Volume of Intracerebral Hemorrhage

A Powerful and Easy-to-Use Predictor of 30-Day Mortality

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Background and Purpose: **The aim of this study was to determine the 30-day mortality and morbidity of intracerebral hemorrhage in a large metropolitan population and to determine the most important predictors of 30-day outcome.**

Methods: **We reviewed the medical records and computed tomographic films for all cases of spontaneous intracerebral hemorrhage in Greater Cincinnati during 1988. Independent predictors of 30-day mortality were determined using univariate and multivariate statistical analyses.**

Results: **The 30-day mortality for the 188 cases of intracerebral hemorrhage was 44%, with half of deaths occurring within the first 2 days of onset. Volume of intracerebral hemorrhage was the strongest predictor of 30-day mortality for all locations of intracerebral hemorrhage. Using three categories of parenchymal hemorrhage volume (0 to 29 cm^3, 30 to 60 cm^3, and 61 cm^3 or more), calculated by a quick and easy-to-use ellipsoid method, and two categories of the Glasgow Coma Scale (9 or more and 8 or less), 30-day mortality was predicted correctly with a sensitivity of 96% and a specificity of 98%. Patients with a parenchymal hemorrhage volume of 60 cm^3 or more on their initial computed tomogram and a Glasgow Coma Scale score of 8 or less had a predicted 30-day mortality of 91%. Patients with a volume of less than 30 cm^3 and a Glasgow Coma Scale score of 9 or more had a predicted 30-day mortality of 19%. Only one of the 71 patients with a volume of parenchymal hemorrhage of 30 cm^3 or more could function independently at 30 days.**

Conclusions: **Volume of intracerebral hemorrhage, in combination with the initial Glasgow Coma Scale score, is a powerful and easy-to-use predictor of 30-day mortality and morbidity in patients with spontaneous intracerebral hemorrhage. (*Stroke* 1993;24:987-993)**

KEY WORDS • **intracerebral hemorrhage** • **survival** • **tomography**

Intracerebral hemorrhage has a reported 30-day mortality of 44% to 51% in population studies during the computed tomographic (CT) era.[1-5] Level of consciousness, volume of parenchymal hemorrhage, and, to a lesser extent, volume of intraventricular hemorrhage have been most consistently linked with poor outcome.[5-18] However, a usable predictive model of outcome incorporating these variables has yet to be validated in a large, well-defined community population.

We report a study of the natural history of intracerebral hemorrhage in the 1.26 million metropolitan population of Greater Cincinnati. Our goals were to determine the most important predictors of morbidity and mortality and to develop an easy-to-use predictive model of 30-day mortality for physicians and researchers involved in the treatment of intracerebral hemorrhage.

Subjects and Methods

All first-ever spontaneous intracerebral hemorrhages that occurred in Greater Cincinnati during 1988 were identified as previously reported.[1] Traumatic intracerebral hemorrhage, hemorrhage due to aneurysmal rupture, and hemorrhagic transformation of a cerebral infarct were excluded. The abstracted clinical data and all available CT and magnetic resonance imaging films for each case were evaluated by a neurologist. Each brain location that contained hemorrhage on CT was recorded (putamen, globus pallidus, thalamus, internal capsule, deep periventricular white matter, cerebral cortex, subcortical white matter, cerebellum, pons, midbrain, and ventricles). The origin of each intracerebral hemorrhage was categorized as deep (basal ganglia, thalamus, internal capsule, deep periventricular white matter, ventricles only); lobar (cortex and subcortical white matter); cerebellar; or pontine. For a few larger hemorrhages, the distinction between hemorrhages that began in the cortex or subcortical white matter (lobar) and those that originated in the periventricular white matter (deep) was difficult. Deep white matter hemorrhages must have had hemorrhage within 1 cm of the body of the lateral ventricle and the majority of the hemorrhage within the deep white matter.

Volume of hemorrhage was measured by two independent methods. 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

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monitor. The region of hemorrhage was identified, and its borders were roughly approximated on the screen using a light pen. The circumscribed area was then 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 hemorrhage was highlighted and that the low-density brain surrounding the hemorrhage was excluded. The number of pixels constituting the area of hemorrhage was determined. Using the 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 parenchymal hemorrhage. The same procedure was also used to calculate separately the total volume of intraventricular hemorrhage.

The second method used to estimate the volume of parenchymal hemorrhage used only the CT films. On the CT image with the largest area of intraparenchymal hemorrhage, the largest diameter of hemorrhage was measured by the study neurologist using the centimeter scale on the film, and then rounded to the nearest half centimeter. The diameter of hemorrhage 90 degrees to the largest diameter was also recorded and rounded to the nearest half centimeter. The number of 1-cm slices on which parenchymal hemorrhage could be seen was recorded. The total volume of parenchymal hemorrhage was then estimated using the formula for an ellipsoid ($4/3\ \pi\ abc$, where a, b, and c represent the respective radii in three dimensions; Fig 1).

Neurological function at presentation was measured by Glasgow Coma Scale,[19] which 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 recorded neurological examination of the physician and nurses in the emergency department. Other data abstracted from the medical record included age, sex, and race; admission blood pressure, pulse, and respiration; estimated time from stroke onset to first medical contact; history of hypertension, diabetes, and prior ischemic stroke; current warfarin and aspirin use; and operative removal of intracerebral hemorrhage. For the 16 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.

Clinical outcome was graded from the medical records using a modified Oxford Handicap Scale,[20] in which 0=no symptoms; 1=minor symptoms that do not interfere with life-style; 2=minor handicap, symptoms that lead to some restriction in life-style but do not interfere with the patient's capacity to look after himself; 3=moderate handicap, symptoms that significantly restrict life-style and prevent totally independent existence; 4=moderately severe handicap, symptoms that clearly prevent independent existence although not needing constant attention; 5=severe handicap, totally dependent patient requiring constant attention night and day; and 6=dead.

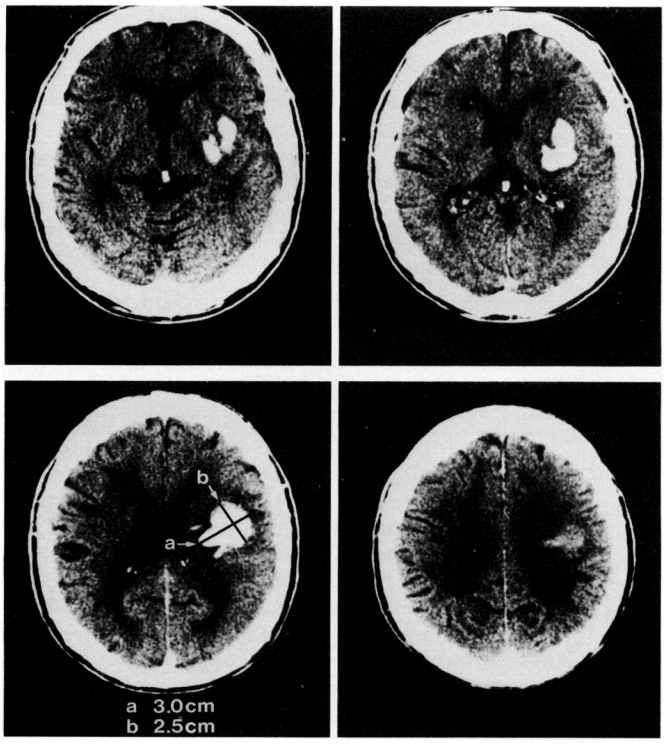

FIG 1. *Computed tomographic images illustrate calculation of ellipsoid volume ($4/3\ \pi$ abc, where* a, b, *and* c *represent the respective radii in three dimensions). In this patient, the largest diameter of hemorrhage was 3.0 cm^3, and the diameter at 90 degrees to this measurement was 2.5 cm^3. The parenchymal hemorrhage is seen clearly on three 1-cm slices and barely seen on a fourth slice (vertical diameter, 3.5 cm^3). Thus, the calculated ellipsoid volume is $4/3\ \pi\ (0.5)(3)(0.5)(2.5)(0.5)(3.5)=13.9$ cm^3. This estimated volume is close to the actual hemorrhage volume of 14.4 cm^3 as measured by computerized image analysis (see "Subjects and Methods").*

Kaplan-Meier 30-day survival curves[21] 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 following independent variables: age, race, sex, initial systolic blood pressure, volume of intracerebral hemorrhage as measured by image analysis, volume of intraventricular hemorrhage, Glasgow Coma Scale, location of hemorrhage, and operation (0=no, 1=yes). All variables in the univariate analysis were included in a stepwise multivariate logistic regression analysis.[22] Values of $P \leq .05$ (two-tailed) were considered significant.

Results

There were 188 cases of spontaneous intracerebral hemorrhage. Only one patient was lost to follow-up after discharge from the hospital. The 30-day mortality for patients with intracerebral hemorrhage was 44%, with half of the deaths occurring within the first 2 days (Fig 2).

For the remaining analyses, we excluded the 1 patient lost to follow-up, the 2 patients identified by autopsy

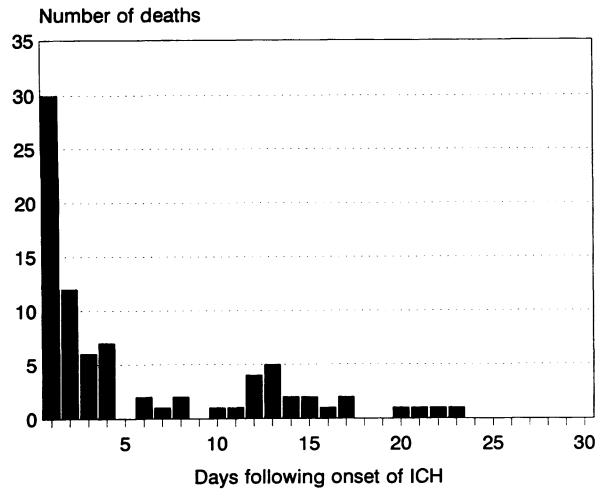

FIG 2. *Bar graph shows timing of deaths after onset of intracerebral hemorrhage (ICH).*

alone, 5 patients with intraventricular hemorrhage only, and 18 patients in whom the original CT film was unavailable for measurement of hemorrhage volume. For the remaining 162 hospitalized patients, the median time from onset of symptoms to first medical contact was 1.2 hours. The 30-day mortality was similar for men (48%), women (41%), whites (44%), and blacks (42%). The 30-day mortality for lobar hemorrhage (39%) was slightly less than for deep (45%), pontine (44%), and cerebellar (64%) hemorrhage.

Volume of parenchymal hemorrhage, as calculated by computerized image analysis, was the most important predictor of 30-day survival for all ages and locations of hemorrhage (Figs 3 through 6). For intracerebral hemorrhages with a volume of more than 60 cm^3, the 30-day mortality for deep hemorrhages was 93% and for lobar hemorrhages was 71%. For hemorrhage volumes of 30 to 60 cm^3, the 30-day mortality was 64% for deep hemorrhages, 60% for lobar hemorrhages, and 75% for cerebellar hemorrhages. For hemorrhages with a volume of less than 30 cm^3, the 30-day mortality was 23% for deep hemorrhages, 7% for lobar hemorrhages, and 57% for cerebellar hemorrhages. All 5 patients with a pontine hemorrhage who survived 30 days had a hemorrhage volume of less than 5 cm^3. Only 1 of the 71 patients with a volume of parenchymal hemorrhage of 30 cm^3 or more could function independently at 30 days (Oxford Handicap Score of 3 or less; Fig 7). Only 16 (18%) of the 91 patients with a hemorrhage volume of less than 30 cm^3 were totally independent at 30 days (Oxford Handicap Score of 2 or less).

There were 11 patients with deep hemorrhages, 4 with cerebellar, 3 with pontine, and 2 patients with lobar hemorrhages with an initial parenchymal hemorrhage volume of less than 30 cm^3 who were dead by 30 days. Of the 11 patients with deep hemorrhages, 9 had substantial intraventricular hemorrhage, and 7 of the 9 were thalamic in origin. The remaining 2 patients had a substantial increase in the volume of hemorrhage on subsequent CT scans. The 2 patients with a lobar hemorrhage volume of less than 30 cm^3 had a marked increase in the volume of hemorrhage on subsequent CT studies. One of the 2 had been taking warfarin; the other had received streptokinase and heparin for a pulmonary embolus.

In the univariate logistic regression analyses, volume of intracerebral hemorrhage ($P<.0001$), volume of intraventricular hemorrhage ($P<.0001$), and initial Glasgow Coma Scale score ($P<.0001$) were significant predictors of 30-day mortality, whereas age, sex, race, systolic blood pressure, and location of hemorrhage were not. Operative removal of hemorrhage was of borderline significance as a predictor of 30-day mortality ($P=.055$). In a multivariate logistic regression model, volume of intracerebral hemorrhage, volume of intraventricular hemorrhage, Glasgow Coma Scale score, and operative removal of hemorrhage were significant independent predictors of 30-day mortality (Table 1). In this model, larger volumes of intracerebral hemorrhage and intraventricular hemorrhage as well as lower Glasgow Coma Scale scores were associated with increased mortality, whereas operative removal was associated with decreased mortality. When the multivariate logistic regression analysis was limited to only the 142 deep and lobar hemorrhages, only volume of parenchy-

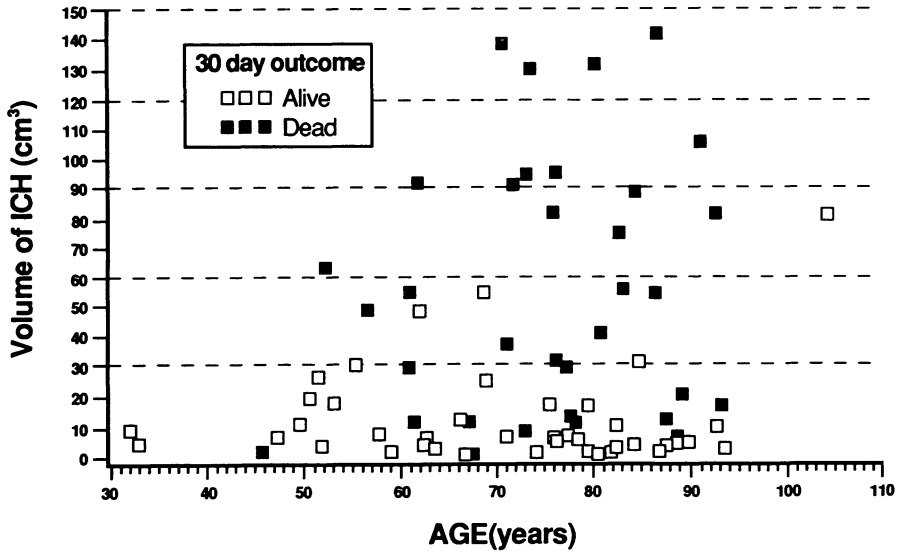

FIG 3. *Plot shows 30-day outcome for the 76 patients with deep hemorrhages according to patient's age and volume of parenchymal hemorrhage. ICH indicates intracerebral hemorrhage.*

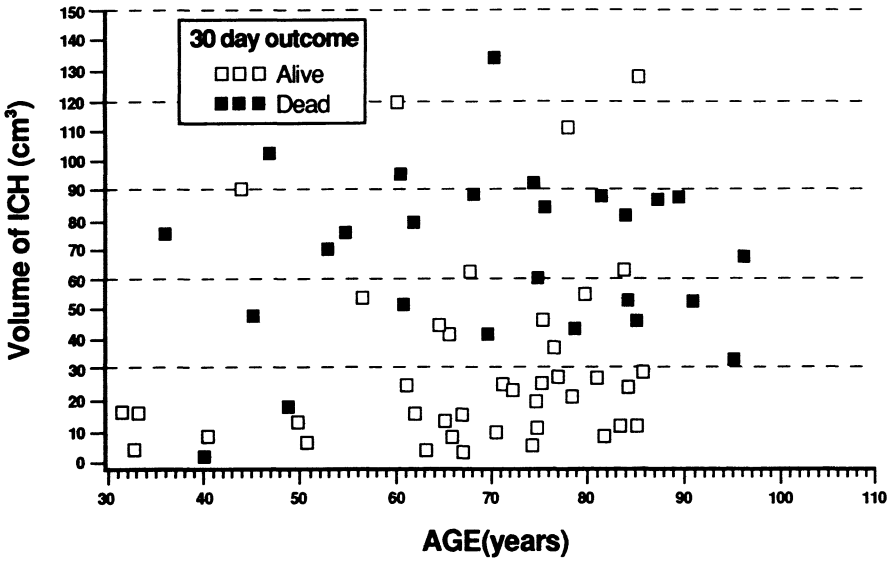

FIG 4. *Plot shows 30-day outcome for the 66 patients with lobar hemorrhages according to patient's age and volume of parenchymal hemorrhage. ICH indicates intracerebral hemorrhage.*

mal hemorrhage ($P<.0001$), volume of ventricular hemorrhage ($P=.0004$), and operative removal of hemorrhage ($P=.008$) were significant predictors of 30-day mortality. However, the mean overall 30-day morbidity and mortality for deep and lobar hemorrhages, as measured by the modified Oxford Handicap Scale, was similar for patients who underwent operation (4.8 ± 1.1) and those who did not (4.7 ± 1.6; Table 2).

To develop a logistic regression model of 30-day mortality that was similar to the model of intracerebral hemorrhage survival reported by Tuhrim and colleagues,[6] we divided the volume of parenchymal hemorrhage, as calculated by computerized image analysis, into three categories (less than 30 cm^3, 30 to 60 cm^3, and 61 cm^3 or greater) and the Glasgow Coma Scale score into two categories (9 or more and 8 or less). Using these two categorical variables, the resulting model correctly predicted 30-day mortality with a sensitivity of 97%, a specificity of 97%, a positive predictive value of 96%, and negative predictive value of 98% (Table 3). When the analysis was limited to the 142 deep and lobar hemorrhages, mortality was correctly predicted with a sensitivity of 97% and a specificity of 98%. When we substituted the volume categories of Tuhrim and colleagues (0 to 26 cm^3, 27 to 72 cm^3, 73 cm^3 or greater) into our model of 30-day mortality for the 142 deep and lobar hemorrhages, the resulting predictive model had almost identical sensitivity (98%) and specificity (98%). Addition of the categorical variable intraventricular blood (yes, no) did not improve the sensitivity or specificity of any of the models.

The volume of intracerebral hemorrhage calculated by image analysis correlated quite well with the volume calculated by hand using the formula for an ellipsoid ($r=.94$). The mean of the difference between the volume calculated by image analysis minus the volume calculated by the ellipsoid method was -3.6 ± 15 cm^3. When we used the ellipsoid volume to classify subjects into the three volume categories (less than 30 cm^3, 30 to 60 cm^3, and greater than 60 cm^3), only 18 of the 162 subjects were categorized differently than when we used volumes calculated by computerized image analysis. When the ellipsoid volume of intracerebral hemorrhage was substituted for the hemorrhage volume calculated

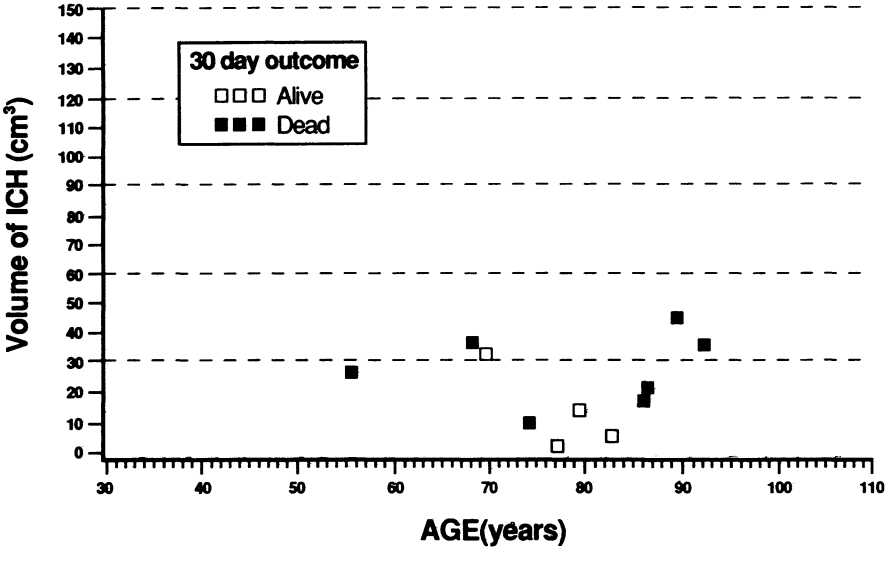

FIG 5. *Plot shows 30-day outcome for the 11 patients with cerebellar hemorrhages according to patient's age and volume of parenchymal hemorrhage. ICH indicates intracerebral hemorrhage.*

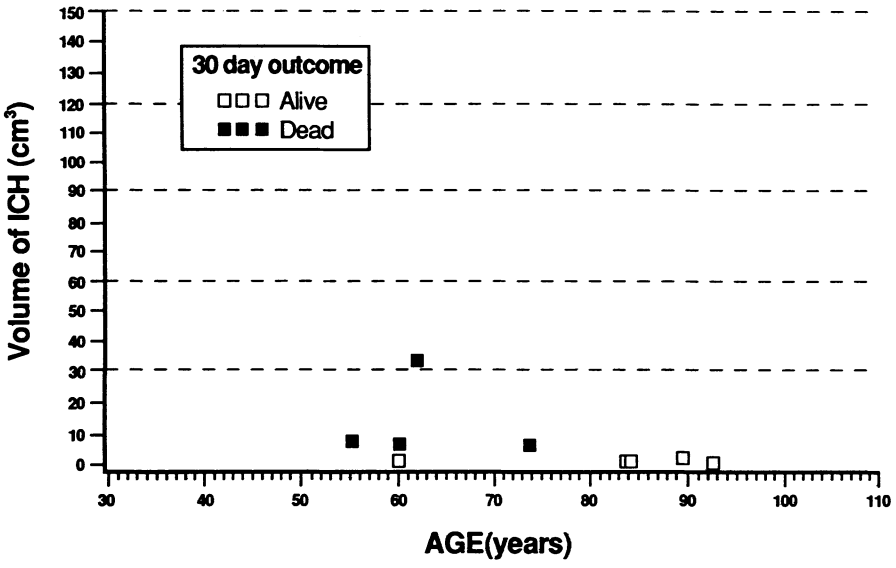

FIG 6. *Plot shows 30-day outcome for the nine patients with pontine hemorrhages according to patient's age and volume of parenchymal hemorrhage. ICH indicates intracerebral hemorrhage.*

by computerized image analysis, the sensitivity (96%) and specificity (98%) of the model of 30-day mortality in Table 3 changed little.

Discussion

Volume of intracerebral hemorrhage is the strongest predictor of 30-day outcome for all locations of spontaneous intracerebral hemorrhage. Our model of 30-day mortality, using three categories of hemorrhage volumes and two categories of Glasgow Coma Scale scores, correctly predicted outcomes with a sensitivity and specificity of 97%. The close similarity of our model to that of Tuhrim and colleagues[6] indicates that both models are applicable to other populations of patients with intracerebral hemorrhage. Accordingly, the probabilities in the last column of Table 3 could be used to stratify patients in future treatment trials of intracerebral hemorrhage and to predict outcome for individual patients. For instance, a patient with parenchymal hemorrhage volume of 15 cm^3 and a Glasgow Coma Scale score of 12 would be predicted to have a 19% chance of dying by 30 days.

Clinical predictors of outcome must be easy to use if they are to gain wide acceptance. Although many CT scanners have the capability to outline and measure areas of hemorrhage on a CT slice, the process is time consuming, as was our own computerized image analysis method. Other methods, such as the method of best-fitting circles,[23] are also relatively cumbersome. To the physician making quick and critical decisions about a patient with an intracerebral hemorrhage, the ideal method is one that gives a reasonable estimation of actual hemorrhage volume as quickly as possible. For this reason, we compared hemorrhage volume estimated by the formula for an ellipsoid with the computerized image analysis method. We found that the simple ellipsoid method, which can easily estimate hemorrhage volume within 1 to 2 minutes, predicts 30-day mortality nearly as well as the more exact method of computerized image analysis. Thus, a physician using a copy of Table 3, the formula for an ellipsoid, the CT film, and the Glasgow Coma Scale score can accurately predict 30-day mortality at the patient's bedside within about 5 minutes.

The lethal volume of parenchymal hemorrhage varies by location. All patients with a pontine hemorrhage greater than 5 cm^3 or cerebellar hemorrhage greater than 30 cm^3 died within 30 days. Of the 11 patients with deep hemorrhages with an initial parenchymal volume of less than 30 cm^3 who died, seven had hemor-

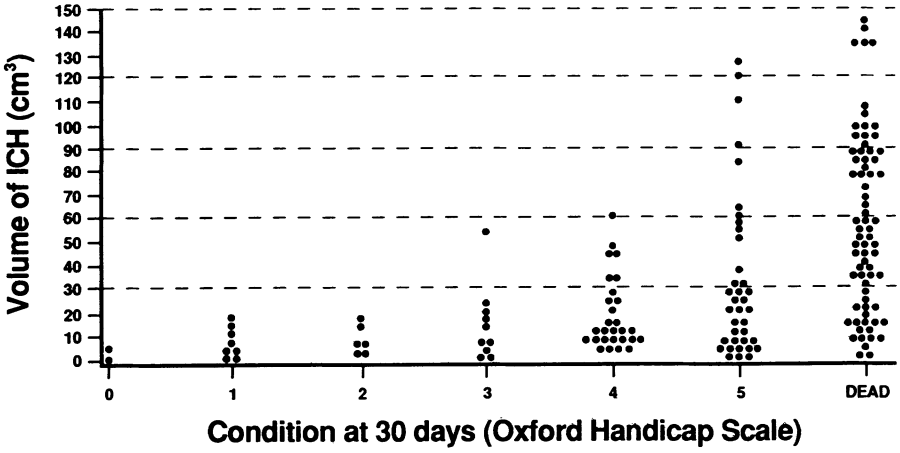

FIG 7. *Plot shows 30-day outcomes for 162 patients with intracerebral hemorrhages (ICH) as measured by modified Oxford Handicap Scale according to volume of parenchymal hemorrhage. According to the modified Oxford Handicap Scale,[20] 0=no symptoms; 1=minor symptoms that do not interfere with life-style; 2=minor handicap; 3=moderate handicap; 4=moderately severe handicap; 5=severe handicap; 6=dead.*

TABLE 1. Multivariate Logistic Regression Model of 30-Day Mortality After Intracerebral Hemorrhage

Variable	Parameter estimate	*P*
Intercept	−2.65	.0769
Volume of parenchymal hemorrhage	0.0312 (per cm^3)	<.0001
Volume of ventricular hemorrhage	0.0366 (per cm^3)	.008
Glasgow Coma Scale	−0.1644 (per unit)	.026
Operation performed (0=no, 1=yes)	−1.4634	.012
	Model R^2=.286	

TABLE 3. Model of 30-Day Mortality Using Volume of Parenchymal Hemorrhage and Glasgow Coma Scale

Glasgow Coma Scale score	ICH volume (cm^3)	No. in risk group	Dead	Expected dead	Probability of death by 30 days
≥9	<30	77	13	15	0.19
≥9	30-60	19	11	9	0.46
≥9	>60	17	12	13	0.75
≤8	<30	15	7	7	0.44
≤8	30-60	15	11	11	0.74
≤8	>60	19	17	17	0.91

ICH, intracerebral hemorrhage.

rhages that were thalamic in origin. The small number of hemorrhages in each of these locations is the reason why location was not a significant predictor of mortality in the multivariate logistic regression models. Volume of intraventricular hemorrhage, on the other hand, is a strong independent predictor of 30-day mortality. However, the difficulty of accurately measuring the volume of intraventricular blood on the CT film at the bedside severely limits its usefulness as a practical predictor of mortality.

Intracerebral hemorrhage kills quickly and often, and survivors have substantial morbidity. Half of the 30-day mortality in the present study occurred by the end of the second day, which is similar to other population studies of intracerebral hemorrhage.[2-5] Only 10% of all hospitalized hemorrhage patients were totally independent at 30 days. Some survivors of intracerebral hemorrhage do improve functionally over the subsequent months, and other case series have reported better outcomes at 6 months and 1 year.[6,8,9,11-14] However, the overall outcome of intracerebral hemorrhage remains grim.

Operative removal of hemorrhage was associated with decreased 30-day mortality, after adjusting for the volume of parenchymal and intraventricular hemorrhage, initial Glasgow Coma Scale score, age, and location of hemorrhage. However, overall morbidity and mortality were not significantly different in patients who underwent operation compared with those who did not. Our study is not a randomized treatment trial of intracerebral hemorrhage and was not designed to evaluate the effectiveness of operative removal of intracerebral hemorrhage. However, our findings are consistent with two of the four small randomized trials of operative removal of intracerebral hemorrhage.[17,24-26] In a 100-patient randomized study of endoscopic removal of hemorrhage within 48 hours of onset, compared with best medical treatment alone, Auer et al[24] found a significantly lower mortality rate at 6 months in the operative group (42%) than in the medically treated group (70%). In patients with large hematomas (greater than 50 cm^3), the quality of survival was not influenced by operation, whereas the mortality rate was significantly lower. In contrast, endoscopic removal of smaller hematomas led to improved quality of survival, but the mortality rate was unaffected. In a randomized study of 52 patients, Juvela and colleagues[17] reported no significant difference in mortality and morbidity between patients who underwent operation and those who did not. In a subgroup analysis of those patients with an initial Glasgow Coma Scale score of 7 to 10, 0 of 4 operated patients had died by 6 months compared with 4 of the 5 medically treated patients (P<.05). All surviving patients in this subgroup were severely disabled.

There is no proven treatment for the estimated 37 000 patients who have an intracerebral hemorrhage in the United States each year.[27] Because of the rapid and severe devastation associated with intracerebral hemorrhage, innovative treatments need to be developed and evaluated. Bedside estimation of the volume of parenchymal hemorrhage can be a powerful tool for selection and stratification of patients in these future treatment studies.

TABLE 2. Thirty-Day Outcome by Operative Category for 142 Patients With Deep and Lobar Hemorrhages

	Oxford Handicap Scale							
	0	1	2	3	4	5	Dead	Total
Operative removal	0	0	1	2	4	10	6	23
No operative removal	2	7	4	7	24	21	54	119
Total	2	7	5	9	28	31	60	142

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References

1. Broderick J, Brott T, Tomsick T, Huster G, Miller R. The risk of subarachnoid and intracerebral hemorrhages in blacks as compared with whites. *N Engl J Med.* 1992;326:733-736.
2. Broderick JP, Phillips SJ, Whisnant JP, O'Fallon WM, Bergstralh EJ. Incidence rates of stroke in the eighties: the end of the decline in stroke? *Stroke.* 1989;20:577-582.
3. Bamford J, Sandercock P, Dennis M, Burn J, Warlow C. A prospective study of acute cerebrovascular disease in the community: the Oxfordshire Community Stroke Project: 1981-86. *J Neurol Neurosurg Psychiatry.* 1990;53:16-22.
4. Giroud M, Grass P, Chadan N, Beuriat P, Milan C, Arveux P, Dumas R. Cerebral haemorrhage in a French prospective population study. *J Neurol Neurosurg Psychiatry.* 1991;54:595-598.
5. Fogelholm R, Nuutila M, Vuorela AL. Primary intracerebral haemorrhage in the Jyvaskyla region, Central Finland, 1985-89: incidence, case fatality rate, and functional outcome. *J Neurol Neurosurg Psychiatry.* 1992;55:546-552.
6. Tuhrim S, Dambrosia J, Price T, Mohr J, Wolf P, Hier D, Kase C. Intracerebral hemorrhage: external validation and extension of a model for prediction of 30-day survival. *Ann Neurol.* 1991;29:658-663.
7. Massaro AR, Sacco RL, Mohr JP, Foulkes MA, Tatemichi TK, Price TR, Hier DB, Wolf PA. Clinical discriminators of lobar and deep hemorrhages: the Stroke Data Bank. *Neurology.* 1991;41:1881-1885.
8. Portenoy R, Lipton R, Berger A, Lesser M, Lantos G. Intracerebral haemorrhage: a model for the prediction of outcome. *J Neurol Neurosurg Psychiatry.* 1987;50:976-979.
9. Daverat P, Castel JP, Dartigues JF, Orgogozo JM. Death and functional outcome after spontaneous intracerebral hemorrhage: a

prospective study of 166 cases using multivariate analysis. *Stroke.* 1991;22:1-6.
10. Young WB, Lee KP, Pessin MS, Kwan ES, Rand WM, Caplan LR. Prognostic significance of ventricular blood in supratentorial hemorrhage: a volumetric study. *Neurology.* 1990;40:616-619.
11. Volpin L, Cervellini P, Colombo F, Zanusso M, Benedetti A. Spontaneous intracerebral hematomas: a new proposal about the usefulness and limits of surgical treatment. *Neurosurgery.* 1984;15: 663-666.
12. Garde A, Bohmer G, Selden B, Neiman J. 100 cases of spontaneous intracerebral haematoma: diagnosis, treatment and prognosis. *Eur Neurol.* 1983;22:161-172.
13. Fieschi C, Carolei A, Fiorelli M, Argentino C, Bozzao L, Fazio C, Salvetti M, Bastianello S. Changing prognosis of primary intracerebral hemorrhage: results of a clinical and computed tomographic follow-up study of 104 patients. *Stroke.* 1988;19:192-195.
14. Douglas M, Haerer A. Long-term prognosis of hypertensive intracerebral hemorrhage. *Stroke.* 1982;13:488-491.
15. Radberg J, Olsson J, Radberg C. Prognostic parameters in spontaneous intracerebral hematomas with special reference to anticoagulant treatment. *Stroke.* 1991;22:571-576.
16. Poungvarin N, Bhoopat W, Viriyavejakui A, Rodprasert P, Buranasiri P, Sukondhabhant S, Hensley M, Strom B. Effects of dexamethasone in primary supratentorial intracerebral hemorrhage. *N Engl J Med.* 1987;316:1229-1233.
17. Juvela S, Heiskanen O, Poranen A, Valtonen S, Kuurne T, Kaste M, Troupp H. The treatment of spontaneous intracerebral hemorrhage: a prospective randomized trial of surgical and conservative treatment. *J Neurosurg.* 1989;70:755-758.
18. Steiner I, Gomori JM, Melamed E. The prognostic value of the CT scan in conservatively treated patients with intracerebral hemorrhage. *Stroke.* 1983;15:279-282.
19. Jennett B, Bond M. Assessment of outcome after severe brain damage: a practical scale. *Lancet.* 1975;1:480-484.
20. Bamford J, Sandercock P, Warlow C, Slattery J. Interobserver agreement for the assessment of handicap in stroke patients. *Stroke.* 1989;20:828.
21. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. *J Am Stat Assoc.* 1958;53:457-481.
22. Breslow NE, Day NE. Statistical methods in cancer research. In: *The Analysis of Case-Control Studies.* Lyon, France: International Urgency for Research on Cancer; 1980: vol 11.
23. Franke CL, Versteege CWM, Van Gijn J. The best fit method: a simple way for measuring the volume of an intracerebral haematoma. *Neuroradiology.* 1988;30:73-75.
24. Auer LM, Deinsberger W, Niederkorn K, Gell G, Kleinert R, Schneider G, Holzer P, Bone G, Mokry M, Körner E, Kleinert G, Hanusch S. Endoscopic surgery versus medical treatment for spontaneous intracerebral hematoma: a randomized study. *J Neurosurg.* 1989;70:530-535.
25. Batjer HH, Reisch JS, Allen BC, Plaizier LJ, Su CJ. Failure of surgery to improve outcome in hypertensive putaminal hemorrhage: a prospective randomized trial. *Arch Neurol.* 1990;47:1103-1106.
26. McKissock W, Richardson A, Taylor J. Primary intracerebral haemorrhage: a controlled trial of surgical and conservative treatment in 180 unselected cases. *Lancet.* 1961;2:221-226.
27. Broderick JP, Brott TG, Tomsick T, Miller R, Huster G. Intracerebral hemorrhage is more than twice as common as subarachnoid hemorrhage. *J Neurosurg.* 1993;78:188-191.