DEATH CERTIFICATE DATA have been used to investigate secular, regional, national, and geographic trends and variations in cerebrovascular disease. Comparison of information collected prospectively, as part of the Framingham Study, offered an unusual opportunity to assess the accuracy of death certification of stroke.

As noted by Acheson, an illness with a short duration from onset to death has a high probability of appearing on the certificate. Although stroke is the third leading cause of death in this country the 30-day fatality rate was only 15% in an earlier study of the Framingham cohort and 30% in the recent National Survey of Stroke. The effect of the time interval from stroke to death on the accuracy of the certificates is therefore of great interest.

Garraway, et al have reported a decline in the yearly incidence and mortality rates of stroke in the population of Rochester, Minnesota. For similar defined populations such as Framingham, Massachusetts and Evans County, Georgia secular trends can be determined that are relatively free from bias due to changes in diagnostic fashion, diagnostic testing and case ascertainment. However, this bias may operate in any study based on death certificate data. To determine the possible effects of diagnostic fashion on death certification of stroke in the Framingham cohort, assessment by calendar year was performed.

Although competent clinicians may differ in classifying a particular case of stroke the poor validity of death certificate studies in reporting the relative frequencies of the types of cerebrovascular disease has been commented on in the past. In particular, several investigations have shown a marked over-reporting of cerebral hemorrhage. We sought to further study disparities between systematic prospective classifications and death certification of stroke type.

Certificates often enter the body of our vital statistics before information from autopsy is made available. Florey, et al noted only a 65% agreement rate in the diagnoses of stroke between autopsies and certificates. The decedents of the Framingham cohort who were subjected to post-mortem examination of the brain were analysed for errors of commission and omission on their certificates.

The methodology of coding certificates has gone through several revisions since the Framingham Study started. Even now determination of the underlying cause of death remains a source for controversy. This investigation was undertaken to assess the accuracy of the basic factors and not to study the judgment of the certifying physician. Therefore an entry of a stroke diagnosis anywhere on the certificate was accepted as encoding the occurrence of stroke.

**Methods**

Since its inception in 1949 the Framingham Study has followed a cohort of 5106 people aged 30 to 62 and free of CHD and CVA at entry with biennial examinations. The method of sampling, composition of the study group, diagnostic criteria and examination procedures have previously been described in detail. Ascertainment of stroke cases seems to have been nearly complete with only approximately 2% of the cohort completely lost to follow-up to the present. Photocopies of the official death certificates of all decedents of the cohort were reviewed.

False negative rates were calculated using decedents with a firm diagnosis of stroke made in accordance to the prospective study criteria as the population at risk.

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Presented at the Second International Joint Conference on Stroke and Cerebral Circulation, February 20, 1982.

Supported in part by Grants numbers NIH IPO INS 16367 and NIH 1R01 NS 17950 01 with Contract number N01 NS 8 2398 (Philip A. Wolf, M.D.), National Institute of Neurological Communicative Disorders and Stroke); Contract numbers NIH N01 HV 92922 and NIH N01 HV 52971 (William B. Kannel, M.D., National Heart, Lung and Blood Institute).

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Received March 17, 1982; revision accepted June 14, 1982.
(denominator). False positive rates were computed using all decedents with a stroke diagnosis on their certificate as the population at risk. False positives did not meet minimal study criteria for the diagnosis of stroke. When stroke was certified as occurring proximate to, or as the cause of death all pertinent records were received and the circumstances surrounding the death discussed with the family, nurses and certifying physician. While some instances of poorly documented stroke may have occurred just prior to death most false positive cases represented either: agonal decreased levels of consciousness occurring in chronically ill persons; and/or sudden unexpected deaths. Episodes of 'sudden death' were not considered as stroke, unless autopsy disclosed an unexpected cerebrovascular lesion. When tabulating false negative rates, entry of 'hemiparesis,' even when its vascular etiology was not noted, was deemed sufficient certification of stroke. Cerebral atherosclerosis was not considered as certifying stroke. All regressions were generated using the method of weighted least squares.

**Results**

Of the 280 stroke victims who died only 167 were recorded (table 1). The overall rate of false negativity was 40%; for men 44% and for women 37%. A stroke diagnosis appeared on 214 death certificates out of 1,980 deaths. Forty-seven of these people were not considered to have had a stroke by our criteria yielding a false positive rate of 22%. There was little difference in the false positive rate between men, 21%; and women, 23%.

There is a strong relationship (P < .001) between time interval from stroke to death and the appearance of stroke on the certificate (fig. 1). If death occurred within one month of the ictus the frequency of false negatives was 7%. But for the 155 people who survived for more than one month 59% did not have stroke certified. Conversely, of the 47 false positives 85% died within one month of the putative stroke.

**Table 1** False Negative Rate and False Positive Rate

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>False negative rate</td>
<td>40%</td>
<td>44%</td>
<td>37%</td>
</tr>
<tr>
<td>False positive rate</td>
<td>22%</td>
<td>21%</td>
<td>23%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>False negative rate</td>
<td>113/280</td>
<td>63/143</td>
<td>50/137</td>
</tr>
<tr>
<td>False positive rate</td>
<td>47/214</td>
<td>21/101</td>
<td>26/113</td>
</tr>
</tbody>
</table>
With increasing age at time of death the false negative rate increased significantly (fig. 2). This may have been due to an increase in individuals with multiple serious, potentially lethal diseases. However, there was no apparent relationship between age and false positive rate (fig. 3).

Over the time span of the study there was a trend toward an increase in the false negative rate and a fall in the false positive rate (fig. 4). Although neither trend reaches significance these results are disconcerting and would tend to produce an apparent fall in the death rates for stroke.

As expected, large disparities in the frequencies of stroke types were noted between prospective study and the certificates (table 2). Whereas 56% of decedents with stroke died subsequent to atherothrombotic brain infarction, only 36% are coded as either infarction or thrombosis. The difference may in part reside in the 26% coded as CVA, stroke, or other non-specific diagnosis. Twenty-seven percent of cases were certified as cerebral hemorrhage. This is a marked discrepancy with the frequency of 6% determined prospectively. Cerebral embolus was grossly under-reported. By prospective analysis 23% of the stroke preceding death were due to embolus. The certificates yielded a relative frequency of only 4%. Subarachnoid hemorrhage with its high case fatality rate fared better. The relative frequency was 7% on the certificates and 12% in the study.

Significantly autopsy with neuropathologic examination of the brain was not performed in the majority of cases. Only 18% (50/280) of the decedents with stroke had brain cutting. Twenty of these individuals with stroke diagnosed by study criteria, and confirmed by neuropathologic study, did not have stroke listed on the death certificate. This 40% (20/50) false negative rate points out that although the occurrence of an autopsy is entered faithfully on certificates the results of the examination are often not incorporated into the certificate entries. There were 34 decedents whose certificates had a stroke entry who also had postmortem pathologic examination of the brain. In four of these (11%), neither autopsy nor clinical data confirmed the stroke diagnosis listed on the death certificate.

**Discussion**

Many authors have requested the validity of death certification of stroke. Possible explanation for these large errors resides in who certifies death and how it is done.

The certifying physician has several obstacles to overcome in certifying the occurrence of stroke. He may know the patient only briefly before death, or in the case of chronic care facilities not at all. He may have treated the patient for a myriad of serious medical illnesses, including a prior stroke, which in his judgment were not related to the patient’s demise. For this reason the stroke diagnosis may be omitted. He may lack sophistication or interest in stroke. He may view certification of death as a laborious chore and enter standard acceptable diagnoses to minimize the expenditure of his time and effort.

The critical weakness in vital statistics concerning stroke lies not in the coding methodology but in the quality of the data itself. Until physicians make use of all the information available to them when certifying death the outlook for improved accuracy remains dim.

**References**

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**TABLE 2** Type of Stroke — Last Event Before Death

<table>
<thead>
<tr>
<th>Type of Stroke</th>
<th>Study criteria</th>
<th>Death certificates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherothrombotic brain infarction</td>
<td>56%</td>
<td>36%</td>
</tr>
<tr>
<td>Cerebral embolus</td>
<td>23%</td>
<td>4%</td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>12%</td>
<td>7%</td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>6%</td>
<td>5%</td>
</tr>
<tr>
<td>Other (vasculitis, migraine, thrombocytopenia, etc.)</td>
<td>3%</td>
<td>26%</td>
</tr>
</tbody>
</table>

**ACCURACY OF DEATH CERTIFICATION OF STROKE THE FRAMINGHAM STUDY: 30 YEARS OF FOLLOW-UP**

**FIGURE 4. Calendar year of death.**
Intracranial Internal Carotid Artery Stenosis: Longterm Prognosis

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JOHN R. LITTLE, M.D.,* MICHAEL T. MODIC, M.D.,† 
AND GEORGE WILLIAMS, PH.D.‡

SUMMARY Sixty-six patients with ≥50% stenosis of an intracranial internal carotid artery (IICA) were followed-up for an average of 3.9 years. Eighteen patients (27.3%) experienced ischemic events; 8 (12.1%) had isolated TIA and 10 (15.2%) a stroke. The observed stroke rate for patients 35 years and older was 13 times the expected infarction rate for a normal population. Patients with tandem extracranial stenosis had a greater risk of stroke than patients with isolated IICA stenosis. Thirty-three patients (50%) died during follow-up and 55% of all deaths were cardiac related. The observed 5 year survival rate was 60% compared to an expected rate of 87%. Patients with IICA stenosis had a higher risk of stroke and death compared to a previously reported referral population with ICA occlusion. IICA stenosis is a marker of extensive cerebrovascular and systemic atherosclerotic disease, especially coronary artery disease.

EXTRACRANIAL TO INTRACRANIAL (EC/IC) ARTERIAL BYPASS surgery is usually done for one of three angio graphic lesions: internal carotid artery (ICA) occlusion, intracranial ICA (IICA) stenosis, or middle cerebral artery (MCA) stenosis/occlusion. While there is some information regarding the long-term risk of stroke in patients with ICA occlusion¹ or MCA stenosis,² the prognosis for atherosclerotic stenosis of the IICA is less clear. Since this information is needed to help evaluate the therapeutic effectiveness of EC/IC arterial bypass surgery, we studied the risk of stroke and death in a population with angiographically proven moderate or severe stenosis of an IICA.

Methods

The cerebral angiographic records of patients seen at the Cleveland Clinic from 1966 through 1977 were reviewed. The angiographic films of all patients with reported stenosis of an IICA were then examined and 69 patients with at least 50% stenosis of an IICA were selected for follow-up. The degree of stenosis was defined as the maximal luminal compromise in any single angiographic plane. Stenoses were localized as follows: intrapetrous (temporal bone to foramen lacerum); intracavernous (foramen lacerum to anterior clinoid); supraclinoid (above anterior clinoid). Comitant stenosis of the extracranial ICA (EICA) and other brain vessels was recorded. Patients with tandem lesions of the EICA were included since isolated IICA stenosis proved too uncommon to permit acquisition of adequate follow-up data.

Follow-up dated from the time of angiography and was accomplished using available Cleveland Clinic records, telephone interviews and/or standardized questionnaires. End points were death, stroke or the performance of EC/IC arterial bypass surgery. Transient ischemic attacks (TIA) or the performance of carotid endarterectomy (CE) were noted but were not sufficient reasons for stopping follow-up. None of the strokes during follow-up were complications of CE. Actuarial methods were used to calculate the net survival during follow-up.³ The Kaplan-Meier product limit method,⁴ in which an estimate is calculated at each unique time of stroke, was used to determine cumulative stroke risk. Original data from a previously reported referral population with ICA occlusion were used for comparing the relative risks of stroke and death for our patients with IICA stenosis.

Results

Follow-up averaged 3.9 years and was achieved in 66 patients (95.7%). The mean age of the 50 males and 16 females at the time of angiography was 61.5 years. The sites of IICA stenosis were: intrapetrous, 14; intracavernous 65; supraclinoid, 6. Nineteen patients

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Acknowledgments: The authors thank D. Caplan, M.D., J. L. Leenders, M.D., R. T. F. O’Brian, M.D., L. F. Rinehart, M.D., H. D. L. Puskas, M.D., J. M. G. Mueller, M.D., C. J. L. H. Ransley, M.D., and C. M. P. K. D. Palma, M.D., for reviewing the manuscript.

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Stroke. 1982;13:818-821
doi: 10.1161/01.STR.13.6.818

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