# Time trends in outcome of subarachnoid hemorrhage

## Population-based study and systematic review

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## ABSTRACT

**Background:** Treatment of aneurysmal subarachnoid hemorrhage (SAH) has changed substantially over the last 25 years but there is a lack of reliable population-based data on whether case-fatality or functional outcomes have improved.

**Methods:** We determined changes in the standardized incidence and outcome of SAH in the same population between 1981 and 1986 (Oxford Community Stroke Project) and 2002 and 2008 (Oxford Vascular Study). In a meta-analysis with other population-based studies, we used linear regression to determine time trends in outcome.

**Results:** There were no reductions in incidence of SAH (RR = 0.79, 95% confidence interval [CI] 0.48–1.29, $p = 0.34$) and in 30-day case-fatality (RR = 0.67, 95% CI 0.39–1.13, $p = 0.14$) in the Oxford Vascular Study vs Oxford Community Stroke Project, but there was a decrease in overall mortality (RR = 0.47, 0.23–0.97, $p = 0.04$). Following adjustment for age and baseline SAH severity, patients surviving to hospital had reduced risk of death or dependency (modified Rankin score > 3) at 12 months in the Oxford Vascular Study (RR = 0.51, 0.29–0.88, $p = 0.01$). Among 32 studies covering 39 study periods from 1980 to 2005, 7 studied time trends within single populations. Unadjusted case-fatality fell by 0.9% per annum (0.3–1.5, $p = 0.007$) in a meta-analysis of data from all studies, and by 0.9% per annum (0.2–1.6%, $p = 0.01$) within the 7 population studies.

**Conclusion:** Mortality due to subarachnoid hemorrhage fell by about 50% in our study population over the last 2 decades, due mainly to improved outcomes in cases surviving to reach hospital. This improvement is consistent with a significant decrease in case-fatality over the last 25 years in our pooled analysis of other similar population-based studies. ***Neurology*** ® **2010;74:1494–1501**

### GLOSSARY

**CI** = confidence interval; **mRS** = modified Rankin score; **OCSP** = Oxford Community Stroke Project; **OXVASC** = Oxford Vascular Study; **SAH** = subarachnoid hemorrhage; **WFNS** = World Federation of Neurosurgical Societies.

Several systematic reviews based on separate observational studies have tended to show declining trends in case-fatality following subarachnoid hemorrhage (SAH) over the last 3 decades.[1-4] If real, these trends have important implications for planning clinical services. SAH occurs at a relatively young age compared to other stroke types; if more patients survive SAH but are severely disabled, the burden of post-SAH care will increase considerably. There may also be consequences for the cost-effectiveness of screening programs for unruptured aneurysms. If SAH outcomes are improving, conservative treatment may become preferable for some patient groups in which the risks and benefits of treating unruptured aneurysms are very closely matched.[5]

However, indirect estimation of apparent time trends from multiple separate studies performed at different times is subject to several sources of bias, including differences between

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studies in case-finding and diagnostic criteria, differences in the age and sex distributions of each study population, and advances in diagnostic techniques over time. The most reliable method for determining time trends in outcomes is repeated studies within the same population over time, using consistent case finding methods and diagnostic criteria,[6] but few such studies have been reported and to our knowledge there have been no recent population-based studies that have correlated trends in case-fatality with more detailed analyses of changes in treatment and functional outcomes. We therefore studied time trends in SAH outcomes by determining changes in outcome over time in our own population-based study, and by pooling our results with other studies of time trends within single populations.

**METHODS** **Oxford Vascular Study and Oxford Community Stroke Project.** All cases of SAH which were first-ever strokes were ascertained from the first 6 years (April 2002–March 2008) of the Oxford Vascular Study (OXVASC), the methods of which have been described previously.[7] In brief, the OXVASC study is a prospective population-based study of all vascular events including stroke, occurring in a population of 91,106 individuals, registered with 63 general practitioners in 9 primary care practices in Oxfordshire, all of which had participated in the Oxford Community Stroke Project (OCSP).[8] Only 2 practices involved in OCSP could not be included in OXVASC, and data from all 4 years of the OCSP study (1981–1984, 1986) have been reanalyzed for this comparison, comprising a mid-study population estimate of 87,861 individuals that excludes cases from these 2 practices.

We reviewed case records, postmortem reports, and brain imaging for all patients diagnosed with SAH in both OCSP and OXVASC. SAH was diagnosed in the context of a consistent clinical presentation and 1) brain imaging showing subarachnoid blood or 2) an adequate postmortem examination or 3) the presence of xanthochromia in a CSF sample.[8] SAH associated with primary intracerebral hemorrhage, or secondary to trauma, drug use, arterial dissection, or vasculitis, was excluded. The overall rate of imaging and/or autopsy was 98% in OXVASC and 89% in OCSP.

In each case, we derived the World Federation of Neurosurgical Societies (WFNS) score[9] using details of the initial clinical presentation, and collected data on vascular imaging and treatment. Functional outcomes at 12 months post SAH were assessed in both studies using the modified Rankin score (mRS). Premorbid risk factor and medication data were obtained by questioning patients and relatives, and by reviewing medical records.

We calculated the incidence rates of SAH in both studies, and standardized these to the 2001 census population of England and Wales. Confidence intervals for incidence were derived assuming a Poisson distribution for the number of events. Poisson regression models adjusted for overdispersion were used to produce the relative incidence of all SAH, and the relative

30-day case-fatality following SAH in OXVASC vs OCSP after adjustment for age and sex. The relative risk of death or dependency (mRS $\geq$ 3) at 12 months, adjusted for age and clinical severity of SAH at presentation (dichotomized as WFNS score at presentation $\leq$3 or >3), was calculated in a similar way. Clinical and risk factor data from OXVASC and OCSP were compared using Fisher exact test and Student t test.

**Standard protocol approvals, registrations, and patient consents.** OXVASC has local research ethics committee approval and written informed consent or assent is obtained for all patients participating in the study.

**Systematic review.** We performed a MEDLINE search using combinations of the following search terms: "subarachnoid hemorrhage/haemorrhage" or "stroke," and "outcome" or "mortality" or "case-fatality," and "population" or "community" or "epidemiology," limited to studies publishing outcome data from 1980 onwards, to include the same period of observation as our population-based study. The reference lists of retrieved studies were also searched. Studies were included if they met the following criteria: 1) use of a population-based study design in which the study population was representative of the population in general, and all death certificates had been reviewed or all nonhospitalized deaths had been reviewed with the coroner; 2) the upper age limit of the study was not <75 years and the lower limit was not >35 years; 3) results for SAH were reported separately if the study was about stroke in general; 4) brain imaging or autopsy was performed in at least 80% of the population; 5) study period was not longer than 10 years unless separate results were given per decade; 6) crude case-fatality rates were given or could be calculated from the data presented; 7) there were at least 5 cases of SAH in any study period. We also included our own data from OXVASC and OCSP.

For each study we recorded the mid-calendar year of the study, total numbers with SAH, crude case-fatality rates, time of follow-up, age and sex distribution of the population with SAH, and the percentage with brain imaging or autopsy. We used linear regression analysis to describe the relationship between crude case-fatality rates and the mid-calendar year of each included study, weighted by the inverse of the standard error of the case-fatality of each study. Age and sex differences between studies were taken into account by entering the mean age and percentage of women in the study into the weighted linear regression model, when these data were available.

Time trends within single populations were also analyzed using outcome data taken at the 2 furthest timepoints within each study, which were required to be at least 5 years apart. The results are expressed as the absolute percentage change in case-fatality per calendar year.

**RESULTS** **OXVASC and OCSP.** In the first 6 years of OXVASC there were 883 incident strokes, of which 38 (4.3%) were due to SAH. Of the incident cases, 2 died prior to reaching hospital and were diagnosed at postmortem, 35 were diagnosed on brain imaging, and 1 was diagnosed on the basis of CSF xanthochromia with a consistent clinical presentation but negative brain imaging.

Over a period of 4 years in OCSP, there were 557 incident strokes in the population corresponding to OXVASC, and 27 (5.0%) were due to SAH. One patient died before reaching hospital and was diag-

**Table 1** Age and sex structure of the OXVASC and OCSP study populations and crude incidence per 1,000 population of incident SAH[a]

| Age, years | Men | | Women | | Total | |
|---|---|---|---|---|---|---|
| | No./no. at risk | Rate (95%CI) | No./no. at risk | Rate (95%CI) | No./no. at risk | Rate (95%CI) |
| **OXVASC** | | | | | | |
| <35 | 0/22,581 | — | 1/20,273 | 0.01 (0.00–0.05) | 1/42,854 | 0.00 (0.00–0.02) |
| 35-54 | 4/13,607 | 0.05 (0.01–0.13) | 10/12,000 | 0.14 0.07–0.26) | 14/25,607 | 0.09 (0.05–0.15) |
| 55-74 | 3/8,426 | 0.06 (0.01–0.17) | 14/8,300 | 0.28 (0.15–0.47) | 17/16,726 | 0.17 (0.10–0.27) |
| 75+ | 1/2,356 | 0.07 (0.00–0.39) | 5/3,563 | 0.23 (0.08–0.55) | 6/5,919 | 0.17 (0.06–0.37) |
| Total | 8/46,970 | 0.03 (0.01–0.06) | 30/44,136 | 0.11 (0.08–0.16) | 38/91,106 | 0.07 (0.05–0.10) |
| **OCSP** | | | | | | |
| <35 | 0/25,267 | — | 2/23,024 | 0.02 (0.00–0.08) | 2/48,291 | 0.01 (0.00–0.04) |
| 35-54 | 4/10,857 | 0.09 (0.03–0.24) | 3/10,546 | 0.07 (0.01–0.21) | 7/21,403 | 0.08 (0.03–0.17) |
| 55-74 | 3/6,762 | 0.11 (0.02–0.32) | 8/7,181 | 0.28 (0.12–0.55) | 11/13,943 | 0.20 (0.10–0.35) |
| 75+ | 1/1,485 | 0.17 (0.00–0.94) | 6/2,739 | 0.55 (0.20–1.19) | 7/4,224 | 0.41 (0.17–0.85) |
| Total | 8/44,371 | 0.05 (0.02–0.09) | 19/43,490 | 0.11 (0.07–0.17) | 27/87,861 | 0.08 (0.05–0.11) |

Abbreviations: CI = confidence interval; OCSP = Oxford Community Stroke Project; OXVASC = Oxford Vascular Study; SAH = subarachnoid hemorrhage.
[a]Population denominators refer to the whole study population without exclusions.

nosed at postmortem. Of the remaining cases surviving to reach hospital, 3 cases were diagnosed at postmortem, 18 were diagnosed on brain and vascular imaging, and 5 were diagnosed on the basis of having a typical clinical presentation plus the presence of CSF xanthochromia.

The age and sex structure of the study populations and crude incidence per 1,000 of SAH are given in table 1. After adjustment to the 2001 census population of England and Wales, the overall incidence of SAH in OXVASC was 0.07 (0.05–0.10) and in OCSP it was 0.10 (0.06–0.13). The decrease in SAH incidence was not significant (rate ratio = 0.79, 95% confidence interval [CI] 0.48–1.29, $p = 0.34$).

Demographic and premorbid clinical risk factor data for cases with SAH in OXVASC and OCSP are shown in table e-1 on the *Neurology*® Web site at www.neurology.org. The mean age and sex distribution of patients in both studies were similar. There was a nonsignificant fall in the proportion of current smokers in OXVASC compared to OCSP, and in the proportion of patients with a past history of hypertension or on treatment for hypertension.

Although SAH severity (distribution of WFNS grades at presentation) was comparable in OXVASC and OCSP (figure e-1), a greater proportion of patients surviving to reach hospital underwent vascular imaging in OXVASC compared to OCSP (24 [67%] vs 6 [23%], $p = 0.001$), and treatment to secure aneurysms (18 [50%] vs 5 [19%], $p = 0.02$). The median delay (IQR) in days to any interventional

treatment was also shorter in OXVASC (2 [1–5] vs 14 [11–16], $p = 0.001$). In OCSP, all 5 treated aneurysms were secured by surgical clipping, whereas in OXVASC, 15/18 (83%) treated aneurysms were secured by endovascular embolization.

Age- and sex-adjusted 30-day case fatality tended to be lower in OXVASC vs OCSP (43% vs 67%, RR = 0.67, 95% CI 0.39–1.13, $p = 0.14$), contributing to a 53% reduction in age and sex-standardized mortality rates between the 2 study periods (RR =

**Figure 1** Twelve-month modified Rankin Scale scores (mRS) in patients surviving to reach hospital in Oxford Community Stroke Project (OCSP) and Oxford Vascular Study (OXVASC)
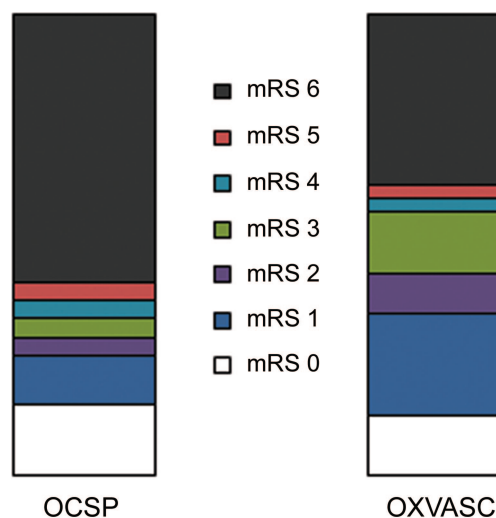
**Table 2**     Characteristics of population-based studies reporting case-fatality after SAH included in the systematic review

| Region | Midyear | Duration | Type | %CT +/− PM | No. of SAH | % Case fatality | Time (days) | Mean age | % Female |
|---|---|---|---|---|---|---|---|---|---|
| South Alabama, US[12] | 1980 | 1980 | All stroke | 80 | 9 | 67 | 14 | 55 | 100 |
| Izumo, Japan[d][14] | 1994 | 90-98 | SAH | 98 | 188 | 36 | 180 | 65 | — |
| Finland[c][15] | 1987 | 83-92 | All stroke | ≥80 | 969 | 47 | 28 | — | 48 |
| Cincinnati, US[17] | 1988 | 1988 | SAH | 86 | 80 | 45 | 30 | — | 61 |
| Washington, US[18] | 1988 | 87-89 | SAH | 97 | 171 | 32 | 30 | — | 65 |
| Devon & Cornwall, UK[22] | 1994 | 92-96 | SAH | 99 | 800 | 44 | 30 | 61 | 64 |
| L'Aquila, Italy[23] | 1994 | 1994 | All stroke | 89 | 24 | 29 | 30 | 61 | 58 |
| Erlangen, Germany[24] | 1995 | 94-96 | All stroke | 95 | 12 | 50 | 28 | 55 | 67 |
| Northern Manhattan, US[25] | 1995 | 93-97 | SAH | 85 | 53 | 26 | 30 | 51 | 60 |
| Innherred, Norway[26] | 1995 | 94-96 | All stroke | 88 | 13 | 39 | 30 | 65 | — |
| Malmo, Sweden[27] | 1995 | 89-99 | All stroke | 99 | 197 | 21 | 28 | 58 | 62 |
| Southern Italy[28] | 1996 | 1996 | All stroke | 96 | 12 | 42 | 28 | 57 | 58 |
| Adelaide, Australia[29] | 1997 | 95-98 | SAH | 92 | 158 | 44 | 28 | 57 | 57 |
| Hobart, Australia[29] | 1997 | 96-98 | SAH | 100 | 35 | 26 | 28 | 51 | 71 |
| Melbourne, Australia[10, 30] | 1998 | 96-99 | All stroke | 90 | 68 | 41 | 28 | — | 76 |
| China[31] | 1998 | 96-2000 | All stroke | ≥75[e] | 147 | 33 | 28 | 59 | 58 |
| French West Indies[32] | 1998 | 98-99 | All stroke | 93 | 20 | 25 | 30 | 52 | 50 |
| Riga, Latvia[33] | 1998 | 96-2000 | SAH | 81 | 292 | 57[b] | 28 | 52 | 57 |
| Scotland, UK[34] | 1999 | 98-2000 | All stroke | 91 | 23 | 17 | 28 | 63 | — |
| Orebro, Sweden[35] | 1999 | 99-2000 | All stroke | 84 | 11 | 45 | 28 | 66 | 64 |
| Iquique, Chile[37] | 2001 | 2000-02 | All stroke | 94 | 15 | 40 | 30 | 56 | 47 |
| Tartu, Estonia[38] | 2002 | 2001-03 | All stroke | 90 | 18 | 44 | 28 | 61 | 72 |
| Belluno, Italy[39] | 1992 | 92-93 | All stroke | 90 | 12 | 8 | 30 | 54 | 50 |
| Arcadia, Greece[11] | 1994 | 93-95 | All stroke | 82 | 14 | 50 | 28 | 68 | 57 |
| **Studies measuring case-fatality at more than one time point** | | | | | | | | | |
| Perth, Australia[19, 29] | 1989 | 89-90 | All stroke | 86 | 10 | 40 | 28 | — | — |
| | 1997 | 95-98 | All stroke | 89 | 156 | 38 | 28 | — | — |
| | 2000 | 00-01 | All stroke | 91 | 12 | 25 | 28 | — | — |
| Valle D'Aosta, Italy[20] | 1989 | 1989 | All stroke | 98 | 6 | 50 | 30 | — | 50 |
| | 1997 | 1997 | All stroke | 98 | 14 | 21 | 30 | — | 79 |
| Lund-Orup, Sweden[c][21] | 1994 | 93-95 | SAH | 89 | 59 | 32 | 28 | 57 | 56 |
| | 2001 | 01-02 | SAH | 94 | 17 | 18 | 28 | 59 | 65 |
| Northern Sweden[16] | 1987 | 85-89 | SAH | 97 | 297 | 40 | 28 | — | — |
| | 1993 | 90-95[a] | SAH | 97 | 335 | 36 | 28 | — | — |
| | 1998 | 96-2000 | SAH | 97 | 271 | 31 | 28 | — | — |
| Auckland, NZ[13] | 1982 | 81-83 | SAH | 85 | 176 | 53 | 28 | 52 | 64 |
| | 1992 | 91-93 | SAH | 97 | 166 | 46 | 28 | 52 | 65 |
| | 1998 | 97-98 | SAH | 86 | 92 | 35 | 28 | 54 | 58 |
| Oxford (OCSP), UK | 1983 | 82-84, 86 | All stroke | 82 | 26 | 60 | 30 | 59 | 70 |
| Oxford (OXVASC), UK | 2005 | 2002-08 | All stroke | 98 | 38 | 38 | 30 | 60 | 79 |
| South London, UK[36] | 1997 | 95-99 | All stroke | 90 | 86 | 44 | 30 | — | — |
| | 2002 | 2000-04 | All stroke | 90 | 85 | 43 | 30 | — | — |

Abbreviations: CT = computed tomography; PM = postmortem; OCSP = Oxford Community Stroke Project; OXVASC = Oxford Vascular Study; SAH = subarachnoid hemorrhage.

[a] Excludes data for 1992.

[b] Age-adjusted. Crude case-fatality rate not given.

[c] First part of study excluded as CT/PM rate too low.

[d] Earlier study period from 1980–89 not included as there was no systematic review of death certificate data during this period.

[e] Proportion with CT ≥80% for 9/10 cohorts and >75% in remaining cohort.

0.47, 0.23–0.97, $p = 0.04$). Among survivors to reach hospital, the relative risk of death or dependency (mRS $\geq$ 3) at 12 months, adjusted for age and SAH severity, was halved in OXVASC (0.51, 0.29–0.88, $p = 0.01$). The range of modified Rankin scores at 12 months in each study is compared in figure 1.

**Systematic review.** The MEDLINE search produced 16,657 articles, from which we identified 186 potentially relevant studies. Including our population-based study, there were 31 studies that met our inclusion criteria[10-39] (covering 41 study periods from 1980 to 2005). Details of the included studies are shown in table 2.

Weighted linear regression analysis of all studies showed that case-fatality decreased by 0.9% per annum (95% CI 0.3–1.5%, $p = 0.007$). Exclusion of 2 studies[12,14] which did not report 1-month outcomes, and 1 study with an exceptionally low case-fatality rate of

8%,[39] did not substantially alter the results. Eighteen studies including our own[11,13,21-25,27-29,31-33,35,37,38] reported the mean age of cases with SAH and proportion of women. After adjustment for age and sex, there was a similar but nonsignificant reduction in case-fatality of 0.9% per annum (−0.2 to 2.0%, $p = 0.10$).

Seven studies including our own (table 2) measured 1-month case-fatality at more than 1 timepoint, separated by an interval of at least 5 years.[13,14,16,19-21,36] Data from the Perth Community Stroke Study from June 1995 to June 1996[19] were published separately from data from December 1996 to February 1998,[29] but these were combined for this analysis because the periods of study were so close. A pooled analysis of time trends within study populations, based on the difference in 1-month case-fatality taken from the 2 furthest available timepoints (figure 2), demonstrated a 0.9% reduction in case-fatality per annum (0.2–1.6, $p = 0.01$). Unadjusted estimates of case-fatality at different timepoints within each study are plotted alongside the weighted regression line using all data from the 7 population-based studies in figure 3. All studies demonstrated a trend of declining case-fatality rates over time, and the rate of that decline was similar to the results of the weighted linear regression analysis from all studies, with the exception of the Valle d'Aosta study and the Perth Community Stroke Study, which had very small numbers of cases of SAH in at least 2 of the periods examined.[19,20,29]

**DISCUSSION** Although a significant reduction in the incidence of SAH has not occurred in the last 25 years in our Oxfordshire population, mortality has halved largely due to improved survival among hospitalized patients, without a corresponding increase in the proportion of survivors with severe disability. Further evidence of improved outcome after SAH comes from our meta-analysis of time trends in case-fatality within other population-based studies showing a 0.9% absolute annual reduction in crude 30-day case-fatality rates, and a similar sized reduction in case-fatality in a meta-analysis of all population-based studies that measured case-fatality at more than one timepoint within the same population.

Strengths of our population-based study include consistent case-finding methods and diagnostic criteria over time, and detailed data collection during both study periods. Consequently, we could show that the improvement in outcomes is unlikely to be due to better detection of milder cases through improved diagnostic techniques, as the distribution of stroke severity was similar in both study periods. In-

**Figure 2** Absolute % annual rate reduction in case fatality among studies reporting outcomes at 2 different timepoints



% Absolute annual rate reduction
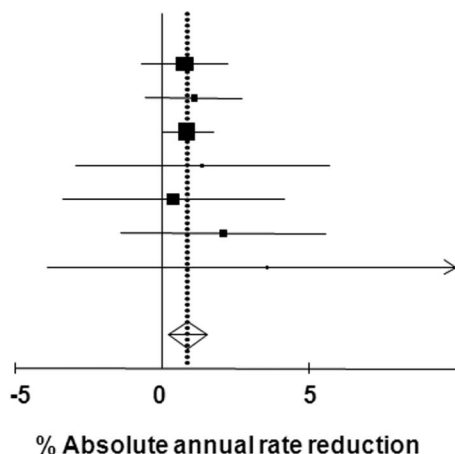
| Study | Deaths / Cases (year) | | Absolute % annual rate reduction | 95% CI |
|---|---|---|---|---|
| | Observation 1 | Observation 2 | | |
| Auckland | 94 / 176 (1982) | 76 / 166 (1992) | 0.8 | -0.7-2.2 |
| Oxford | 16 / 27 (1983) | 14 / 38 (2004) | 1.1 | -0.6-2.7 |
| Sweden | 119 / 297 (1987) | 84 / 271 (1998) | 0.8 | -0.1-1.7 |
| Perth | 4 / 10 (1989) | 3 / 12 (2000) | 1.4 | -3.0-5.7 |
| Sth London | 38 / 86 (1998) | 36 / 85 (2002) | 0.4 | -3.4-4.1 |
| Lund Orup | 19 / 59 (1994) | 3 / 17 (2001) | 2.1 | -1.4-5.5 |
| Valle d'Aosta | 3 / 6 (1989) | 3 / 14 (1997) | 3.6 | -3.9-11.0 |
| TOTAL | | | 0.9 | 0.2-1.6 |

Sig: $p=0.01$
Het: $p=0.99$

**Figure 3**  Crude 1-month case fatality vs year for each population-based study reporting time-trend data plotted alongside the weighted regression line combining all 7 studies
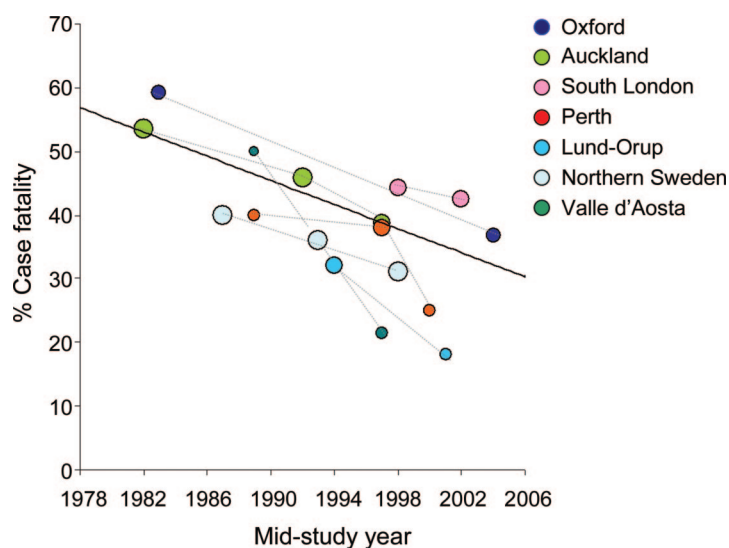
Size of each circle proportional to the log of the number of cases in each study.

stead it is more likely to reflect improvements in the management of SAH. In OXVASC, these include the earlier and more frequent use of neurovascular investigations and interventions compared to OCSP, and greater use of endovascular therapies.

There have been few data published from other population-based studies examining trends in case-fatality following SAH despite ongoing improvements in treatment. Nevertheless, our pooled analysis of these studies demonstrates a significant fall in case-fatality per annum over the last 30 years, consistent with time trends observed in our Oxfordshire population. The majority of data in the pooled analysis come from the Auckland[13,29] and Northern Swedish populations,[16] with the latter study showing a significant decline in case-fatality. Smaller studies including the Perth Community Stroke Study (1989–2001),[19,29] the Lund Registry,[21] and the Valle d'Aosta study (1989–1997)[20] also reported reductions in case-fatality of around 40%, but were not adequately powered to show a significant fall, and the South London Stroke Registry[36] may not have covered a long enough period of time to examine the cumulative effect of changes in SAH management.

Each of the methods we used to study trends in outcome has limitations. Our population-based study is limited by small numbers of fatal cases of SAH in each study period, meaning that chance effects cannot be excluded. There were also few time trend data from other population-based studies. The more indirect method of studying outcomes from separate population-based studies at different time-points included larger numbers of patients from a

wider range of regions, but was subject to bias from differences in case-finding and diagnostic criteria between studies. Nevertheless, the consistency of the results from each of these analyses suggests that the trend for improving outcomes following SAH is real. Furthermore, the 55% reduction in mortality that has occurred in our own population-based study is comparable to the reduction in mortality reported by the Oxford Linkages study in an analysis of routinely collected data within the wider Oxfordshire population over a similar time period.[40]

A further source of bias in analyzing time trends in SAH outcome is the improvement in availability and quality of brain imaging, which has reduced misdiagnosis of SAH and probably improved the detection of milder cases in later study periods. We have tried to minimize this source of bias by limiting our analysis to studies in which the proportion of stroke classified by brain imaging or postmortem was >80% throughout the study. Furthermore, as has been discussed, this bias does not entirely explain the improvements in outcome observed in our own population-based study, as the distributions of stroke severity at presentation in OCSP and OXVASC were similar, and functional outcomes among hospitalized cases still improved after adjustment for differences in age and stroke severity between both study periods. Instead it is more likely that the lower rate of brain imaging/autopsy in OCSP meant that more cases of fatal SAH were missed, resulting in an underestimation of the reduction in case-fatality.

We found no significant decline in the incidence of SAH. If a real decline has occurred, then it has been modest compared to the 40% reduction in stroke incidence overall in OXVASC compared to OCSP.[8] This observation is consistent with the findings of recent systematic reviews of trends in SAH incidence worldwide.[3,4]

Our finding that the SAH outcomes are improving has important implications for clinical practice and research. SAH occurs at a relatively young age compared to other stroke types, and a concern is that if more patients now survive SAH but are severely disabled, the burden of rehabilitation or residential care will increase considerably. However, our results show that there has not been a substantial increase in the proportion of survivors with more severe disability. There will also be consequences for the cost-effectiveness of programs to screen for and treat unruptured aneurysms. The risks and benefits of treating unruptured aneurysms may be closely matched depending on patient age,[9] as well as size and location of the aneurysm, and if outcomes following SAH are improving, then conservative treatment may become more cost-effective for some

patient groups. Finally, our findings have implications for planning randomized trials of treatment in SAH. Given the trend for improving outcomes and greater early survival, greater numbers of patients will need to be recruited to achieve adequate statistical power.

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1. French J, Gronseth G. Lost in a jungle of evidence: we need a compass. Neurology 2008;71:1634–1638.

2. Gronseth G, French J. Practice parameters and technology assessments: what they are, what they are not, and why you should care. Neurology 2008;71:1639–1643.

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