not predictive of 30-day mortality.

The World Federation of Neurological Surgeons Scale score ($P=.0001$) and age ($P=.005$) were other significant predictors of 30-day mortality in the univariate analyses. Age ($r=.48$, $P=.0001$) and the World Federation of Neurological Surgeons Scale score ($r=.55$, $P=.0001$) were also significantly correlated with the volume of subarachnoid hemorrhage.

Only volume of subarachnoid hemorrhage ($P=.01$) and the World Federation of Neurological Surgeons Scale score ($P=.006$) remained significant independent predictors of 30-day mortality in the multivariate logistic regression analyses. In the analysis using death or total dependence at 30 days as the outcome variable, only age ($P=.008$) and the World Federation of Neurological Surgeons Scale score ($P=.0001$) were significant predictors of outcome.

Of the later events during hospitalization, rebleeding ($\chi^2$, $P<.001$) and operative clipping of aneurysm ($\chi^2$, $P<.001$) were significant predictors of 30-day outcome. All of the eight patients who had rebleeding died. Rebleeding occurred on day 1 (1), day 2 (2), day 5 (1), day 12 (1), day 16 (1), day 22 (1), and day 27 (1). Only 1 (3%) of the 31 patients who underwent operative clipping of a ruptured aneurysm died before 30 days. This patient with multiple aneurysms had rupture of a partially clipped ruptured aneurysm and died from rebleeding 27 days after the initial hemorrhage.

Of the 31 operated patients, 7 had clipping of the aneurysm within the first 72 hours after onset, whereas the remainder had later operations. Patients who underwent operative clipping of an aneurysm were much younger, had smaller volumes of subarachnoid hemorrhage and intraventricular hemorrhage, and had much better World Federation of Neurological Surgeons Scale scores on initial evaluation than did patients who did not undergo operative clipping (Table 2). The overall outcome of operated patients, as measured by the Modified Oxford Handicap Scale, was significantly better than for nonoperated patients ($\chi^2$ test, $P=.001$, Fig 4).

Clinical vasospasm was not a significant predictor of mortality. Although 31 (44%) of the 71 hospitalized patients had neurological deterioration after day 2 of their hospital course that was possibly due to vasospasm, clinical vasospasm played a role in the death of only 2 patients. Only 3 patients with clinical vasospasm had documented ischemic infarcts by subsequent CT scans that were associated with clinical neurological deficits. Most of the patients with clinical vasospasm who survived did not have subsequent CT scans, so the actual incidence of cerebral infarction due to suspected vasospasm could not be determined.

Documented aneurysms involved the anterior cerebral artery complex in 25% of cases, the carotid artery

TABLE 2. Comparison of Hospitalized Patients Who Underwent Aneurysm Clipping (n=31) and Those Who Did Not (n=40)

<table>
<thead>
<tr>
<th></th>
<th>Operated Patients</th>
<th>Nonoperated Patients</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>51±13</td>
<td>59±19</td>
<td>.03</td>
</tr>
<tr>
<td>Volume of SAH*, cm³</td>
<td>10±13</td>
<td>28±24</td>
<td>.0006</td>
</tr>
<tr>
<td>Volume of IVH*, cm³</td>
<td>4±11</td>
<td>14±31</td>
<td>.07</td>
</tr>
<tr>
<td>Volume of ICH†, cm³</td>
<td>28±41</td>
<td>27±10</td>
<td>.98</td>
</tr>
<tr>
<td>WFNS§</td>
<td>2.9±1.5</td>
<td>4.2±1.9</td>
<td>.003</td>
</tr>
<tr>
<td>30-Day mortality</td>
<td>4%</td>
<td>63%</td>
<td>.0001</td>
</tr>
</tbody>
</table>

SAH indicates subarachnoid hemorrhage; IVH, intraventricular hemorrhage; ICH, intracerebral hemorrhage; and WFNS, World Federation of Neurological Surgeons Scale score.

*Mean volumes include only those 24 operated and 35 nonoperated patients with computed tomographic scan measurements.
†Mean volumes include only operated patients (n=4) and nonoperated patients (n=4) with an intraparenchymal hemorrhage.
The results suggest that there is a critical volume of subarachnoid hemorrhage below which survival is highly likely unless rebleeding occurs. None of the 29 patients with a volume of subarachnoid hemorrhage of less than 15 cm³ died except for 2 patients who had documented rebleeding and 1 patient who also had 87 cm³ of ventricular hemorrhage.

Older age was an independent predictor of poor outcome at 30 days as in other population-based outcome studies.15,16 Enlarged cerebrospinal fluid spaces due to brain atrophy, which is often seen in the elderly, is the likely explanation for the significant correlation between advancing age and the volume of subarachnoid hemorrhage in our study. However, volume of subarachnoid hemorrhage remained a significant independent predictor of 30-day mortality after adjusting for age.

Rebleeding was the second leading cause of death and, except for one case, occurred after the first day. Thus, it is the major preventable cause of death in patients hospitalized with subarachnoid hemorrhage. In addition, our 11% rate of rebleeding during the first 30 days, which is lower than rates in other studies, is likely an underestimate because of the strictness of our criteria. The importance of rebleeding as a cause of 30-day mortality adds to the rationale for early clipping of ruptured aneurysms. The Cooperative Aneurysm Study reported that early surgery does reduce the risk of rebleeding in patients with ruptured aneurysms. However, overall outcome in patients with early and later clipping of aneurysms was not significantly different.

In conclusion, studies that exclude patients who are moribund at admission grossly underestimate the importance of the initial hemorrhage and overestimate the importance of vasospasm as a cause of death and morbidity after subarachnoid hemorrhage in large populations. For the US population, approximately 15,000 cases of subarachnoid hemorrhage occur yearly. Our results suggest that approximately 6000 patients will die suddenly, from either the initial bleeding or rebleeding, but only 500 or so from the effect of delayed cerebral vasospasm. The results provide further support for prevention as the most effective strategy for lowering mortality from subarachnoid hemorrhage.
Acknowledgments

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