Decline in Autopsies for Deaths Attributed to Cerebrovascular Disease

Douglas J. Lanska, MD, MS

Background and Purpose: United States national autopsy rates have declined in recent years. In the present study, changes in autopsy rates for deaths due to stroke are examined and compared with changes in autopsy rates for all deaths.

Methods: National Center for Health Statistics data on United States national autopsy rates were examined for the years 1955, 1958, and 1972-1988.

Results: Since at least 1955, nonstroke deaths were more than twice as likely to be autopsied as deaths due to cerebrovascular disease. The annual autopsy frequency for all deaths, for deaths due to stroke, and for deaths due to each stroke subtype declined precipitously after 1972. Since 1982, less than 5 percent of deaths attributed to stroke have been documented by autopsy. Information obtained at autopsy was frequently ignored in the determination of cause of death on the death certificate.

Conclusions: Careful consideration of the value of autopsy for education, research, and quality assurance is urgently needed. Unless the present problems in obtaining, processing, disseminating, and using autopsy data are adequately addressed, the autopsy rate will continue to decline. (Stroke 1993;24:71-75)

KEY WORDS • autopsy • cerebrovascular disorders • magnetic resonance imaging • mortality • tomography, x-ray computed

United States national autopsy rates have declined in recent years1-3 despite pleas, admonitions, and proposals from professional organizations and individual physicians.4-9 In this study, changes in autopsy rates for deaths due to stroke are examined and compared with changes in autopsy rates for all deaths.

Subjects and Methods

National autopsy data were abstracted from volumes of Vital Statistics of the United States for 1955, 1958, and 1972-1988. For these years, autopsy data were available by cause-of-death categories, selected disease rubrics, and gender (1958 and 1979-1988 only). For 1972-1977, the National Center for Health Statistics also tabulated whether the autopsy findings were used to determine the cause of death in selected registration areas; such data are not available for later years because the item "autopsy findings used" was deleted from the US Standard Certificate of Death in 1978. Approximate 95% confidence intervals (CI) for odds ratios (ψ) were calculated by Woolf’s method10; the χ² test for association in a 2×2 table was used as an approximate test of the null hypothesis of no association (H₀: ψ=1).10

Results

The reported autopsy frequency for deaths due to all causes, for deaths due to cerebrovascular disease, and for deaths due to specific stroke subtypes is shown in Table 1 for periods of the last four revisions of the International Classification of Diseases. For each period, nonstroke deaths were more than twice as likely to be autopsied as deaths due to cerebrovascular disease (1955: ψ=2.52; 95% CI, 2.47-2.56; p<<0.0001; 1958: ψ=2.46; 95% CI, 2.42-2.50; p<<0.0001; 1972-1978: ψ=2.82; 95% CI, 2.80-2.84; p<<0.0001; 1979-1988: ψ=3.30; 95% CI, 3.28-3.33; p<<0.0001).

The annual autopsy frequency for all deaths rose slightly from 17.8/100 deaths in 1955 to a peak of 19.1/100 deaths in 1958, then declined to 11.6/100 deaths in 1988 (Figure 1). Similarly, the annual autopsy frequency for deaths attributed to cerebrovascular disease increased slightly from 8.5/100 deaths in 1955 to a peak of 9.4/100 deaths in 1958, then declined to 3.9/100 deaths in 1988 (Figure 1). For all deaths and for deaths due to stroke, the decline in autopsy frequency was precipitous after 1972. Although the trends for the stroke subtypes are more erratic, progressive declines in autopsy frequency have occurred since 1972 for cerebral hemorrhage (from 15.3 to 8.4/100 deaths), cerebral thrombosis (from 6.4 to 4.1/100 deaths), cerebral embolism (from 10.9 to 4.9/100 deaths), and all other cerebrovascular diseases (from 7.9 to 2.9/100 deaths).

Autopsies were performed more frequently in men than women. In the period 1979-1988, autopsies were performed for 1,790,241 (16.5%) of 10,823,947 deaths among men and 893,883 (9.3%) of 9,574,206 deaths among women (ψ=1.92; 95% CI, 1.92-1.93; p<<0.0001). For deaths attributed to cerebrovascular
Table 1. Autopsy Frequencies for Deaths Attributed to All Causes, Cerebrovascular Disease, and Stroke Subtypes for the Years 1955, 1958, 1972–1978, and 1979–1988

<table>
<thead>
<tr>
<th>Years</th>
<th>ICD revision*</th>
<th>Rubric</th>
<th>Cause of death</th>
<th>Total deaths</th>
<th>Autopsies</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>n</td>
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<tr>
<td>1955</td>
<td>6</td>
<td>All</td>
<td>All causes</td>
<td>1,527,691</td>
<td>271,797</td>
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<td></td>
<td>330–334</td>
<td>Vascular lesions affecting CNS</td>
<td>173,541</td>
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<tr>
<td>1958</td>
<td>7</td>
<td>All</td>
<td>Vascular lesions affecting CNS</td>
<td>1,647,886</td>
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<td>Vascular lesions affecting CNS</td>
<td>190,758</td>
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<td></td>
<td>330</td>
<td>Subarachnoid hemorrhage</td>
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<td>Cerebral hemorrhage</td>
<td>113,601</td>
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<td>332</td>
<td>Cerebral embolism and thrombosis</td>
<td>55,548</td>
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<td></td>
<td>333</td>
<td>Spasm of cerebral arteries</td>
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<td>5</td>
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<td></td>
<td>334</td>
<td>Other and ill-defined vascular lesions affecting CNS</td>
<td>17,429</td>
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<td>1,109</td>
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<td>1972–1978</td>
<td>8</td>
<td>All</td>
<td>All causes</td>
<td>13,501,089</td>
<td>2,365,281</td>
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<td>CVD</td>
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<td>1,375,305</td>
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<td>430–438</td>
<td>Intracerebral and other intracranial hemorrhage</td>
<td>199,344</td>
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<td>433</td>
<td>Cerebral hemorrhage</td>
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<td>Cerebral embolism</td>
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<td>434, 435–438</td>
<td>All other CVD</td>
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<td>828,322</td>
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<td>CVD</td>
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<td>Intracerebral and other intracranial hemorrhage</td>
<td>205,331</td>
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<td>435–438</td>
<td>Cerebral embolism</td>
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<td>434.1</td>
<td>Cerebral embolism</td>
<td>7,780</td>
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<td>531</td>
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<td></td>
<td>430, 433, 435–438</td>
<td>All other and late effects of CVD</td>
<td>1,088,272</td>
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<td>39,575</td>
</tr>
</tbody>
</table>

n. Number of autopsies performed. CNS, central nervous system; CVD, cerebrovascular diseases.

*Revision of the International Classification of Diseases in use during the period.

disease in the same period, autopsies were performed for 39,289 (6.2%) of 634,004 deaths among men and 33,915 (3.6%) of 939,893 deaths among women (ψ=1.76; 95% CI, 1.74–1.79; p << 0.0001). Similar gender differences in autopsy frequency were observed for each of the specified stroke subtypes.

Information obtained at autopsy was frequently ignored in the determination of cause of death on the death certificate. In the period 1972–1978, autopsy findings were used in the determination of cause of death in 1,162,631 (72.6%) of the 1,601,324 autopsied deaths of residents of registration areas in which the information was recorded. For deaths attributed to cerebrovascular disease in the same period, autopsy findings were used in 48,437 (67.1%) of the 72,194 autopsied deaths. There was relatively little variation in the use of autopsy findings by stroke subtype: autopsy findings were used in 14,400 (70.6%) of 20,399 autopsied deaths attributed to cerebral hemorrhage, 9,664 (69.5%) of 13,912 autopsied deaths attributed to cerebral thrombosis, 223 (59.0%) of 378 autopsied deaths attributed to cerebral embolism, and 24,150 (64.4%) of 37,505 autopsies attributed to all other and ill-defined cerebrovascular diseases.

Discussion

Autopsies have been used to clarify the causes and pathogenesis of disease, to verify diagnoses, to evaluate and monitor new diagnostic techniques, to help ensure that newer treatments are safe and effective, and to provide educational opportunities for physicians and medical students. Despite the previous successes and despite the potential of autopsy data for research, education, and quality assurance, the autopsy frequency has declined drastically in the United States for all deaths and specifically for deaths attributed to stroke. Support for autopsies has dwindled among hospitals, professional organizations, physicians, and the public. Numerous reasons for this progressive loss of interest in autopsies have been suggested, including perceived

![Figure 1. Graph showing annual autopsy frequencies for deaths from all causes (solid line) and deaths from stroke (broken line) for the years 1955, 1958, and 1972–1988.](image-url)
irrelevance in an era of sophisticated laboratory analytic techniques and advanced radiological diagnostic capabilities, changing responsibilities and priorities among pathologists, shifting of hospital financial support away from nonreimbursed services, and the presence of various apprehensions among clinicians (e.g., litigation) and the public (e.g., disfigurement).1-3,5,6,11 Partly in response to the changing climate for autopsies, in 1970 the Joint Commission on the Accreditation of Hospitals (JCAH) eliminated its recommendation for a 20–25% autopsy rate for hospital deaths; prior to this, the JCAH recommendation was interpreted as a mandate by hospitals and by practicing physicians.1 In addition, autopsies are no longer emphasized in medical education or in the accreditation process for medical schools.

The incorporation of computed tomography (CT) into clinical practice probably contributed significantly to the decline in autopsy rates in the United States.1,12-14 Many physicians considered CT so accurate for clinical purposes that autopsy was superfluous; as expressed by Oldendorf,15 for example, “The CT scan, in essence, permits the clinician to perform a gross brain autopsy at any time during the course of an illness.” Diagnostic evaluations incorporating CT did in fact produce more accurate stroke diagnoses than could be obtained with previously established diagnostic technologies.12,16-25 Moreover, the information provided by CT was available to affect patient management, whereas autopsy could only verify the clinical diagnosis a posteriori.

The temporal pattern of CT diffusion and use is consistent with the contention that adoption of CT technology had a major impact on autopsy rates. The exponential increase in the availability and use of CT26--29 occurred simultaneously with the precipitous decline in autopsy rates in the 1970s. By 1980, approximately 1,500 scanners were in operation in the United States, with scanners in more than 90% of large (over 500 beds) hospitals, in every state, and in the District of Columbia.26,27,29 By this time, probably at least 150,000 patients were being evaluated for cerebrovascular disease annually using the new technology (1,500 scanners×2,200 median number of scans performed per scanner×0.06 to 0.21 scans for cerebrovascular disease per scan performed×0.78 patients per scan performed). Assuming 0.3–0.5 deaths due to stroke per patient with clinical stroke (the approximate ratio of US stroke mortality rates to stroke incidence rates),31 an estimated 45,000–75,000 of the 170,000 people (26–44%) who died in the United States in 1980 of stroke had a CT scan before death. These figures are similar to those found in the Minnesota Heart Survey, in which CT had been obtained in 31% of fatal possible stroke cases in 1980.18 Although more rigorous modeling may alter these figures somewhat, by 1980 certainly many more patients who died of stroke had a CT scan than the 5% who subsequently had an autopsy.18,32 Furthermore, in many of the autopsied stroke cases (at least half of those in the Minnesota Heart Survey)18 CT scans had previously been performed; both autopsy rates and CT use are highest in large hospitals.28,29,32 What incremental information the autopsy added to the diagnosis of stroke in the cases with previous CT scans is not clear, but it was probably not great,28 and it was almost certainly less than that provided in the very small percentage of stroke deaths documented by autopsy with no previous diagnostic imaging.

The introduction of magnetic resonance imaging (MRI) in the 1980s probably had less impact on the autopsy rate for stroke than did the introduction of CT. Diffusion of MRI has been much slower and utilization less than for CT at a comparable point after its introduction.35-39 In addition, MRI has so far had a lesser incremental diagnostic impact than that produced by CT, probably partly because of the greater availability and use of CT and because in many cases both techniques provide similar information. Neither technique is preferred for all of the common clinical situations in which brain imaging is used in the evaluation of cerebrovascular disease. For example, MRI has advantages for imaging acute infarcts, the posterior fossa, and the deep cerebral white matter; CT is better for evaluating acute intracerebral hemorrhage, hemorrhagic infarction, and subarachnoid hemorrhage.40-45 The ultimate role of MRI in the diagnosis and clinical management of stroke patients and any subsequent effect this may have on the autopsy rate remain speculative.

Despite the improved diagnostic capability provided by the introduction of CT and MRI, autopsy studies continue to document frequent diagnostic errors that, had they been known beforehand, would have influenced patient management and possibly improved outcome.13,46 Because of such findings, it remains controversial whether current diagnostic technology is sufficiently accurate and appropriately used to justify current autopsy rates.13,33,47 Also, for research purposes, no present form of cerebral or arterial imaging can elucidate the pathology or pathogenesis of stroke as completely as autopsy.

At the present low autopsy rates, it is difficult to generalize the information obtained because autopsied deaths are not necessarily representative of all deaths or even of hospital deaths.48-50 Indeed, the small percentage of autopsied deaths are selected preferentially from among minorities, men, younger individuals, traumatic deaths, and deaths following complicated or puzzling illnesses.11,22,49-55 In studies using autopsy data, bias introduced by differential selection of patients for autopsy may, for example, mask a real relation, produce a spurious association, or distort rates; only a small part of the bias can be measured or avoided.48

Although many proponents of the autopsy have eloquently championed increasing the frequency of autopsies, this goal is insufficient and somewhat misdirected. As noted by King,56 “It is a pernicious misconception that the mere performance of postmortem dissection leads to progress in medical science. . . . Those who believe that the more autopsies we perform, the more medical science will advance, are actually pleading not for more autopsies but for more persons who can profitably utilize the data of autopsies. . . .” Unfortunately, the information collected at autopsy is often improperly or insufficiently used,5 limiting any potential value of the exercise for research.57,58 Education,59 or quality assurance.7,60,61 Vast quantities of autopsy data have been routinely collected but remain largely unused because the information is not aggregated as a usable data source and because much of this “routine” data is of little clinical relevance or research value.57-58 In the present study and others,58 information obtained at
autopsy was frequently ignored in the determination of the cause of death listed on the death certificate; moreover, when autopsy information was used, it did not necessarily result in an etiologically precise death certificate diagnosis.

In the clinical arena, autopsy results are often not communicated well, and what is communicated apparently has had little impact. In many hospitals, autopsy results are not made available to clinicians or families promptly, understandably, or diplomatically. On the other hand, clinicians frequently show little interest in autopsy results and seldom attend the autopsies performed. Furthermore, despite a wealth of opinion, there is presently no objective evidence that the identification of errors or discrepancies by autopsy has altered subsequent clinical management of similar cases.

Careful consideration of the value of autopsy for education, research, and quality assurance is urgently needed. Unless the present problems in obtaining, processing, disseminating, and using autopsy data are adequately addressed, the autopsy rate will continue to decline. This trend might potentially be reversed if one could demonstrate that autopsies disclose new, useful information on cerebrovascular disorders; that opportunity may cease to exist, however, if autopsies are no longer performed or if autopsies are performed in a routine fashion by personnel who are either inadequately trained or insufficiently prepared to answer the pertinent questions.

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References

47. Burrows S: The postmortem examination: Scientific necessity or folly? JAMA 1975;233:441–443
48. Mainland D: The risk of fallacious conclusions from autopsy data on the incidence of diseases with applications to heart disease. Am Heart J 1953;54:646–654
53. McManus CA: Age-sex distributions of selected groups of human autopsied cases. Arch Pathol 1962;73:40–47
54. Guptill CS: How autopsy data are biased by age factors. Geriatrics 1976;31:78–82
57. Starr I: Potential values of the autopsy today. JAMA 1956;160:1144–1145
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