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Stroke 1987;18;823-829
Stroke is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0039-2499. Online ISSN: 1524-4628

Declining Morbidity and Mortality of Carotid Endarterectomy

The Wake Forest University Medical Center Experience

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The 30-day mortality as well as morbidity for stroke and myocardial infarction were determined by review of the charts for every carotid endarterectomy (N = 389 operations on 356 patients) performed at Wake Forest University Medical Center from 1979 through 1983 to ascertain whether the 16% morbidity and 6% mortality documented in our previous report of 1978 had changed over time. For endarterectomies performed on asymptomatic patients (n = 155), major morbidity included 2 myocardial infarctions and 1 stroke (1.9%). There were 3 fatalities — 2 myocardial infarctions and 1 stroke (1.9%). For the symptomatic group (n = 234), major morbidity was 2.1%, mortality 2.6%. The combined morbidity for asymptomatic and symptomatic carotid stenosis was 2%, mortality 2.3%. Perioperative stroke rate (morbidity plus mortality) was 2.6%, 9 ipsilateral to the carotid endarterectomy, suggesting distal embolism as its probable cause. We contend that quality control measures implemented to correct the unacceptable rates reported in 1978 have contributed to dramatic and sustained reductions in complication rates. (*Stroke* 1987;18:823–829)

In 1978, we reported a series of 124 patients with transient ischemic attacks (TIAs) who had undergone carotid endarterectomy at our institution.[1] Mortality was 6%, and the stroke morbidity was 16%. This was an unacceptable complication rate, and measures were implemented to reduce it. The measures included a TIA and stroke registry with continuing audit of patient management and outcome, more rigorous criteria for selecting candidates for endarterectomy, change from general anesthesia to superficial cervical plexus block so that the alert patient is used as his own monitor of brain function, the use of a shunt only for patients found not to tolerate carotid clamping, and ambulation of the patient within 12 hours after surgery. Here we report results from 1979–1983 to determine whether these changes have contributed to a decrease in morbidity and mortality. Furthermore, because of poor results recently reported, we were particularly desirous of comparing our results with those of others.[2,3]

Subjects and Methods

The hospital records of every patient who underwent carotid endarterectomy at our tertiary care hospital from January 1979 through December 1983 were reviewed by one of three neurologists who reviewed nurses' and physicians' notes, laboratory findings, carotid ultrasounds, angiograms, anesthesia and operative records, and 30-day follow-up data. The vast majority of the patients subjected to endarterectomy had been evaluated by neurologists who also participated in the postoperative care. Asymptomatic patients were those who responded negatively to detailed questioning by neurologists or neurosurgeons. All patients had been classified as having had a stroke if neurologic impairment persisted for > 24 hours and as having had a TIA if for less. Patients were classified as hypertensive if they received antihypertensive medication or if their systolic blood pressure exceeded 140 mm Hg or their diastolic exceeded 90 mm Hg. Diabetics were those on hypoglycemic agent, diabetic diet, or who had elevated fasting blood sugar or an abnormal glucose tolerance test. Preoperative and postoperative medications such as aspirin, ibuprofen, warfarin, heparin, indomethacin, or dipyridamole, which might affect outcome, were noted, as were complete blood and platelet count, prothrombin and partial prothrombin times, automated blood panel analysis, and urinalysis. The surgeon, type of anesthesia, use of a shunt, and special monitoring were noted. All complications occurring within 30 days of surgery were tabulated with special note of TIA, reversible ischemic neurologic deficit (RIND), strokes, wound infection, hypertension, hypotension, myocardial infarction, or death. All data were entered into a VAX 730 computer.

Results

Three hundred eighty-nine endarterectomies were performed on 356 patients, 323 unilateral and 33 on the other carotid artery within 90 days of the first. If the time between surgeries exceeded 3 months, they were considered 2 separate operations. Table 1 displays the

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Supported in part by the Colonel C.C. Smith Stroke Research Fund and United States Public Health Service NIH Grant NS06655.

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Received August 25, 1986; accepted April 20, 1987.

Table 1. Characteristics of 356 Patients

	Number	Percent
Demographics		
Caucasian	349	98
Male	229	64
Age >65 years	151	42
Risk factors		
Hypertension	218	61
Cardiac decompensation	21	6
Myocardial infarction	78	22
Angina pectoris	95	27
Cardiac dysrhythmia	42	12
Valvular disease	24	7
Coronary bypass	27	8
Diabetes mellitus	42	12
Tobacco use	238	67
Hypertension and/or tobacco	315	88
Any of the above	335	94

demographics and some of the preoperative medical conditions that might have indicated higher operative risk. The preoperative blood studies revealed hemoglobin, prothrombin time, partial prothrombin time, platelet count, and cholesterol levels to be within normal limits in the majority of the patients.

Operative indications ipsilateral to the side of surgery were TIA, RIND, or amaurosis fugax in 182 endarterectomies (47%), and minor deficit from infarction in 52 (13%); 155 (40%) were asymptomatic. Tabulated by patient rather than by procedure, 204 patients (57%) had TIA, 60 (17%) minor infarction, and 92 (26%) no symptoms.

Arteriography in the common or internal carotid arteries showed that 70% of the arteries had 76–99% atherostenosis on the side chosen for endarterectomy (Table 2). Twelve endarterectomies were performed on patients with ulcerated lesions but < 25% stenosis. Surgery on 15 patients with occlusion reestablished flow in 7. Three patients with stroke-in-evolution and occlusions had thrombectomy and endarterectomy with no worsening of their neurologic status. In no instance was there a new neurologic deficit postoperatively among the patients with occlusion. One asymptomatic patient had a subclavian–carotid bifurcation bypass graft for an occluded common carotid artery. Eight symptomatic patients had an external carotid endarterectomy with ligation of the internal carotid stump to prevent emboli.

Seventy-six percent of the operations were performed under local anesthesia, 5% with a shunt — usually a Javid indwelling catheter. Only 24% of the procedures were performed under general anesthesia, but 67% of the complications occurred in this group. A trend toward lower blood pressure (< 170 mm Hg systolic) during the clamp period was noted in the patients having neurologic deficits immediately postoperatively.

Table 2. Distribution of Preoperative Common or Internal Carotid Artery Stenosis

Side of operation	Arteriographic stenosis				
	0–25%	26–50%	50–75%	76–99%	100%
Ipsilateral	12 (3)	27 (7)	64 (16)	269 (70)	15 (4)
Contralateral	132 (34)	84 (22)	47 (12)	93 (24)	31 (8)

387 arteriograms. Percent follows in parentheses.

Minor operative complications included 14 patients with transient hypotension, 4 with transient hypertension, and 14 with wound-related complications including small hematoma or some dehiscence that resolved uneventfully. Four patients had transient ipsilateral facial weakness with complete resolution; 1 patient had transient bilateral hypoglossal nerve dysfunction after bilateral procedures.

Table 3 summarizes the surgical mortality and morbidity, by procedure rather than by patient, for stroke and myocardial infarction by time, which may give clues about their cause. For example, those complications occurring within 1 day might be related to case selection or surgical technique, whereas those complications occurring later could be the result of postoperative management.

The 1-day mortality was 2 of 389 (0.5%), and the stroke morbidity was 5 of 389 (1.3%). One asymptomatic patient died undergoing a combined endarterectomy and coronary artery bypass. One symptomatic patient had a mild stroke ipsilateral to the endarterectomy and died of a hemorrhagic infarct immediately after surgery. Of the 5 patients (4 symptomatic) who suffered strokes, 1 asymptomatic patient had a subclavian–left carotid graft with endarterectomy, and 1

Table 3. Perioperative All-Cause Mortality, Myocardial Infarctions, and Stroke Morbidity

	Myocardial infarction		Stroke		
Day	Non-fatal	Fatal	Non-fatal	Fatal	Other fatality
Asymptomatic stenosis (n = 155)					
0–1	0	1	1	0	0
2–7	0	0	0	1	0
8–14	0	0	0	0	0
15–30	2	1	0	0	0
Symptomatic patients					
TIA (n = 182)					
0–1	0	0	4	0	0
2–7	0	1	0	0	0
8–14	0	0	0	0	1
15–30	0	0	0	1	1
Infarction (n = 52)					
0–1	0	0	0	1	0
2–7	0	0	0	0	0
8–14	0	0	1	1	0
15–30	0	0	0	0	0

n, number of endarterectomies.

symptomatic patient underwent revision of an endarterectomy with a patch graft. Neurologic deficits in this group were mild — no discharge was delayed because of neurologic impairment, and all patients were ambulatory.

Within the first week, an asymptomatic patient died of cerebral infarction after discharge, and there was 1 cardiac death in a symptomatic patient.

In the second week, there were 2 strokes in the symptomatic group; 1 was fatal. The first patient developed fever, was found to have a hepatic abscess, and suffered a stroke intraoperatively during the laparotomy. The second patient returned to the hospital in *status epilepticus* and was found to have a holohemispheric infarct from which he died. A third symptomatic patient with chronic respiratory disease died of nosocomial pneumonia and sepsis.

In the second 2 weeks, there were 3 myocardial infarctions in the asymptomatic group — 1 was fatal. One patient had a nonfatal infarct after discharge. A second patient underwent ileal loop revision 1 week after the endarterectomy; he subsequently developed urosepsis and hypotension and suffered a nonfatal anterior wall myocardial infarction. The third patient died suddenly. In the symptomatic group, there were 2 additional deaths, 1 fatal cerebral infarction contralateral to the operation, and 1 death from multiple pulmonary emboli.

In the asymptomatic group, there was 1 myocardial infarction on the day of surgery and 3 others that occurred 15–30 days thereafter, suggesting that they were temporally related to but not caused by surgery. Three patients were felt to have had medically stable coronary artery disease while 1 patient had no history or evidence of heart disease. Stroke morbidity was 1 of 155 (0.65%), and perioperative mortality was 3 of 155 (1.9%).

In the symptomatic group, by 30 days there were 6 deaths, 3 from cerebral infarction. Five additional nonfatal strokes occurred for a total of 8, 7 ipsilateral to the endarterectomy. The stroke morbidity was 5 of 234 (2.1%), and the operative mortality was 6 of 234 (2.6%).

Stroke morbidity for the asymptomatic and symptomatic groups combined was 6 of 389 (1.5%), and operative mortality was 9 of 389 (2.3%).

Table 4. Stroke Morbidity and Mortality of Endarterectomy in Patients With Asymptomatic Carotid Stenosis in U.S. Hospitals

Author	Inclusive years	Publication date	Number	Postoperative			
				Stroke		Death	
Medical centers							
Gaal and Wong[4]	57–63	1964	5	0	0%	0	0%
DeBakey et al[5]	53–63	1965	48	1	2%	2	4%
Heyman et al[6]	58–64	1967	4	0	0%	0	0%
Young et al[7]	64–68	1969	33	0	0%	1	3%
DeWeese et al[8]	61–68	1971	50	3	6%	0	0%
Javid et al[9]	65–70	1971	56	2	4%	1	2%
Ojemann et al[10]	?	1975	19	0	0%	0	0%
Kanaly et al[11]	70–76	1977	14	0	0%	0	0%
Hertzer et al[12]	74–76	1978	94	0	0%	2	2%
Duke et al[13]	67–77	1979	17	0	0%	0	0%
Moore et al[14]	61–77	1979	72	0	0%	0	0%
Thompson and Talkington[15]	58–78	1979	132	2	1.5%	0	0%
White et al[16]	74–79	1981	32	0	0%	0	0%
Lees and Hertzer[17]	69–73	1981	83	1	1%	3	4%
Burke et al[18]	65–79	1982	57	1	2%	0	0%
Bunt and Haynes[19]	80–84	1985	45	0	0%	1	2%
Fode et al[20]	81	1986	572	20	3.5%	15	3%
This study	79–83	1987	92	1	1%	2	2%
Community hospitals							
Nunn[3]	63–73	1975	28	0	0%	0	0%
Easton and Sherman[21]	70–76	1977	11	2	18%	0	0%
Cornell[22]	66–76	1978	4	0	0%	0	0%
Kremer and Ahlquist[23]	72–78	1979	42	0	0%	0	0%
Carmichael[24]	67–78	1980	27	0	0%	0	0%
Modi et al[25]	76–82	1983	74	2	3%	2	3%
Brott and Thalinger[26]	80	1984	130	10	8%	4	3%

Analysis of the results by patients rather than by procedure increases the stroke morbidity to 6 of 356 (1.7%) and the total mortality to 9 of 356 (2.5%).

Eighty-five percent of the patients received at least one postoperative medication: 141 received aspirin, 134 Ascriptin, 276 dipyridamole, 8 warfarin, and 8 other nonsteroidal anti-inflammatory medications.

Discussion

Numerous authors have reported the endarterectomy complication rates for various categories of cerebrovascular ischemia.[46,47] Tables 4–6 list those reports from which data on complications could be extracted by subgroups of asymptomatic stenosis, TIA, or estab-

Table 5. Stroke Morbidity and Mortality of Endarterectomy in Patients With Carotid Transient Ischemic Attacks in U.S. Hospitals

Author	Inclusive years	Publication date	Number	Postoperative Stroke		Postoperative Death	
Medical centers							
Siekert et al[27]	54–58	1963	32	5	16%	3	9%
DeBakey et al[5]	53–63	1965	324	15	5%	16	5%
Yashon et al[28]	?	1966	121	Unknown		5	4%
Heyman et al[6]	58–64	1967	49	4	8%	4	8%
Bloodwell et al[29]	56–66	1968	191	5	3%	8	4%
Young et al[7]	64–68	1969	104	6	6%	5	5%
Erikson et al[30]	?	1970	29	0	0%	0	0%
Fields et al[31]	62–68	1970	169	13	8%	6	4%
DeWeese et al[8]	61–68	1971	187	18	10%	4	2%
Smith et al[32]	58–69	1971	37	Unknown		1	3%
DeWeese et al[33]	61–66	1973	103	6	6%	1	1%
Hooshmand et al[34]	60–70's	1974	17	0	0%	0	0%
Ford et al[35]	71–74	1975	46	0	0%	0	0%
Ojemann et al[10]	?	1975	104	3	3%	1	1%
Kanaly et al[11]	70–76	1977	69	3	4%	2	3%
Mungas and Baker[36]	71–75	1977	80	1	1%	0	0%
Stanford et al[37]	69–76	1978	154	4	3%	0	0%
Hertzer et al[12]	74–76	1978	143	3	2%	1	1%
Toole et al[1]	62–73	1978	124	20	16%	7	6%
Duke et al[13]	67–77	1979	65	2	3%	1	2%
Thompson and Talkington[15]	58–78	1979	575	Unknown		7	1%
Riles et al[38]	62–76	1980	28	1	4%	1	4%
Owens et al[39]	74–79	1980	109	2	2%	0	0%
White et al[16]	74–79	1981	104	2	2%	1	1%
Carson et al[40]	77–79	1981	24	0	0%	0	0%
Lees and Hertzer[17]	69–73	1981	117	11	9%	Unknown	
Whisnant et al[41]	70–74	1983	151	5	3%	1	1%
Bunt and Haynes[19]	80–84	1985	127	2	2%	0	0%
Fode et al[20]	81	1986	1619	57	3.5%	18	1.1%
This study	79–83	1987	204	4	2%	2	1%
Community hospitals							
Nunn[3]	63–73	1975	160	4	2.5%	2	1%
Easton and Sherman[21]	70–76	1977	73	13	18%	4	5.5%
Cornell[22]	66–76	1978	61	0	0%	0	0%
Park[42]	72–77	1979	65	3	5%	3	5%
Kremer and Ahlquist[23]	72–78	1979	40	1	2.5%	0	0%
Carmichael[24]	67–78	1980	294	7	2%	1	0.3%
Modi et al[25]	76–82	1983	249	4	2%	1	0.4%
Brott and Thalinger[26]	80	1984	141	17	12%	6	4%

Table 6. Stroke Morbidity and Mortality of Endarterectomy in Patients With Established Cerebral Infarction in U.S. Hospitals

Author	Inclusive years	Publication date	Number	Postoperative Stroke		Postoperative Death	
Medical centers							
DeBakey et al[5]	53–63	1965	406	25	6%	29	7%
Yashon et al[28]	?	1966	50	Unknown		7	14%
Heyman et al[6]	58–64	1967	42	2	5%	6	14%
Erikson et al[30]	?	1970	71	9	13%	12	17%
Thompson et al[43]	57–70	1970	217	11	5%	16	7%
DeWeese et al[8]	61–68	1971	35	Unknown		2	6%
Smith et al[32]	58–69	1971	56	Unknown		6	11%
Ojemann et al[10]	?	1975	50	5	10%	2	4%
Kanaly et al[11]	70–76	1977	20	3	15%	4	20%
Hertzer et al[12]	74–76	1978	23	2	9%	0	0%
Duke et al[13]	67–77	1979	51	2	4%	1	2%
Riles et al[38]	62–76	1980	24	1	4%	0	0%
Lees and Hertzer[17]	69–73	1981	50	4	8%	Unknown	
White et al[16]	74–79	1981	25	2	8%	3	12%
Bardin et al[44]	70–79	1982	127	5	4%	4	3%
McCullough et al[45]	76–83	1985	59	2	3%	1	2%
Bunt and Haynes[19]	80–84	1985	28	3	11%	2	7%
Fode et al[20]	81	1986	477	26	5%	3	0.6%
This study	79–83	1987	60	1	1.7%	2	3.4%
Community hospitals							
Nunn[3]	63–73	1975	6	Unknown		1	17%
Easton and Sherman[21]	70–76	1977	99	15	15%	9	9%
Cornell[22]	66–76	1978	35	2	6%	4	11%
Kremer and Ahlquist[23]	72–78	1979	10	2	20%	0	0%
Brott and Thalinger[26]	80	1984	74	8	11%	1	1%

lished cerebral infarction, respectively. Our effort to summarize the results from various centers appears to have been a relatively simple task until one realizes the many ways in which patients and outcomes are categorized in these reports; therefore, caution must be taken in interpreting these tables.

Reports also differ in many ways that may be critical to the outcome measure, i.e., postoperative stroke or death from surgery. Patient characteristics such as prognostic severity, degree of stenosis, presence of cardiac risk factors, and concomitant anticoagulant treatment, etc., have not been identified that allow delineation of subgroups of patients sufficiently similar for comparisons. Homogeneity in these tables should therefore not be assumed. For example, the category of ischemic cerebrovascular disease is not always consistent across studies. Reports concerning results of management of asymptomatic carotid disease may refer to carotid bruits rather than to hemodynamically significant stenosis. Symptoms may refer to the patient's overall cerebrovascular disease and not necessarily to only those symptoms ipsilateral to the side of surgery. Furthermore, TIA was defined as 1 hour in the study by Siekert et al in 1963,[27] whereas DeBakey et al[5] used a few days.

Another problem is the diverse nature of the studies in terms of design and methods. Across medical centers and community hospitals, there are differences in surgical and anesthetic techniques. Some reports of operative complications are based on the results per procedure, others on results per patient. In our survey of the literature, we tabulated patients rather than procedures when at all possible; natural history and therapy are always based on the number of patients. The classification of the "perioperative period" is the first few postoperative days for some but several months for others. In this report, 30 days was used for data analysis in Table 3, but in Tables 4–6, 2 weeks was used to allow comparisons with other studies.

Though no statistical conclusions can be drawn, several general observations can be made. The reductions in major morbidity and mortality following carotid endarterectomy from the 1960s until 1985 are undoubtedly multifactorial. A priori, one assumes that poor results are underreported. Whether this reticence has increased cannot be assessed, but, if so, it is a

hidden contributor to an apparent reduction in complication rates. Furthermore, with time the population has become more aware of TIA and risk factors and physicians more aware of carotid bruits, so intervention may take place earlier in the course of the disease, when the patient may be a better risk. Beginning in the mid 1970s, many patients received antiplatelet medication, which may have reduced the tendency for postoperative thromboembolism. Other considerations are improved surgical technique, use of more skilled surgeons, more effective medical therapeutics, and better patient selection. We attribute the dramatic improvement in operative mortality and morbidity in our institution between 1977 to 1985 to institutional safeguards such as 1) a stroke research center that highlights cerebrovascular disorders for the institution and makes all physicians and other health care providers aware of the disorders' catastrophic effects; 2) use of a stroke registry into which all patients are accessed so that an ongoing audit of management results can be carried out by neurologists and surgeons; 3) improved case selection, influenced to a large degree by close interaction among neurologists, cardiologists, and surgeons; 4) a thorough evaluation of all patients for coronary artery disease before endarterectomy, and if advanced disease is found, treatment of the coronary artery disease and medical management of the cerebrovascular disorder; 5) use of our most skilled surgeons, not house officers, for performing this seemingly simple but very dangerous operation [2 neurosurgeons (59%), 4 vascular surgeons (27%), 6 general surgeons (9%), and house officers (5%) performed the surgery at our institution]; 6) use of regional anesthesia so the patient is awake and communicating during the operation, acting as his own monitor of brain function, and so the anesthesiologist can ascertain instantly if cross-clamping of the artery has resulted in a neurologic deficit or if neurologic deficit from any cause is developing; 7) selective use of shunts because they add time to the procedure, but more importantly because they can cause trauma to the artery during their placement and serve as cul de sacs for debris that may embolize; and 8) meticulous removal of plaque and all detritus, with prolonged retrograde flushing of blood from the internal to the external carotid artery prior to arteriotomy closure.

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KEY WORDS • carotid endarterectomy • morbidity • mortality • transient ischemic attacks • stroke • asymptomatic stenosis