Intracerebral hemorrhage mortality is not changing despite declining incidence

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ABSTRACT

Objective: To determine trends in incidence and mortality of intracerebral hemorrhage (ICH) in a rigorous population-based study.

Methods: We identified all cases of spontaneous ICH in a South Texas community from 2000 to 2010 using rigorous case ascertainment methods within the Brain Attack Surveillance in Corpus Christi Project. Yearly population counts were determined from the US Census, and deaths were determined from state and national databases. Age-, sex-, and ethnicity-adjusted incidence was estimated for each year with Poisson regression, and a linear trend over time was investigated. Trends in 30-day case fatality and long-term mortality (censored at 3 years) were estimated with log-binomial or Cox proportional hazards models adjusted for demographics, stroke severity, and comorbid disease.

Results: A total of 734 cases of ICH were included. The age-, sex-, and ethnicity-adjusted ICH annual incidence rate was 5.21 per 10,000 (95% confidence interval [CI] 4.36, 6.24) in 2000 and 4.30 per 10,000 (95% CI 3.21, 5.76) in 2010. The estimated 10-year change in demographic-adjusted ICH annual incidence rate was –31% (95% CI –47%, –11%). Yearly demographic-adjusted 30-day case fatality ranged from 28.3% (95% CI 19.9%, 40.3%) in 2006 to 46.5% (95% CI 35.5, 60.8) in 2008. There was no change in ICH case fatality or long-term mortality over time.

Conclusions: ICH incidence decreased over the past decade, but case fatality and long-term mortality were unchanged. This suggests that primary prevention efforts may be improving over time, but more work is needed to improve ICH treatment and reduce the risk of death. *Neurology*® 2014;82:2180–2186

GLOSSARY

BASIC = Brain Attack Surveillance in Corpus Christi; **CI** = confidence interval; **GCS** = Glasgow Coma Scale; **ICD-9** = *International Classification of Diseases-9*; **ICH** = intracerebral hemorrhage; **MA** = Mexican American; **NHW** = non-Hispanic white; **NIHSS** = NIH Stroke Scale; **RR** = risk ratio; **SSN** = social security number; **TDH** = Texas Department of Health.

Intracerebral hemorrhage (ICH) accounts for about 10% of all strokes but a disproportionate amount of stroke mortality.¹ Incidence of ischemic stroke is declining,² and due to shared risk factors, ICH incidence may be expected to be declining as well. Limited data exist on recent trends in ICH burden. Trends in ICH incidence and case fatality were the subject of a 2010 systematic review,³ though most of the included 36 studies were from before 2000, and few were specifically designed to investigate time trends. An understanding of time trends in the epidemiology of ICH incidence, case fatality, and long-term mortality is necessary to gauge the effectiveness of stroke prevention and treatment efforts.

Due to improved control of risk factors such as hypertension over the past decade,⁴ there is a need for more recent data on trends in ICH. The objective of this analysis was to investigate trends in ICH incidence, 30-day case fatality, and long-term mortality from 2000 to 2010 within the population-based Brain Attack Surveillance in Corpus Christi (BASIC) Project.

Supplemental data
at Neurology.org

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METHODS Identification of cases. Cases of spontaneous ICH among individuals older than 44 years were identified from January 1, 2000, through December 31, 2010, using the population-based BASIC Project. Detailed study methods have been reported.² Briefly, all cases of acute neurologic disease presenting to one of the 7 hospitals in Nueces County, Texas, are identified through a combination of active and passive surveillance, including review of emergency department and hospital admission logs, as well as review of discharge ICD-9 codes for stroke (codes 430–438, excluding codes 433.X0 and 434.X0 [X = 1–9], 437.0, 437.2, 437.3, 437.4, 437.5, 437.7, 437.8, and 438). BASIC surveillance includes only patients 45 years and older to maintain the efficiency of the active surveillance process. Board-certified neurologists validate the diagnosis of ICH based on review of source documentation. ICH due to trauma, tumor, or hemorrhagic conversion of ischemic stroke or venous sinus thrombosis is excluded, whereas ICH associated with arteriovenous malformation or anticoagulation is included. In earlier years, the project included rigorous out-of-hospital case capture methods, though these have been eliminated over time to improve project efficiency as previously described.² Surveillance of primary care physician and cardiologist offices had identified no cases of ICH, and surveillance of neurologists' offices had identified 2 of 328 ICH cases at the time these methods were eliminated. Passive surveillance data were missing from 1 hospital system for a 6-month timeframe in 2008, though active surveillance continued. In other 6-month time frames from this system, passive surveillance typically identified either 0 or 1 ICH case missed by active surveillance. Only the first ICH captured by BASIC per individual was included, and therefore the included ICH may have been the first or recurrent event for an individual. Individuals with ethnicity other than Mexican American (MA) or non-Hispanic white (NHW) were excluded due to low numbers.

Data collection. Trained abstractors reviewed the medical record for information on clinical presentation and medical comorbidities. Medical history including hypertension, history of prior stroke/TIA, diabetes, atrial fibrillation, coronary artery disease, and high cholesterol as well as insurance status were determined based on chart documentation. Smoking status was determined from the medical record and categorized as never (referent), current, or former smoker. First recorded NIH Stroke Scale (NIHSS)⁵ and Glasgow Coma Scale (GCS) were recorded from the medical record or abstracted based on the first documented examination from the chart. Age, sex, and ethnicity (MA or NHW) were determined from the medical record, as we have previously shown excellent agreement ($\kappa = 0.94$) between medical record and self-report in this community for ethnicity.⁶

Population counts. Population counts in 2000 and 2010 were obtained from the 2000 and 2010 Census. Yearly population counts for 2001–2009 were obtained from intercensal estimates.⁷ Counts were downloaded in 5-year increments by sex and ethnicity (MA or NHW), and aggregated to 3 age categories: 45–59, 60–74, and 75 and older. Counts for Hispanics, regardless of race, were used as the population at risk for the MA group because among the population 45 and older, only 2.7% report ancestry from Spain, Central or South America, or Puerto Rico.⁸ Figures were generated to demonstrate time trends in the Nueces County population by age category, sex, and ethnicity.

Identification of deaths. All-cause mortality was identified through medical record review, Texas Department of Health (TDH) death certificate data, Social Security Death Index, or

direct contact with families when scheduling follow-up assessments, as previously described.⁹ In the early years, death was identified strictly from medical record review or TDH death certificate data. In 2005, the TDH would not allow matches using social security number (SSN); during this time, the Social Security Death Index became the primary source for identification of death, supplemented with TDH data and medical record review. More recently, due to a change in policy at TDH, TDH data (including SSN) were the primary source of identification of mortality supplemented with medical record data. Regardless of the source, cases were considered a match if 4 of 5 identifiers (first name, last name, date of birth, SSN, and permanent residence) were identical to confirm a case as deceased, though some manual review was required for special circumstances such as transposed numbers.

Statistical analysis. Descriptive statistics were calculated for all variables and presented as proportions, medians and interquartile ranges, or means and SDs. Descriptive statistics were reported overall, and also during the first 3 years (2000–2002) and the final 3 years (2008–2010) to describe changes over time. Poisson regression models were used to calculate adjusted ICH incidence rates and trends over the 2000–2010 time period. The outcome for the models was the observed number of ICH cases within categories of age (45–59, 60–74, and 75+), ethnicity, and sex, and log population counts within those categories were used as the offset (i.e., population at risk). Yearly demographic-adjusted rates were calculated by treating year as a series of indicator variables and including age, sex, and ethnicity as additional predictors. In separate models, time trends were modeled as a linear term of years since 2000 to determine if the trend in ICH incidence rates was decreasing, increasing, or not changing significantly over time. Incidence models were performed first for all ages and then sex- and ethnicity-adjusted models were repeated stratified by each age category (45–59, 60–74, and 75+) as we have previously shown differences in incidence by age category.⁶ An interaction term for ethnicity and time was explored in the incidence models but not included as it was not statistically significant. To determine whether the trends were different in more recent years compared with earlier years, a post hoc sensitivity analysis was done allowing for the trend to change using a linear spline with a knot at 2005.

To examine risk of death after ICH, log-binomial regression models¹⁰ were used to model 30-day mortality (e.g., case fatality), and Cox proportional hazards were used to model long-term mortality (censored at 36 months post-ICH presentation or December 31, 2011, whichever came first). Time trends in 30-day mortality risk were examined with year as a series of dummy variables or as a linear trend, similar to the strategy for incidence. Time trends in long-term mortality were investigated treating year as a linear term. Thirty-day and long-term mortality models were initially adjusted for demographics including age (quadratic due to nonlinear effect), sex, and ethnicity, and then repeated additionally adjusting for prespecified mortality risk factors including NIHSS (quadratic due to nonlinear effect), GCS, coronary artery disease, prior stroke/TIA, atrial fibrillation, diabetes, and smoking. The fully adjusted long-term mortality model added time-varying recurrence of any stroke (ICH or ischemic stroke). Cumulative sums of residuals were used to assess the linearity assumption for the time trend and other continuous variables for all models, and to check the proportional hazards assumption for long-term mortality models.¹¹ Variables that did not satisfy the proportional hazards assumption had interaction terms with log-transformed time added to the models. Data on ICH volume and limitations in life-sustaining treatments were only available for 2000–2003 and therefore not included as model covariates.¹²

Standard protocol approvals, registrations, and patient consents. The Institutional Review Board at the University of Michigan and the individual Corpus Christi hospital systems approved this project.

RESULTS A total of 734 cases of ICH met the inclusion criteria. Baseline characteristics of the study population are shown in table 1 for the overall population, as well as for time periods 2000–2002 and 2008–2010. When comparing time periods, risk factors of hypertension, diabetes, and high cholesterol were all noted more commonly in 2008–2010 than in 2000–2002. Mean age at onset was lower in the later years of the study, though initial stroke severity, as measured by NIHSS and GCS, was similar in the early and late time epochs. Population trends over time in the community by age category, sex, and ethnicity are shown in figure e-1 on the *Neurology*® Web site at Neurology.org. The community population remained generally stable in those 75 and older, with population growth seen in the younger age groups, particularly among younger MAs.

The trend of age-, sex-, and ethnicity-adjusted ICH annual incidence rates is shown in figure 1A, and trends by age strata (sex- and ethnicity-adjusted) are shown in figure 1B. The model-predicted year 2000 adjusted ICH annual incidence rate was 5.21 per 10,000 (95% confidence interval [CI] 4.36–6.24)

and for 2010 was 4.30 per 10,000 (95% CI 3.21–5.76). In the model treating time as a linear variable, the estimated 10-year change in demographic-adjusted ICH incidence rate was –31% (95% CI –47%, –11%). These results and the findings by age strata are summarized in table 2. The decline in incidence rates over time was most prominent in those aged ≥ 75 and 60–74. The youngest age group (45–59) showed no decline in incidence rates over time and if anything there was a non-significant trend toward an increase in the incidence rate in this age group (table 2 and figure 1B).

MAs were more likely to have ICH than NHWs (overall risk ratio [RR] 1.75, 95% CI 1.48, 2.07), with a larger difference noted at ages 45–59 (RR 2.50, 95% CI 1.82, 3.42) than at ages 60–74 (RR 1.88, 95% CI 1.49, 2.37) or ≥ 75 (RR 1.37, 1.09, 1.74). In exploratory analysis, there was no evidence of effect modification of ethnicity on time trends (interaction term $p \geq 0.40$ overall and within each age category). The post hoc sensitivity analysis exploring a differential trend in incidence comparing 2000–2005 to 2006–2010 found no difference in the trend between the 2 time periods ($p = 0.44$).

Annual demographic-adjusted 30-day case fatality ranged from as low as 28.3% (95% CI 19.9%, 40.3%) in 2006 to 46.5% (95% CI 35.5, 60.8) in 2008. When time was treated as a linear variable, there was no change in demographic-adjusted ICH case fatality over time (table 2). In addition, adjusting for stroke severity (NIHSS and GCS) and comorbidities did not change the trend. The trends in 30-day case fatality (demographic and fully adjusted) are shown in figure 2. For long-term 36-month mortality, there was no change over time whether adjusting for demographics alone or when accounting for demographics, severity, comorbidities, and recurrent stroke (table 2). Considering ethnic differences in risk of death, there was a trend toward lower risk of death for MAs compared with NHWs in the fully adjusted models for 30-day case fatality (RR 0.87, 95% CI 0.75–1.00, $p = 0.06$) and 36-month mortality (hazard ratio 0.82, 95% CI 0.66–1.01, $p = 0.06$).

DISCUSSION We found declining incidence of ICH but unchanged case fatality and long-term mortality between 2000 and 2010 in this population-based study with rigorous case ascertainment methods. This declining incidence is encouraging and is possibly representative of improved control of hypertension in the community in this time frame.⁴ We do not have data on trends in overall hypertension prevalence or control specific to this community in order to test this hypothesis. While hypertension was noted more commonly in ICH patients in later years, this definition was based on chart documentation and is unlikely to reflect a true measure of hypertension

Table 1 Demographics and stroke risk factors over time

	Overall (% unless specified, n = 734)	2000–2002 (% unless specified, n = 209)	2008–2010 (% unless specified, n = 191)
Mean age, y (SD)	69.7 (13.1)	72.1 (12.5)	67.4 (13.7)
Female	46.6	51.7	42.4
Mexican American	61.4	57.4	66.0
Median NIHSS (Q1, Q3)	10 (4, 22)	11 (3, 23)	11 (5, 22)
Median GCS (Q1, Q3)	13 (6, 15)	12 (5, 15)	13 (6, 15)
Hypertension	77.4	70.0	80.5
History of stroke or TIA	29.9	29.5	27.9
Diabetes mellitus	33.2	28.0	34.2
Atrial fibrillation	8.1	6.8	6.8
Coronary artery disease	27.3	28.5	28.9
High cholesterol	23.2	15.9	30.5
Smoking status			
Current	15.1	12.3	16.3
Former	13.5	15.9	8.4
Never	71.4	71.8	75.3
Insurance	86.7	91.7	83.2

Abbreviations: GCS = Glasgow Coma Scale; NIHSS = NIH Stroke Scale; Q = quartile.

Figure 1 Trend in adjusted intracerebral hemorrhage incidence rate

Points represent the model-predicted incidence treating year as a categorical variable. Smoothed lines represent the model-predicted incidence assuming a linear trend. (A) Overall age-, sex-, and ethnicity-adjusted rates for all individuals 45 and older. (B) Sex- and ethnicity-adjusted rates by age group.

prevalence or control within the community. The decline in incidence was almost entirely driven by declines in the older age groups, particularly in individuals over 74 years old, with no decline seen in younger individuals (age 45–59). Fortunately, ICH is much less common in younger individuals, though

better efforts at primary prevention in all age groups are warranted due to the greater potential for long-term disability in younger people with ICH.

Reasons for the lack of decline in incidence in the youngest age group are unclear. Mean age at the time of ICH was younger in the later years of the study. This observation is likely due to a combination of the decrease in the incidence rate in older individuals and an increase in the number of younger individuals in the at-risk community population (figure e-1) leading to a relative increase in the raw number of younger ICH cases over time in our sample. It is likely that hemorrhage etiology is different by age,^{13,14} with older individuals more likely to have amyloid angiopathy or antithrombotic-associated hemorrhage than younger individuals. We do not have data available on suspected ICH etiology (e.g., amyloid, anticoagulant, or antiplatelet associated), and therefore we cannot be certain of the degree to which differences in ICH etiology by age group, or over time,^{14,15} may contribute to the observed trends. Differential trends in awareness or control of hypertension by age could also contribute to the stable incidence in the younger age group. However, a report based on the National Health and Nutrition Examination Survey suggests that hypertension awareness, management, and control have been improving in both younger and older comparable age groups from 1999 to 2010, making this explanation unlikely.⁴

We have previously reported data from this community showing that ICH is more common in MAs compared with NHWs.⁶ Considering ethnic differences in trends is important to understand if we are making progress towards eliminating disparities in incidence. We did not detect an interaction between

Table 2 Time trends in intracerebral hemorrhage incidence, 30-day mortality, and 36-month mortality

Outcome	Risk ratio for time per 10 years (95% CI)	p
Incidence		
All ages (≥45 y) ^a	0.69 (0.53, 0.89)	0.004
45–59 y ^b	1.23 (0.81, 1.86)	0.33
60–74 y ^b	0.57 (0.39, 0.81)	0.002
≥75 y ^b	0.53 (0.37, 0.78)	0.001
30-day case fatality		
Demographic adjusted ^a	0.93 (0.71, 1.21)	0.60
Fully adjusted ^c	1.06 (0.85, 1.33)	0.61
36-month mortality		
Demographic adjusted ^d	0.96 (0.70, 1.31)	0.80
Fully adjusted ^e	0.90 (0.65, 1.24)	0.51

Abbreviation: CI = confidence interval; GCS = Glasgow Coma Scale; NIHSS = NIH Stroke Scale.

^aAdjusted for age (quadratic in mortality models), sex, and ethnicity.

^bAdjusted for sex and ethnicity.

^cAdjusted for age (quadratic), sex, ethnicity, NIHSS (quadratic), GCS, coronary disease, prior stroke/TIA, atrial fibrillation, diabetes, and smoking.

^dAdjusted for age (quadratic), sex, ethnicity, and interaction between age × log (time) to satisfy the proportional hazards assumption.

^eAdjusted for age (quadratic), sex, ethnicity, NIHSS (quadratic), GCS, coronary disease, prior stroke/TIA, atrial fibrillation, diabetes, smoking, recurrent stroke, and interaction terms between age × log (time) and NIHSS × log (time) to satisfy the proportional hazards assumption.

Figure 2 Trend in adjusted intracerebral hemorrhage 30-day case fatality

Points represent the model-predicted mortality treating year as a categorical variable. Smoothed lines represent the model-predicted mortality assuming a linear trend with time. (A) Adjusted for age (quadratic due to nonlinear effects), sex, and ethnicity. (B) Additionally adjusted for NIH Stroke Scale (quadratic due to nonlinear effects), Glasgow Coma Scale, coronary disease, prior stroke/TIA, atrial fibrillation, diabetes, and smoking.

ethnicity and time in our model, suggesting that there is no difference in the rate of decline by ethnicity and therefore the ethnic disparity in ICH incidence remains.⁶ This finding should be interpreted with some caution as we had limited power to detect an interaction between ethnicity and time. MAs were less likely to die after ICH than NHWs, though this finding was of borderline statistical significance.

Our findings of a decrease in ICH incidence over time are in contrast to studies that have shown ICH incidence to be stable^{13,16–20} or increasing.²¹ Other studies have shown a decline in ICH incidence over time, though these reports used data from prior to 2000 as a comparison group^{14,22} and therefore our results may not be comparable. Possible reasons for the disparate findings across studies may include different methodologies (administrative data vs population-based case ascertainment), regional differences in ICH trends, or differences in time period or populations studied. These differences complicate a direct comparison of results across studies. To assess whether inclusion of more recent data may have affected our findings, we explored the possibility of a period effect in a post hoc sensitivity analysis but did not detect a difference in the time trend before vs after 2005.

The lack of improvement in case fatality or long-term mortality is not surprising, given that there have been no specific therapies proven to reduce risk of death after ICH adopted during the time period of this study. Recent studies of blood pressure lowering in ICH have shown a possible benefit on functional outcome²³ but no impact on mortality. Our results were similar whether adjusting for demographics

alone or demographics plus stroke severity and comorbid illness. Other studies reporting trends in ICH case fatality have had mixed results, with most showing generally stable case fatality^{3,14,17,19,24} and others showing a modest decrease.^{25–27} Some of the studies that did show a decrease examined in-hospital death based on administrative datasets with more than 100,000 ICH cases and therefore had greater power to detect small differences. With the sample size of our study, we cannot rule out the possibility of a smaller but clinically meaningful reduction in mortality over time.

Early decisions to limit the intensity of treatment provided are common in ICH and potent independent predictors of mortality.¹² We only have detailed data on treatment limitations for 2000–2003 and therefore do not know how these factors may impact trends in mortality. Future efforts to reduce mortality after ICH should involve a careful examination of these decisions that involve a trade-off between early death and survival with potential for disability. This community did not have a dedicated neurologic intensive care unit and therefore we cannot comment on any impact of neurologic critical care on mortality trends.²⁸

Our study has limitations. There were minor changes to case ascertainment methods over time,² though these changes primarily involved updates to identifying nonhospitalized cases and would be unlikely to affect detection of ICH cases. Nueces County, Texas, has a large Hispanic population and therefore incidence rates may not be applicable to other regions with different race–ethnic distribution. We did not have any data on risk factor treatment or

control in a community control group and cannot comment on how these factors may have influenced ICH incidence. We did not report cause of death, as BASIC no longer collects this information due to difficulties in determining cause of death in stroke patients.²⁹ We did not have detailed CT findings to adjust for ICH volume in the mortality analyses, though we have previously shown that ICH volume adds little to model predictive ability when detailed clinical findings (NIHSS and GCS) are included.³⁰

Our data from a population-based study with rigorous surveillance methods suggest that incidence of ICH is decreasing over the past decade, but mortality remains unchanged. These findings suggest that efforts to improve stroke prevention have been effective but that more work is needed to improve treatment of ICH and reduce risk of death.

Comment: Intracerebral hemorrhage incidence declines, but it is still highly fatal

Overall stroke mortality has declined over the last decades—a welcome trend.¹ However, recent trends in intracerebral hemorrhage (ICH) incidence are not as clear. On the one hand, there has been increasingly good population control of hypertension; on the other hand, there is increasing use of anticoagulants and antithrombotic drugs, an epidemic of obesity and diabetes, and changing racial and ethnic demographics.

Estimating the incidence of stroke and ICH requires good quality case ascertainment over a long period of time using consistent, standardized methods. In the future, standardized electronic health records may provide “big data” for public health surveillance. In the present, we are fortunate to have high-quality prospective epidemiology studies such as the Brain Attack Surveillance in Corpus Christi (BASIC) project in Nueces County, Texas. Zahuranec et al.² use data from BASIC to report on trends in the incidence of ICH over time. Encouragingly, they find a 31% decrease in ICH incidence from 2000 to 2010. However, ICH is still a highly fatal disease, with 30-day case fatality approaching 40%. The lack of improvement in case fatality rate over time should be interpreted cautiously, as the study probably lacks adequate statistical power to detect improvements from better neurocritical care and stroke unit care.

There are 2 concerning findings. First, the declines in ICH incidence were limited to older patients; there was no decline in patients 45–59 years old. Second, Mexican American participants were at higher risk for ICH than white participants throughout the study period, even though incidence in both groups declined at a similar rate. One wonders whether ICH in younger patients has different risk factors and mechanisms; future research is needed to better define the epidemiology of ICH in younger (<60 years) people.

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AUTHOR CONTRIBUTIONS

Dr. Zahuranec: wrote initial draft of the manuscript, study concept/design, analysis/interpretation of data, acquisition of data. Dr. Lisabeth: study concept/design, analysis/interpretation of data, revision of manuscript for important content, obtained funding, study supervision. Dr. Sánchez: study concept/design, analysis/interpretation of data, revision of manuscript for important content, statistical analysis. Dr. Smith: revision of manuscript for important content, acquisition of data, study coordination. Dr. Brown: revision of manuscript for important content, acquisition of data. N.M. García: revision of manuscript for important content, acquisition of data, study coordination. Dr. Skolarus: revision of manuscript for important content, acquisition of data. Dr. Meurer: revision of manuscript for important content, acquisition of data. Dr. Burke: revision of manuscript for important content, acquisition of data. Dr. Adelman: revision of manuscript for important content, acquisition of data. Dr. Morgenstern: study concept/design, analysis/interpretation of data, revision of manuscript for important content, obtained funding, study supervision. All authors gave final approval of the submitted version.

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