Temporal trends in early case-fatality rates in patients with intracerebral hemorrhage

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ABSTRACT

Objective: To assess whether temporal trends in very early (within 48 hours) case-fatality rates may differ from those occurring between 48 hours and 30 days in patients with spontaneous intracerebral hemorrhage (ICH).

Methods: All cases of ICH that occurred in Dijon, France (151,000 inhabitants), were prospectively collected between 1985 and 2011, using a population-based registry. Time trends in 30-day case fatality were analyzed in 3 periods: 1985–1993, 1994–2002, and 2003–2011. Cox regression models were used to evaluate associations between time periods and case fatality within 48 hours and between 48 hours and 30 days, after adjustments for demographics, risk factors, severity, and ICH location.

Results: A total of 531 ICH cases were recorded (mean age 72.9 ± 15.8, 52.7% women). Thirty-day case fatality gradually decreased with time from 40.9% in 1985–1993 to 33.5% 1994–2002 and to 29.6% in 2003–2011 (adjusted hazard ratio [HR] 0.71, 95% confidence interval [CI] 0.47–1.07, $p = 0.106$, for 1994–2002, and adjusted HR 0.49, 95% CI 0.32–0.73, $p < 0.001$, for 2003–2011). Over the whole study period, 43.6% of 1-month deaths occurred within the first 48 hours following ICH onset. There was no temporal change in case fatality occurring within the first 48 hours but a decrease in deaths occurring between 48 hours and 30 days was observed with time (HR 0.53, 95% CI 0.31–0.90, $p = 0.02$, for 1994–2002, and HR 0.32, 95% CI 0.32–0.55, $p < 0.01$, for 2003–2011, compared with 1985–1993).

Conclusion: Although 30-day case fatality significantly decreased over the last 27 years, additional improvements in acute management of ICH are needed since very early case-fatality rates (within 48 hours) did not improve. ***Neurology*® 2017;88:985–990**

GLOSSARY

CI = confidence interval; **HR** = hazard ratio; **ICH** = intracerebral hemorrhage; **NIHSS** = NIH Stroke Scale.

Intracerebral hemorrhage (ICH) accounts for 10% of all symptomatic strokes in high-income countries, but the weight of hemorrhagic strokes in developing countries is much higher in terms of incidence and mortality rates.[1,2] While the incidence of ischemic strokes decreased in recent decades, that of spontaneous ICH did not.[3] In this context of stable incidence, a meta-analysis of available population-based studies concluded that early case-fatality rates of ICH did not improve from 1980 to 2008.[3] More recently, the BASIC project reported a worryingly stable 30-day case-fatality rate over the last 10 years.[4] These findings could suggest that ICH did not benefit from major improvements in acute medical treatment, but caution must be exercised before drawing any definite conclusions because of several limitations of available studies on this topic.

We hypothesized that case-fatality rates improved in the context of stroke unit care by reducing delayed post-ICH complications.[5] Since no major change in the acute treatment of ICH occurred over time, very early ICH case fatality may not have evolved.

Therefore, to assess this hypothesis, we used data from a prospective and continuous population-based registry to determine whether temporal trends in very early ICH case-fatality rates (within 48 hours) may differ from those occurring between 48 hours and 30 days.

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Supplemental data at Neurology.org

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METHODS

Study population and case ascertainment procedures. All first-ever cases of ICH occurring among people living in Dijon, France (152,000 inhabitants), were prospectively collected from 1985 to 2011 through the Dijon Stroke Registry.[6,7] To comply with quality criteria for stroke incidence studies,[8,9] this ongoing population-based registry use multiple overlapping sources of information to attempt to achieve an exhaustive collection of stroke cases, as previously described.[6,7] Hence, stroke patients are identified through hot and cold pursuit procedures based on both prospective and retrospective review of medical records of both clinical and radiologic departments of the University Hospital and the 3 private hospitals of Dijon, regular search for diagnostic codes of stroke through computerized hospital lists, and monthly consultation of computer-generated lists of all requests for imaging. Finally, investigators collaborate with general practitioners of the city to identify outpatients, and death certificates are regularly checked for ascertaining fatal strokes outside the hospital and determining vital status of collected patients. All medical files of patients suspected of having a stroke are reviewed by stroke-trained study investigators for adjudication.

ICH definition and location classification. WHO diagnostic criteria were used to define stroke.[10] For the present study, only first-ever spontaneous ICH were considered. All cases were documented by brain imaging. ICH related to a trauma, tumor, vascular malformation, or hemorrhagic transformation of a cerebral infarct were excluded. Based on the review of brain imaging data as previously described,[7] ICH location was classified as (1) lobar (frontal, temporal, parietal, insular, or occipital); (2) deep (originating from the lenticular or caudate nuclei, thalamus, or internal or external capsule); or (3) infratentorial (originating from brainstem or cerebellum). Cases for which ICH location was uncertain were discussed by the investigators so as to define the most probable origin of bleeding, and ICH was classified as undetermined when the origin remained uncertain, if hemorrhages overlapped 2 territories, or when data were missing.

Data collected. The following vascular risk factors were collected with the same methodology over the whole study period, based on interview of patients, their relatives, of their general practitioners, and consultation of medical files[6,7]: hypertension, diabetes mellitus, hypercholesterolemia, atrial fibrillation, history of coronary heart disease, peripheral artery disease, heart failure, cancer, alcohol intake, smoking, and previous TIA.

Treatments prior to stroke were recorded: oral anticoagulants (warfarin, acenocoumarol, or fluindione), antiplatelet agents (aspirin, clopidogrel, ticlopidine, or dipyridamole), and antihypertensive treatment. Statins were not included because they were only recorded from 2006 onwards. Prestroke residence in a nursing home was used as a proxy of dependence. Stroke severity at admission was evaluated using clinical features (motor impairment, aphasia, and decreased consciousness corresponding to patients with either drowsiness or coma), since systematic recording of the NIH Stroke Scale (NIHSS) score was only introduced in 2006. We previously demonstrated a good correlation between these proxies and the NIHSS score.[11]

Case fatality assessment. The outcome was all-cause case fatality at 1 month (30 days) based on death certificate collection. Information was available for every patient.

Statistical analysis. Proportions and mean values of baseline characteristics were compared between groups using the χ^2 test, and the Wilcoxon-Mann-Whitney test when appropriate. Person-days were calculated from the date of ICH onset until death, the last contact date, and the end of follow-up at 30 days. Survival curves were obtained using Kaplan-Meier analysis. Cox regression models were used to estimate hazard ratios (HRs) of 30-day case fatality and their 95% confidence intervals (CIs). In the first multivariable analysis model, we introduced age, sex, and time period (1985–1993, 1994–2002, and 2003–2011). Additional models were performed by successively introducing baseline characteristics (risk factors and premorbid treatments), ICH location, intraventricular extension of ICH, and proxies of severity if p value was <0.20 in unadjusted models. So as to evaluate temporal trends in very early case fatality vs later death, separate analyses for deaths occurring within the first 48 hours following stroke, and time period from 48 hours to 30 days, were then performed using the same procedure. A dummy indicator for smoking status was used because of missing data in 17.5% of cases for this variable. The corresponding proportion for other variables was less than 2%. p Values <0.05 were considered statistically significant. STATA@ 9.0 software (StataCorp LP, College Station, TX) was used for the statistical analysis.

Standard protocol approvals, registrations, and patient consents. The Dijon Stroke Registry was approved by the Comité d'Evaluation des Registres (French National Committee of Registers).

RESULTS

Characteristics of the ICH cohort. Over the 27-year study period, 4,650 first-ever strokes were recorded, including 531 ICH (11.4%, mean age 72.9 ± 15.8 years, 52.7% women). The proportion of ICH among stroke cases did not change over time (11.2% in 1985–1993 vs 11.7% in 1994–2002 vs 11.4% in 2003–2011, $p = 0.91$). Baseline characteristics of ICH patients according to study periods are shown in table 1. Mean age at onset increased between the first and the 2 last study periods. Some variations in the prevalence of other risk factors and premorbid treatments were also observed. In contrast, no significant changes in clinical features including proxies of stroke severity and place of admission were noted over time. Regarding ICH location, 262 were lobar (49.3%), 201 were deep (37.9%), 58 were infratentorial (10.9%), and 10 were undetermined (1.9%). The distribution of ICH location remained stable over time.

Trends in 30-day case-fatality rates. During the whole study period, the 30-day case-fatality rate was 34.1% (95% CI 30.0–38.1). Case-fatality rates were 40.9% (95% CI 32.9–48.9) in 1985–1993, 33.5% (95% CI 26.5–40.6) in 1994–2002, and 29.6% (95% CI 23.3–35.9) in 2003–2011 (figure 1). In univariate analyses, compared with the first time period, 2003–2011 was associated with a lower 30-day case-fatality rate (HR 0.69, 95% CI 0.48–0.98, $p = 0.037$), but not for 1994–2002 (HR 0.80, 95% CI 0.56–1.14, $p = 0.21$). After adjustment for confounding variables, a gradual decrease in 30-day case fatality was observed with

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Table 1 Baseline characteristics of intracerebral hemorrhage (ICH) patients according to time periods

	1985-1993 (n = 149)		1994-2002 (n = 176)		2003-2011 (n = 206)		
	No. (%)	95% CI	No. (%)	95% CI	No. (%)	95% CI	p
Age, y, mean ± SD	68.9 ± 16.8		74.0 ± 14.4		74.7 ± 15.9		0.002
Male sex	69 (46.3)	38.2-54.4	85 (48.3)	40.8-55.7	97 (47.1)	40.2-54.0	0.94
Hypertension	88 (59.1)	51.1-67.0	107 (60.8)	53.5-68.1	140 (68.0)	61.5-74.4	0.17
Diabetes	15 (10.1)	5.2-15.0	30 (17.1)	11.4-22.7	31 (15.1)	10.1-20.0	0.19
Hypercholesterolemia	13 (8.7)	4.1-13.3	39 (22.2)	16.0-28.4	40 (19.4)	14.0-24.9	0.004
Atrial fibrillation	15 (10.1)	5.2-15.0	40 (22.7)	16.5-29.0	37 (17.0)	11.8-22.2	0.01
Smoking							<0.001
Yes	41 (27.5)	20.3-34.8	34 (19.3)	13.4-25.2	55 (26.7)	20.6-32.8	
No	95 (63.8)	55.9-71.6	75 (42.6)	35.2-50.0	138 (67.0)	60.5-73.5	
Unknown	13 (8.7)	4.1-13.3	67 (38.1)	30.8-45.3	13 (6.3)	3.0-9.8	
Coronary artery disease	16 (10.7)	5.7-15.8	24 (13.6)	8.5-18.8	18 (8.7)	4.8-12.6	0.31
TIA	22 (14.8)	9.0-20.5	18 (10.2)	5.7-14.7	12 (5.8)	2.6-9.1	0.019
Peripheral artery disease	10 (6.7)	2.6-10.8	14 (8.0)	3.9-12.0	14 (6.8)	3.3-10.3	0.88
Alcohol	20 (13.4)	7.9-19.0	17 (9.7)	5.3-14.1	24 (11.7)	7.2-16.1	0.57
Heart failure	7 (4.7)	1.2-8.1	13 (7.4)	3.5-11.3	25 (12.1)	7.6-16.6	0.037
Cancer	6 (4.0)	0.8-7.2	10 (5.7)	2.2-9.1	16 (7.8)	4.1-11.5	0.33
Premorbid treatments							
Antiplatelet agents	12 (8.1)	3.6-12.5	44 (25.0)	18.5-31.4	31 (15.1)	10.1-20.0	<0.001
Anticoagulants	6 (4.0)	0.8-7.2	22 (12.5)	7.6-17.4	39 (18.9)	13.5-24.3	<0.001
Antihypertensive treatments	58 (38.9)	31.0-46.8	86 (48.9)	41.4-56.3	80 (38.8)	32.1-45.5	0.09
Living in a nursing home	10 (6.7)	2.6-10.8	10 (5.7)	2.2-9.1	10 (4.9)	1.9-7.8	0.76
Clinical features							
Motor impairment	114 (76.5)	69.6-83.4	138 (78.8)	72.7-85.0	158 (77.5)	71.7-85.0	0.87
Aphasia	53 (35.6)	27.8-43.3	67 (38.3)	31.0-45.6	62 (30.7)	24.3-37.1	0.29
Decreased level of consciousness	91 (61.1)	53.2-69.0	85 (48.3)	40.8-55.8	110 (53.4)	46.5-60.3	0.07
ICH location (n = 521)							0.13
Lobar	69 (46.3)	38.2-54.4	89 (50.6)	43.1-58.0	104 (50.5)	43.6-57.4	
Deep	52 (34.9)	27.2-42.6	68 (38.6)	31.4-45.9	81 (39.3)	32.6-46.0	
Infratentorial	20 (13.4)	7.9-19.0	17 (9.7)	5.2-14.1	21 (10.2)	6.0-14.4	
Undetermined	8 (5.4)	1.7-9.0	2 (1.1)	0.0-2.7	0 (0)	—	
Intraventricular extension (n = 519)	21 (14.1)	8.9-20.8	34 (19.3)	13.7-25.6	66 (32.0)	25.7-38.6	<0.001
Place of admission							0.36
University teaching hospital	134 (89.9)	85.0-94.8	163 (92.6)	88.7-94.8	187 (90.8)	86.8-94.8	
Private hospital	9 (6.1)	2.2-9.9	6 (3.4)	0.7-6.1	15 (7.3)	3.7-10.9	
Outpatients	6 (4.0)	0.8-7.2	7 (4.0)	1.1-6.9	4 (1.9)	0.0-4.0	

Abbreviation: CI = confidence interval.

time, although only statistically significant for 2003–2011 (HR 0.71, 95% CI 0.47–1.07, $p = 0.106$ for 1994–2002, and HR 0.49, 95% CI 0.32–0.73, $p < 0.001$ for 2003–2011) (table 2).

Trends in very early vs later case-fatality rates. Over the whole study period, 43.6% of deaths at 30 days occurred within the first 48 hours following ICH onset. Case-fatality rates by study periods and time of death are shown in table 3. Case fatality occurring within the first 48 hours did not change over time, whereas case fatality occurring between 48 hours and 30 days decreased markedly (25.5% for 1985–1993 vs 19.3% for 1994–2002 vs 14.6% for 2003–2011). In multivariable analyses, there was no temporal change in case fatality occurring within the

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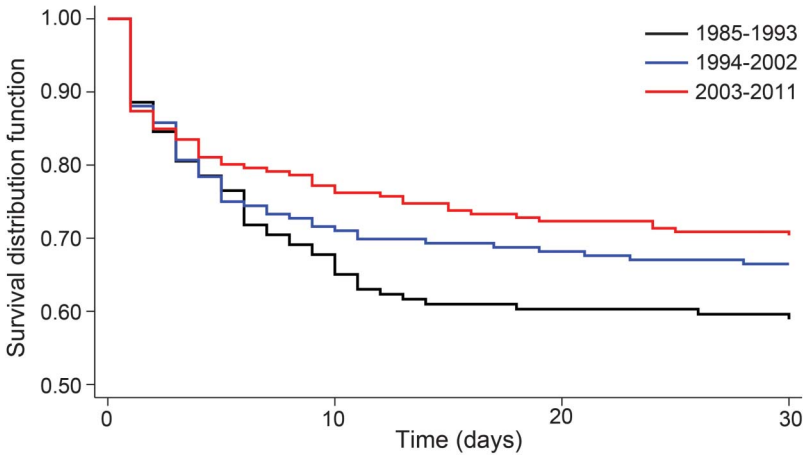
Figure 1 Kaplan-Meier estimates of 30-day survival stratified by time periods



first 48 hours (table 4 and table e-1 at Neurology.org). Conversely, a decrease in death occurring between 48 hours and 30 days was observed with time (HR 0.53, 95% CI 0.31–0.90, $p = 0.02$ for 1994–2002 and HR 0.32, 95% CI 0.32–0.55, $p < 0.01$ for 2003–2011, compared with 1985–1993) (tables 4 and e-1). The proportion of patients with comatose state on admission did not change over time (figure e-1, p for χ^2 test = 0.08). Moreover, among these patients, the distribution of outcomes at 30 days remained unchanged (p for χ^2 test = 0.13).

Table 2 Association between time period and 30-day case mortality in multivariable Cox analysis

	HR	95% CI	p
Model 1[a]			
1985-1993	Ref.		
1994-2002	0.72	0.50-1.03	0.068
2003-2011	0.59	0.41-0.85	0.004
Model 2[b]			
1985-1993	Ref.		
1994-2002	0.58	0.39-0.85	0.006
2003-2011	0.55	0.38-0.79	0.002
Model 3[c]			
1985-1993	Ref.		
1994-2002	0.56	0.38-0.84	0.006
2003-2011	0.42	0.28-0.62	<0.001
Model 4[d]			
1985-1993	Ref.		
1994-2002	0.71	0.47-1.07	0.106
2003-2011	0.49	0.32-0.73	<0.001

Abbreviations: CI = confidence interval; HR = hazard ratio.
[a] Adjusted for age and sex (n = 531).
[b] Adjusted for age, sex, risk factors, and premorbid treatments with $p < 0.2$ in univariate analyses: hypercholesterolemia, atrial fibrillation, alcoholism, smoking status, peripheral arterial disease, anticoagulants (n = 531).
[c] Model 2 plus intraventricular extension and ICH location (n = 519).
[d] Model 3 plus proxies of severity: motor impairment and decreased level.

DISCUSSION ICH remains a life-threatening event: 1 patient in 3 will die within the first month following ICH onset. Our population-based study demonstrated that 30-day case fatality decreased over the last 27 years. Although encouraging, this trend should not mask the fact that additional improvements in acute management are needed since we showed for the first time that very early case-fatality rates (within 48 hours) did not decrease over time.

This large population-based study on ICH was conducted owing to a prospective and continuous case collection in a geographically well-defined population. Data collection was based on overlapping sources of information so as to attempt to ensure an exhaustive case ascertainment.[7] Because of low migration, the population of Dijon was very stable over time. As a result, changes in ethnic mix and economic status of residents were limited, thus avoiding potential biases. However, several limitations must be considered for interpretation of the results. It could be argued that potential improvements in diagnostic procedures may have led to better identification of patients with minor ICH, and thus contributed in part to the observed better prognosis of patients. Although information about the NIHSS score was not available for the whole study period to verify this assumption, comparisons between the prevalence of the proxies of initial severity of ICH by study periods, including motor impairment, aphasia, and decreased level of consciousness, did not reveal any significant change in the clinical profile of ICH patients over time. In multivariable Cox models, the associations observed between both early and delayed case fatality and study periods were consistent after introducing these proxies of severity, thus suggesting that there was no major change in the severity of recorded ICH. Moreover, access to brain imaging of stroke patients in the Dijon Stroke Registry has been excellent since the beginning of the study. Among all first-ever strokes recorded during our study, the proportion of patients who underwent brain imaging for diagnosis ranged from 96.3% during the first period to 99.6% during the last one. Hence, these observations argue against a major contribution of changes in diagnostic procedures in the observed findings. We had no prospective data about the proportion of patients who underwent hematoma evacuation, although such a procedure was uncommon in local practice. Finally, no information about changes in time to hospital arrival was available, thus preventing us from analyzing the effectiveness of stroke care pathway.

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Table 3 Case-fatality rates by study periods and time of death

	1985–1993 (n = 149), % (95% CI)	1994–2002 (n = 176), % (95% CI)	2003–2011 (n = 206), % (95% CI)	Overall % (95% CI)
Overall 30-day	40.9 (32.9–48.9)	33.5 (26.5–40.6)	29.6 (23.3–35.9)	34.1 (30.0–38.1)
Deaths occurring within the first 48 hours	15.4 (9.6–21.3)	14.2 (9.0–19.4)	15.1 (10.1–20.0)	14.9 (11.8–17.9)
Deaths occurring between 48 hours and 30 days	25.5 (18.4–32.6)	19.3 (13.4–25.2)	14.6 (9.7–19.4)	19.2 (15.8–22.6)

Abbreviation: CI = confidence interval.

In a recent review and meta-analysis of population-based studies, stable case-fatality rates after ICH were reported between 1980 and 2008.[3]

Table 4 Associations between study periods and case fatality occurring within the first 48 hours and between 48 hours and 30 days in patients with ICH

	HR	95% CI	p
Death occurring within the first 48 hours			
Univariate analysis			
1985–1993	Ref.		
1994–2002	0.92	0.52–1.62	0.78
2003–2011	0.98	0.57–1.68	0.93
Model 1[a]			
1985–1993	Ref.		
1994–2002	0.77	0.40–1.47	0.42
2003–2011	0.66	0.36–1.22	0.18
Model 2[b]			
1985–1993	Ref.		
1994–2002	0.94	0.49–1.81	0.87
2003–2011	0.78	0.42–1.42	0.42
Death occurring between 48 hours and 30 days			
Univariate analysis			
1985–1993	Ref.		
1994–2002	0.72	0.45–1.14	0.17
2003–2011	0.63	0.32–0.84	0.007
Model 3[c]			
1985–1993	Ref.		
1994–2002	0.43	0.25–0.72	0.001
2003–2011	0.29	0.17–0.48	<0.001
Model 4[d]			
1985–1993	Ref.		
1994–2002	0.53	0.31–0.90	0.019
2003–2011	0.32	0.19–0.55	<0.007

Abbreviations: CI = confidence interval; HR = hazard ratio.
[a] Adjusted for age, sex, hypercholesterolemia, atrial fibrillation, smoking status, anticoagulants, ICH location, and intraventricular extension.
[b] Model 2 plus proxies of severity: motor impairment and decreased level of consciousness.
[c] Adjusted for age, sex, smoking status, coronary artery disease, peripheral artery disease, TIA, and intraventricular extension.
[d] Model 3 plus proxies of severity: motor impairment, aphasia, and decreased level of consciousness.

However, the authors were careful in their conclusions because of several limitations. Hence, most studies were performed during the 1990s, i.e., before the implementation of organized inpatient (stroke unit) care. Furthermore, longitudinal data about ICH case fatality were scarce, and comparisons were made according to crude case-fatality rates obtained from different studies conducted at different time points, irrespective of changes in the case mix of studied populations and prognostic factors, including variations in mean age and prevalence of baseline vascular risk factors. Since these potential changes in characteristics of studied populations may act as confounding variables in the interpretation of temporal trends in case-fatality rates after ICH, adjustments were made for these variables in the present study. In line with our overall decline in 30-day case fatality, the same trend was found in the Netherlands between 1980 and 2010 in patients younger than 75 years,[12] as well as in the United Kingdom.[13] However, data on very early mortality were not available. However, in the South Texas study, 30-day case fatality did not decline, but the period of observation was shorter (10 years vs 30 years in our study) and data on very early death were not studied.[4]

In our study, the better survival following ICH was explained by a decrease in deaths occurring between 48 hours and 30 days. In contrast, very early case fatality did not improve over time, and accounted for an increasing proportion of overall deaths, from 38% to 51%, although in-house management protocols in our setting were in line with the French Neurovascular Society recommendations, and since 2006 with European Guidelines (European Stroke Initiative/European Stroke Organisation).[14] This result illustrates the fact that effective hyperacute treatment of ICH patients needs to be developed. Until now, strategies to reduce ICH growth did not improve outcomes, with the exception of the recent Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial–II trial, the results of which were published after the end of our observation period.[15] Therefore, it cannot be excluded that the absence of acute curative therapy for severe ICH may have influenced the decision-making process of doctors and family with regard to the limitation of

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aggressive life-sustaining measures. In line with this observation, a previous study concluded that early do not resuscitate orders were associated with a greater risk of death in ICH patients, despite adjustment for predictors of poor outcome.[16] This result raised the question of self-fulfilling prophecy to account for reluctance of clinicians to offer aggressive care in patients with severe ICH. Conversely, although data about causes of death were not available in our registry, it could be assumed that the better outcome of ICH patients who survived this hyperacute stage probably highlights improvements in care, including the implementation of dedicated stroke networks, organized intensive care units,[5] and the development of guidelines dedicated to the management of ICH patients.[17]

Despite some improvements over the last 30 years, ICH remains a life-threatening event. None of our strategies has managed to improve very early mortality. While primary prevention remains the most valuable strategy to reduce the burden of ICH, very early interventions, including prehospital mobile stroke units, may be promising.

AUTHOR CONTRIBUTIONS

Yannick Béjot: study concept and design, acquisition of data, analysis and interpretation of data, study supervision, obtaining funding, drafting/revising the manuscript for content. Michael Grelat: analysis and interpretation of data, critical revision of manuscript for intellectual content. Benoit Delpont: acquisition of data, analysis and interpretation of data, critical revision of manuscript for intellectual content. Jérôme Durier: acquisition of data, analysis and interpretation of data. Olivier Rouaud: acquisition of data, analysis and interpretation of data, critical revision of manuscript for intellectual content. Guy-Victor Osseby: acquisition of data, analysis and interpretation of data, critical revision of manuscript for intellectual content. Marie Hervieu-Bègue: acquisition of data, analysis and interpretation of data, critical revision of manuscript for intellectual content. Maurice Giroud: study concept and design, acquisition of data, obtaining funding, analysis and interpretation of data, critical revision of manuscript for intellectual content. Charlotte Cordonnier: study concept and design, analysis and interpretation of data, drafting/revising the manuscript for content.

STUDY FUNDING

The Dijon Stroke Registry is supported by the French Institute for Public Health Surveillance (InVS) and Institut National de la Santé et de la Recherche Médicale (INSERM).

DISCLOSURE

Y. Béjot received honoraria for participation in advisory boards or as a symposium speaker for AstraZeneca France, Daiichi-Sankyo, Pfizer, Covidiem, and MSD France. M. Grelat, B. Delpont, J. Durier, O. Rouaud, G. Osseby, and M. Hervieu-Bègue report no disclosures relevant to the manuscript. M. Giroud received honoraria for participation in advisory boards for Daiichi-Sankyo. C. Cordonnier was an investigator in clinical trials for Pfizer and Astra-Zeneca and participated in advisory boards for Bayer and Medtronic. Fees were paid to ADRINORD or Lille University Hospital research account (no personal funding). Go to Neurology.org for full disclosures.

Received May 2, 2016. Accepted in final form October 25, 2016.

REFERENCES

1. Qureshi AI, Mendelow AD, Hanley DF. Intracerebral haemorrhage. Lancet 2009;373:1632–1644.
2. Feigin VL, Krishnamurthi RV, Parmar P, et al. Update on the global burden of ischemic and hemorrhagic stroke in 1990-2013: the GBD 2013 study. Neuroepidemiology 2015;45:161–176.
3. van Asch CJ, Luitse MJ, Rinkel GJ, van der Tweel I, Algra A, Klijn CJ. Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. Lancet Neurol 2010;9:167–176.
4. Zahuranec DB, Lisabeth LD, Sánchez BN, et al. Intracerebral hemorrhage mortality is not changing despite declining incidence. Neurology 2014;82:2180–2186.
5. Langhorne P, Fearon P, Ronning OM, et al. Stroke unit care benefits patients with intracerebral hemorrhage: systematic review and meta-analysis. Stroke 2013;44:3044–3049.
6. Béjot Y, Daubail B, Jacquin A, et al. Trends in the incidence of ischaemic stroke in young adults between 1985 and 2011: the Dijon Stroke Registry. J Neurol Neurosurg Psychiatry 2014;85:509–513.
7. Béjot Y, Cordonnier C, Durier J, Aboa-Eboulé C, Rouaud O, Giroud M. Intracerebral haemorrhage profiles are changing: results from the Dijon population-based study. Brain 2013;136:658–664.
8. Malmgren R, Warlow C, Bamford J, Sandercock P. Geographical and secular trends in stroke incidence. Lancet 1987;2:1196–1200.
9. Sudlow CL, Warlow CP. Comparing stroke incidence worldwide: what makes studies comparable? Stroke 1996;27:550–558.
10. WHO. The World Health Report 2000: Health Systems Improving Performance. Geneva: WHO; 2000.
11. Grimaud O, Leray E, Lalloué B, et al. Mortality following stroke during and after acute care according to neighbourhood deprivation: a disease registry study. J Neurol Neurosurg Psychiatry 2014;85:1313–1318.
12. Jolink WM, Klijn CJ, Brouwers PJ, Kappelle LJ, Vaartjes I. Time trends in incidence, case fatality, and mortality of intracerebral hemorrhage. Neurology 2015;85:1318–1324.
13. González-Pérez A, Gaist D, Wallander MA, McFeat G, García-Rodríguez LA. Mortality after hemorrhagic stroke: data from general practice (The Health Improvement Network). Neurology 2013;81:559–565.
14. Steiner T, Kaste M, Forsting M, et al. Recommendations for the management of intracranial haemorrhage: part I: spontaneous intracerebral haemorrhage. Cerebrovasc Dis 2006;22:294–316.
15. Anderson CS, Heeley E, Huang Y, et al. Rapid blood-pressure lowering in patients with acute intracerebral hemorrhage. N Engl J Med 2013;368:2355–2365.
16. Zahuranec DB, Brown DL, Lisabeth LD, et al. Early care limitations independently predict mortality after intracerebral hemorrhage. Neurology 2007;68:1651–1657.
17. Morgenstern LB, Hemphill JC III, Anderson C, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2010;41:2108–2129.

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