01 Jan 1985

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Recommended Citation

P. B. Gorelick et al., "Racial Differences in the Distribution of Posterior Circulation Occlusive Disease," *Stroke*, vol. 16, no. 5, pp. 785 - 790, Lippincott, Williams & Wilkins; American Heart Association, Jan 1985.
The definitive version is available at https://doi.org/10.1161/01.STR.16.5.785

Racial Differences in the Distribution of Posterior Circulation Occlusive Disease

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SUMMARY **We compared clinical and arteriographic features in 27 white and 24 black patients with symptomatic posterior circulation occlusive disease. The degree of arterial stenosis was measured independently by two examiners at 12 sites within the vertebrobasilar territory. Racial comparisons were made based upon the distribution of extra- and intracranial occlusive lesions and symptomatic sites of the lesions.**

White patients had significantly more angina pectoris, more lesions of the origin of the left vertebral artery and more high grade lesions of the extracranial vertebral arteries. Black patients had significantly higher mean diastolic blood pressure, more diabetes mellitus, more lesions of the distal basilar artery, more high grade lesions of intracranial branch vessels and more symptomatic intracranial branch disease. Race was found to be the only factor increasing the risk of intracranial posterior circulation occlusive disease.

Knowledge of the contribution of race to the distribution of posterior circulation lesions will help guide evaluation and treatment strategies for patients with vertebrobasilar occlusive disease.

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STUDY OF THE VERTEBROBASILAR VESSELS at necropsy by Hutchinson and Yates suggested that occlusive cerebrovascular disease was common in the cervical portions of the vertebral artery but was rare intracranially.[1] Later, arteriographic studies by Meyer et al[2] and Bauer et al[3] corroborated the earlier pathologic findings and also noted a high prevalence of intracranial occlusive disease. In post-mortem examinations, Castaigne et al[4] and others[5-8] have observed the frequent occurrence of occlusion or stenosis of the intracranial vertebral artery, and the basilar artery.[9, 10]

We have recently described a racially mixed group of patients with anterior circulation ischemic stroke who had cerebral angiography.[11] We found that black patients had more severe occlusive disease of the middle cerebral artery whereas white patients had more severe disease of the internal carotid artery origin. Differences were not explained by racial differences in the prevalence of hypertension, diabetes, hypercholesterolemia or ischemic heart disease. Since parallel racial differences might exist in the distribution of occlusive posterior circulation disease, we studied a racially mixed group of patients with angiographically documented ischemic vertebrobasilar disease.

Methods

Study Population

Four medical centers pooled their resources to determine racial differences in the distribution of vertebrobasilar territory occlusive disease. Stroke records of all patients with a discharge diagnosis of acute, symptomatic posterior circulation occlusive cerebrovascular disease (stroke or transient cerebral ischemia) who had cerebral angiography were reviewed. Patients with the following discharge diagnoses were then eliminated: emboli of cardiac origin, artery-to-artery emboli causing distal vessel occlusion, cerebral aneurysms, arteriovenous malformations, fibromuscular dysplasia, arterial dissection, vasculitis, hematologic disease, and subdural and epidural hematomas. Discharge diagnoses were based upon the clinical and laboratory criteria set forth in the Harvard Cooperative Stroke Registry.[12] Only patients with angiographic lesions in the vertebrobasilar system were included. Angiographic studies were available in 51 patients including 27 whites (19 men and 8 women) and 24 blacks (17 men and 7 women).

Study Design

Rapid serial cerebral angiography was performed by conventional (nonmagnification) biplane technique in 44 cases. Routine anteroposterior and lateral views were supplemented by oblique projections in special instances. Digital subtraction arteriography by direct arterial injection was performed in an additional 7 cases. Routine examinations included anteroposterior and lateral projections.

All conventional and digital subtraction arteriographic studies were performed by either a transfemoral or brachial route. The pertinent angiographic films were selected, and patient identification markings were masked. All films were reviewed independently by two graders. Each examiner measured the degree of stenosis at the following sites: vertebral artery origin (1st portion); vertebral artery as it traverses through the foramina transversaria (2nd portion); vertebral artery as it curves around the atlanto-axial junction (3rd portion); intracranial vertebral artery (4th portion); proximal, middle and distal basilar artery segments; origins of the posterior inferior cerebellar artery (PICA), anterior inferior cerebellar artery

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This work was supported in part by a grant from the AMOCO Foundation and NIH Contract #N01-NS-2-2399.

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Received September 10, 1984; revision #1 accepted January 23, 1985.

(AICA), superior cerebellar artery (SCA) and posterior cerebral artery (PCA) (P_1 segment or peduncular portion); and the second segment of the posterior cerebral artery (P_2 or ambient segment). The first three portions of the vertebral artery were designated as *extracranial*. The remaining vessel sites were considered *intcracranial*. The PICA, AICA, SCA, P_1 and P_2 portions of the PCA, basilar branches and thalamoperforants were designated as *branch* arteries. The remaining vertebral and basilar vessel sites were considered *trunk* arteries.

Perforating arteries, poorly visualized vessels distal to high-grade stenosis, and the distal PICA, AICA, SCA, and PCA branches were not studied. Luminal encroachment was the only factor analyzed. Kinks, tortuosity, dilatation and extrinsic compression by osteophytes or other structures were ignored.

Each examiner's measurements were tallied separately. The degree of stenosis (expressed as a percentage) was calculated by the method of Alter et al[13] After the initial rating, the values of the two observers were compared. A significant discrepancy was defined as disagreement between a rating of total occlusion or stenosis (less than 50% residual lumen diameter); stenosis or plaque (between 50 and 99% residual lumen diameter); or plaque versus normal. Each discrepancy was reviewed and a consensus rating was reached. Only 10% of all lesion sites required arbitration.

Patient Characteristics

The following data were collected on each patient: (1) age; (2) sex; (3) race; (4) history of diabetes (defined as dependence on insulin or oral hypoglycemic agents, diet-controlled with a fasting blood glucose greater than 130 mg%, or a persistent fasting blood glucose greater than 130 mg% during acute hospital admission); (5) history of transient ischemic attack (occurring within one year of admission); (6) history of hypertension (defined as past use of antihypertensive medication for blood pressure elevation or past blood pressure recordings with systolic > 140 mm Hg and diastolic > 90 mm Hg); (7) systolic and diastolic blood pressure recordings (taken during the first 24 months of hospitalization); (8) history of myocardial infarction; (9) serum cholesterol level; (10) atrial fibrillation (diagnosed prior to or during acute hospitalization); and (11) past history of angina pectoris or limb claudication.

Statistical Methods

Group means were compared by the two-tailed t-test (approximate normality established) for continuous variables. Proportions were compared by the chi-square test for 2 × 2 contingency tables. Logistic regressions were utilized to investigate the influence of covariates.[14]

Results

Patient Characteristics

Black patients had a higher mean diastolic blood pressure ($p = 0.04$). Racial differences in age, mean systolic blood pressure and mean cholesterol level were not significant (table 1). Diabetes was more common in blacks ($p = 0.007$) and angina pectoris was more common in whites ($p = 0.05$). Racial differences in the incidence of transient ischemic attacks, hypertension, myocardial infarction and claudication were not statistically significant. The sex ratios in the two racial groups did not differ significantly (table 1).

Distribution of the Stenotic Lesions

For each vessel site in the vertebrobasilar territory, racial differences in the number of vascular lesions of any type (plaques, stenoses or occlusions) and in the number of tight lesions (>49% stenosis) were analyzed by two-by-two contingency tables (table 2).

Extracranial sites. In the right vertebral artery whites had more tight lesions of the 1st portion ($p = 0.02$). In the left vertebral artery whites had more lesions ($p = 0.02$) and more tight lesions ($p = 0.05$) of the first portion (table 2).

Intracranial sites. In the basilar artery blacks had more lesions of the distal segment ($p = 0.05$). There were no other differences in the distribution of stenotic lesions at individual sites (table 2).

Severity of the Stenotic Lesions

The severity of stenotic lesions was compared by counting the number of occlusive lesions that were greater than 0% stenosis and greater than 49% stenosis (table 3). Black patients were more likely to harbor one or more intracranial branch lesions of greater than 0%

TABLE 1 *Patient Characteristics*

	Mean ± SD (range)	
	Whites N = 27	Blacks N = 24
Age (years)	55.6 ± 12.6 (30–81)	58.7 ± 11.2 (35–76)
Systolic blood pressure (mm Hg)	145.3 ± 30.5 (100–230)	149.7 ± 22.4 (120–210)
Diastolic blood pressure (mm Hg)	84.1 ± 12.7 (50–100)	91.0 ± 10.0* (70–112)
Cholesterol (mg %)	231.3 ± 58.3 (139–317)	272.3 ± 79.5 (187–438)
Percentages		
males	71%	70%
diabetes	12%	46%†
transient ischemic attacks	65%	41%
hypertension	73%	88%
myocardial infarction	35%	17%
atrial fibrillation	0%	0%
angina	31%‡	8%
claudication	15%	9%

*Means differ, $p = 0.04$, two-tailed t test, df = 46.
†Groups differ, $p = 0.007$, chi-square test, df = 1.
‡Groups differ, $p = 0.05$, chi-square test, df = 1.

TABLE 2 *Effects of Race on Distribution of Angiographic Lesions**

Site	Any lesion		Tight lesion†	
	White (%)	Black (%)	White (%)	Black (%)
Right vertebral artery				
1st portion	67	38	67‡	13
2nd portion	6	5	6	5
3rd portion	0	10	0	4
4th portion	27	55	27	50
Left vertebral artery				
1st portion	57‡	0	43§	0
2nd portion	11	16	11	5
3rd portion	0	11	0	0
4th portion	80	83	45	39
Basilar artery				
proximal	14	10	10	5
middle	15	21	10	16
distal	15	42§	10	16
Right PICA origin	0	0	0	0
Left PICA origin	0	5	0	5
Right AICA origin	0	0	0	0
Left AICA origin	0	0	0	0
Right SCA origin	0	10	0	16
Left SCA origin	0	0	0	0
Right PCA origin	5	20	5	15
Left PCA origin	5	14	5	5
Right PCA-2nd portion	0	5	0	0
Left PCA-2nd portion	0	5	0	0

*Based upon 14 aortic arch, 36 left vertebral, 14 right vertebral, 14 right brachial, 2 left brachial and 3 right subclavian angiograms.
†>49% stenosis.
‡Black and white groups differ, $p = 0.02$, df = 1.
§Black and white groups differ, $p = 0.05$, df = 1.

stenosis and one or more intracranial branch lesions of greater than 49% stenosis than the whites. White patients were more likely to have one or more extracranial main trunk lesions of greater than 49% stenosis. There were no racial differences in the number of occlusive lesions of greater than 0% or greater than 49% stenosis when comparisons were made for combined basilar artery sites, combined intracranial main trunk artery sites, the first segment of the vertebral artery; or for combined, extracranial main trunk artery sites of greater than 0% stenosis.

Symptomatic Loci

For each patient, a determination was made about the vascular lesions most likely to cause the patient's symptoms (either stroke or transient ischemia). At each vessel site, racial differences in the occurrence of symptomatic lesions were analyzed by two-by-two contingency tables (table 4). No racial differences were detected at individual main trunk or branch sites. However, when all symptomatic main trunk lesions were pooled and compared with symptomatic branch disease, blacks were found to have more symptomatic intracranial branch disease than whites ($p < 0.05$, df = 1, chi-square test).

TABLE 3 *Severity of Angiographic Lesions According to Race*

Site*	Degree of stenosis			
	>0%		>49%	
	White (%)	Black (%)	White (%)	Black (%)
Basilar artery	33	38	22	25
Branch arteries	7	33†	7	29‡
Extracranial arteries	48	38	48§	13
Intracranial arteries	85	92	74	79
Vertebral artery (1st portion)	63	83	48	46

*Sites have been pooled and were selected if at least one lesion was observed.
†Groups differ, $p = 0.02$, chi-square test, df = 1.
‡Groups differ, $p = 0.04$, chi-square test, df = 1.
§Groups differ, $p = 0.006$, chi-square test, df = 1.

Influence of Other Factors on the Distribution of Vascular Lesions

The black and white groups differed with respect to diastolic blood pressure, incidence of angina pectoris and incidence of diabetes mellitus. A logistic regression analysis showed that in addition to race, diabetes was the only factor which appeared to be associated with the site of occlusive disease in the posterior circulation. This association, however, could not be rigorously supported as the number of diabetic patients

TABLE 4 *Distribution of Symptomatic Lesions by Race*

Artery site*	Symptomatic sites	
	Whites (N = 27)	Blacks (N = 24)
Vertebral		
1st portion	26%	8%
2nd portion	0%	0%
3rd portion	0%	0%
4th portion	41%	25%
bilateral†	4%	8%
Basilar		
proximal	7%	4%
middle	7%	8%
distal	7%	17%
PICA origin	0%	4%
AICA origin	0%	0%
SCA origin	0%	4%
PCA origin	7%	13%
PCA-2nd portion	0%	0%
Thalamoperforant	0%	4%
Basilar branch	0%	4%

*Right and left vascular territories combined.
†Represents a combination of bilateral intracranial, extracranial and intracranial or bilateral extracranial occlusive disease.

studied was too small for conclusive results. Blood pressure, angina, TIAs, cholesterol, sex, age, claudication, atrial fibrillation, history of hypertension or history of myocardial infarction did not predict the site of posterior circulation occlusive disease.

Among non-diabetic subjects, there were significantly more intracranial lesions ($x^2 = 7.9, p = 0.005$, df = 1) and significantly more symptomatic intracranial disease ($x^2 = 4.9$, $p = 0.03$, df = 1) among blacks as compared to whites. Among diabetic subjects no differences were observed, although the statistical power may have been too low to detect such differences. In prior studies, we have found diabetes to be more prevalent among subjects with middle cerebral artery occlusive disease as compared to extracranial carotid artery disease.[23]

Discussion

In these patients with angiographically proven occlusive cerebrovascular disease, white patients had more angina pectoris, more lesions at the origin of the left vertebral artery and more high grade lesions of the extracranial vertebral arteries. Black patients had a higher mean diastolic blood pressure, more diabetes mellitus, more lesions of the distal basilar artery, more high grade lesions of intracranial branch vessels and more symptomatic intracranial branch disease.

These results suggest several racial differences in the distribution of posterior circulation occlusive disease: (1) whites have more frequent and severe extracranial disease of the vertebral artery and (2) blacks have more severe and more symptomatic intracranial branch disease.

Atherosclerotic lesions consisting of fatty streaks, fibrous plaques, calcified lesions, and complicated lesions are frequently identified at necropsy within the larger vessels of the vertebrobasilar system and do not differ qualitatively from atherosclerosis of the anterior circulation or systemic vessels.[4-10, 15] Ulceration has been less frequently identified and when present has usually involved the subclavian artery at the origin of the vertebral artery.[7] The most common site of atherosclerotic stenosis is the origin of the vertebral arteries.[1, 7, 8, 15-17] At times, subclavian plaques extend into the proximal 1–3 mm of the vertebral artery producing a ring-like encirclement of the vessel.[7, 8, 15, 16] The intracranial vertebral artery after it pierces the dura to enter the cranium (4th portion) is another frequent site of occlusive disease.[4, 5, 7, 15] Aside from these two sites, fibrous plaques and fatty streaks are distributed along the vertebral artery without any single site of predilection but ladder like arrangements of fibrous plaques are often found in the 2nd portion of the vertebral artery adjacent to cervical spondylitic bars.[7] Within the basilar artery, fatty sudanophilic plaques are more prevalent on the ventral surface and stenosis or occlusion is most frequent in the proximal 2 cm of the vessel with less stenosis distally. In the series of Castaigne et al,[4] six occlusions affected the lower third of the basilar artery, five the middle third and 3 the distal third. In another series, distal basilar atheroma was more common.[5] The proximal posterior cerebral arteries are also sites of atherosclerotic lesions but stenosis occurs here less frequently than in the middle cerebral artery.[5, 7] Atherosclerotic plaques in the major intracranial vertebral or basilar arteries can block branch orifices but the larger posterior circulation branches (PICA, AICA, SCA, anterior spinal arteries) have not been frequent sites for atherosclerotic narrowing. None of the necropsy reports describing the morphology or sites of predilection for atherosclerosis have studied or discussed racial differences.

Angiographic studies[2, 3, 18-20] have corroborated the high frequency of lesions at the vertebral origin, intradural vertebral artery[21] and basilar artery.[2, 3] Bauer et al[3] in a biracial population, found that lesions causing more than 25% obstruction or complete occlusion of cervical vessels were more common in white than black patients. In the Joint Study of Extracranial Arterial Occlusion,[22] whites consistently exceeded nonwhites in the percentage of patients with lesions judged as surgically accessible (extracranial). Other stroke studies have not addressed the issue of racial differences in the distribution of posterior circulation occlusive disease.[1, 2, 4, 5, 8]

In a study of racial differences in the distribution of anterior circulation occlusive disease, we reported more frequent and severe extracranial internal carotid artery disease in whites and more frequent and severe middle cerebral artery and supraclinoid carotid artery disease in blacks. Black patients were also younger, more often female, and had fewer TIAs.[11] When patients with intrinsic disease of the mainstem middle cerebral artery or its major subdivisions were studied in more detail and compared to patients with extracranial internal carotid artery disease, important clinical differences emerged.[23] Middle cerebral artery disease patients were more often diabetic, had fewer TIAs, and clinical deficits evolved gradually over a longer period of time. Extracranial internal carotid artery disease more often caused acute onset deficits, led to more TIAs, and more often was not associated with cerebral infarction.[23] It was postulated that internal carotid artery disease, with its known predilection for ulceration, frequently caused embolic occlusions of middle cerebral artery branches, whereas intrinsic middle cerebral artery lesions were more likely to cause deficits by a "low flow" mechanism.

Racial differences in the distribution and nature of vascular diseases have been noted by others. In a biracial study group Heyden et al[24] found a much higher ratio of middle cerebral artery occlusions to internal carotid artery occlusive lesions in blacks. Heyman et al[25] also noted more intracranial occlusive disease in blacks. Pathologic confirmation of these differences has been provided by McGarry and Solberg.[26] In our study pathologic confirmation is lacking, and the stenotic vessel wall abnormalities are assumed to be atherosclerotic in nature.

Pathologic data about other racial and ethnic groups has been reviewed by Baker.[27] The Japanese, like blacks, have a high incidence of middle cerebral artery

disease.[28] Several pathologic and arteriographic studies have confirmed this relationship.[29-34]

Our study population was a hospital-based, mixed racial group composed of 53% white patients and 47% black patients. At two of the participating centers (Michael Reese Hospital [urban] and the University of Illinois Hospital [urban]), the stroke populations were predominantly black (76%). At the other two hospitals (New England Medical Center [urban] and Loyola University Medical Center [suburban]), the stroke populations were exclusively white except for one case. Because our centers represented tertiary care facilities with both referral and comunity-based patients, we cannot exclude selection bias in subjects chosen for angiography. Other study limitations have been reviewed in our previous communication.[11]

Clinical patterns and mechanisms of posterior circulation disease have been less well studied than those in the anterior circulation.[35] Lesions of the vertebral artery origin (including subclavian steal) frequently produce transient ischemic attacks but less often stroke.[36-38] Disease of the intradural vertebral artery is a frequent cause of the lateral medullary syndrome[39] and of cerebellar infarction.[40] Bilateral intracranial vertebral artery occlusive disease has a dire prognosis due to "low flow" infarcts in the medulla and cerebellum.[41] Basilar artery disease has a variable prognosis: death or "locked-in" state[9] in some patients while others survive with little or no deficit.[42] Branch disease of the penetrating basilar branches is well known[43, 44] but intrinsic lesions of the cerebellar artery branches (PICA, AICA, SCA) and posterior cerebral arteries have not been well described. There are significant differences in the clinical picture seen with lesions at various sites in the posterior circulation.[45] Surgical and medical treatments also differ depending on the site of the vascular lesion and its mechanisms of symptom production.[46, 47] Diagnostic strategies will also vary depending on the likelihood of an extracranial or intracranial site. Non-invasive studies and intravenous DSA can define significant extracranial vertebral artery diseases but are not as useful for intracranial definition. Knowledge of the contribution of race to the distribution of posterior circulation lesions will help guide evaluation and treatment strategies for patients with vertebrobasilar occlusive disease.

References

1. Hutchinson EC, Yates PO: Carotico-vertebral stenosis. Lancet **1:** 2–8, 1957
2. Meyer JS, Sheehan S, Bauer RD: An arteriographic study of cerebrovascular disease in man. I. Stenosis and occlusion of the vertebral-basilar arterial system. Arch Neurol **2:** 27–45, 1960
3. Bauer RB, Sheehan S, Wechscler N, Meyer JS: Arteriographic study of the sites, incidence and treatment of arteriosclerotic cerebrovascular lesions. Neurology (Minneap.) **12:** 698–711, 1962
4. Castaigne P, Lhermitte F, Gautier JC, et al: Arterial occlusions in the vertebral-basilar system. A study of 44 patients with postmortem data. Brain **96:** 133–154, 1973
5. Baker AB, Iannone A: Cerebral infarction: I. The large arteries of the Circle of Willis. Neurology (Minneap.) **9:** 321–332, 1959
6. Feigin I, Budzilovich G: The general pathology of cerebrovascular disease. *In:* Vinken P, Bruyn G, eds.: Handbook of Clinical Neurology Vol. II, Vascular Diseases of the Nervous System, part I. Amsterdam: North Holland Publishing Company, pp. 128–167, 1972
7. Moossy J: Morphology, sites and epidemiology of cerebral atherosclerosis. Proc Assoc Res Nerve Ment Dis **51:** 1–22, 1966
8. Moossy J: Cerebral infarcts and the lesions of intracranial and extracranial atherosclerosis. Arch Neurol **14:** 124–128, 1966
9. Kubik C, Adams RD: Occlusions of the basilar artery; a clinical and pathologic study. Brain **69:** 73–121, 1946
10. Cornhill JF, Akins D, Hutson M, Chandler A: Localization of atherosclerotic lesions in the human basilar artery. Atherosclerosis **35:** 77–86, 1980
11. Gorelick PB, Caplan LR, Hier DB, Parker SL, Patel D: Racial differences in the distribution of anterior circulation occlusive disease. Neurology (Cleveland) **34:** 54–59, 1984
12. Mohr JP, Caplan LR, Melski JW, et al: The Harvard cooperative stroke registry: A prospective registry. Neurology (NY) **28:** 754–762, 1978
13. Alter M, Kieffer S, Resch J, Ansari K: Cerebral infarction: Clinical and angiographic correlations. Neurology (Minneap.) **22:** 590–602, 1972
14. Berenson ML, Levine DM, Goldstein M: Intermediate Statistical Methods and Applications. Englewood Cliffs, New Jersey: Prentice-Hall, 1983
15. Fisher CM, Gore I, Okabe N, White PD: Atherosclerosis of the carotid and vertebral arteries — extracranial and intracranial. J Neuropathol Exp Neurol **24:** 455–476, 1965
16. Martin MJ, Whisnant JP, Sayre GP: Occlusive vascular disease in the extracranial cerebral circulation. Arch Neurol **5:** 530–538, 1960
17. Schwartz CJ, Mitchell JRA: Atheroma of the carotid and vertebral arterial systems. Br Med J **2:** 1057–1063, 1961
18. Stein B, McCormick W, Rodriguez J, Taveras J: Incidence and significance of occlusive vascular disease of the extracranial arteries as documented by postmortem angiography. Trans Am Neurol Assoc **86:** 60–66, 1961
19. Udea K, Toole J, McHenry L: Carotid and vertebrobasilar ischemic attacks: clinical and angiographic correlation. Neurology (NY) **29:** 1094–1101, 1979
20. Callow A, Moran J, Kahn P, Deterling R: Human atherosclerosis: Vascular surgeon's viewpoint. Ann NY Acad Sci **149:** 974–988, 1968
21. Thompson J, Simmons C, Hasso A, Hinshaw D: Occlusion of the intradural vertebrobasilar artery. Neuroradiology **14:** 219–229, 1978
22. Fields WS, North RR, Hass WK, et al: Joint study of extracranial arterial occlusion as a cause of stroke. I. Organization of study and survey of patient population. JAMA **203:** 153–158, 1968
23. Caplan L, Babikian V, Helgason C, et al: Middle cerebral artery occlusive disease: clinical and epidemiological features. Neurology **34** (suppl 1): 200, 1984
24. Heyden S, Heyman A, Goree JA: Nonembolic occlusion of the middle cerebral and carotid arteries: A comparison of predisposing factors. Stroke **1:** 363–369, 1970
25. Heyman A, Fields WS, Keating RD: Joint study of extracranial arterial occlusion. VI. Racial differences in hospitalized patients with ischemic stroke. JAMA **222:** 285–289, 1972
26. Solberg LA, McGarry P: Cerebral atherosclerosis in Negroes and Caucasians. Atherosclerosis **16:** 141–154, 1972
27. Baker AB: The geographic pathology of atherosclerosis: A review of the literature with some personal observations on cerebral atherosclerosis. *In:* Tower DB, ed.: The Clinical Neurosciences. New York: Raven Press, 1975, pp. 137–146
28. Barnett HJM: The international collaboration study of superficial temporal artery — middle cerebral artery anastomosis. *In:* Rose FC, ed.: Advances in Stroke Therapy. New York: Raven Press, 1982, pp. 179–182
29. Kieffer SA, Takeya Y, Resch JA, Amplatz K: Racial differences in cerebrovascular disease: Angiographic evaluation of Japanese and American populations. AJR **101:** 94–99, 1967
30. Tomita T, Mihara H: Cerebral angiographic study on C.V.D. in Japan. Angiology **23:** 228–239, 1972
31. Brust RW Jr: Patterns of cerebrovascular disease in Japanese and other population groups in Hawaii: An angiographical study. Stroke **6:** 539–542, 1975
32. Mitsuyama Y, Thompson LR, Hayashi T, et al: Autopsy study of

cerebrovascular disease in Japanese men who lived in Hiroshima, Japan and Honolulu, Hawaii. Stroke **10:** 389–395, 1979
33. Kameyama M, Okinaka S: Collateral circulation of the brain: With special reference to atherosclerosis of the major cervical and cerebral arteries. Neurology (Minneap.) **13:** 279–286, 1963
34. Nishimaru K, McHenry Jr. LC, Toole JF: Cerebral angiographic and clinical differences in carotid system transient ischemic attacks between American Caucasian and Japanese patients. Stroke **15:** 56–59, 1984
35. Caplan L: Vertebrobasilar disease: time for a new strategy. Stroke **12:** 111–114, 1981
36. Baker R, Rosenbaum A, Caplan L: Subclavian steal syndrome. Contemporary Surgery pp. 96–104, 1974
37. Fisher CM: Occlusion of the vertebral arteries. Arch Neurol 22:13–19, 1970
38. Moufarrij NA, Little JR, Furlan AJ, Williams G, Marzewski DJ: Vertebral artery stenosis: Long-term follow-up. Stroke **15:** 260–263, 1984
39. Fisher CM, Karnes W, Kubik C: Lateral medullary infarction: the pattern of vascular occlusion. J Neuropath Exp Neurol **20:** 323–379, 1961
40. Sypert G, Alvord E: Cerebellar infarction: a clinicopathological study. Arch Neurol **32:** 357–363, 1975
41. Caplan LR: Bilateral distal vertebral artery occlusion. Neurology (NY) **33:** 552–558, 1983
42. Caplan LR: Occlusion of the vertebral or basilar artery. Follow-up analysis of some patients with benign outcome. Stroke **10:** 277–282, 1979
43. Fisher CM, Caplan LR: Basilar artery branch occlusion: A cause of pontine infarction. Neurology **21:** 900–905, 1971
44. Fisher CM: Bilateral occlusion of basilar artery branches. J Neurol Neurosurg Psychiatry **40:** 1182–1189, 1977
45. Caplan LR: Vertebrobasilar occlusive disease. *In:* Barnett HJM, Mohr JP, Stein B, Yatsu F, eds.: Stroke. Churchill Livingstone Company (In Press)
46. Imparato AM, Riles TS, Kim G-E: Cervical vertebral angioplasty for brainstem ischemia. Surgery **90:** 842–852, 1981
47. Ausman J, Diaz F, de los Reyes A, et al: Extracranial — intracranial anastomoses in the posterior circulation. *In:* Berguer R, Bauer R, eds.: Vertebrobasilar Arterial Disease. New York: Raven Press, 1984, pp 313–319

Baseline Hemodynamic State and Response to Hemodilution in Patients with Acute Cerebral Ischemia

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SUMMARY Hemodynamic data were obtained in 9 patients (mean age 65 yrs) with carotid territory cerebral infarct within the preceding 24 hours (mean 14 ± 8) as part of a pilot study testing the feasibility and safety of hypervolemic hemodilution. Pulmonary arterial catheters (PACs) were placed without complication in all patients, and after baseline measurements were obtained, up to 1500 cc of 6% hetastarch in 0.9% sodium chloride was administered the first day and up to 1000 cc per day the second and third days. Pulmonary wedge pressure (PWP) rose from 6.3 ± 3.5 to 14.4 ± 3.4 mm Hg ($p < 0.001$) without development of congestive heart failure in any patient. This was accompanied by a drop in hematocrit (Hct) from 40.3 ± 3.4 to 32.9 ± 2.0 ($p < 0.001$) and rise in cardiac output (CO) from 4.3 ± 1.0 to 5.3 ± 0.6 ($p < 0.05$). Phlebotomy of 250 cc was performed in 2 patients and 500 cc in one in order to reduce Hct to desired levels. The volume of fluid needed to raise PWP to 15 was unpredictable (2361 ± 1106 cc) and therefore PACs were necessary to monitor the rate and volume of fluid administration. The data show that PWP is sufficiently low and Hct sufficiently high following stroke in most patients that hemodilution by volume expansion with phlebotomy added if necessary can be undertaken safely with appropriate monitoring of hemodynamic function, and that this therapy results in optimal reduction of Hct and increased CO without risk of hypotension.

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HEMODILUTION THERAPY is presently the subject of intensive evaluation for patients with acute cerebral infarction. The rationale for this therapy assumes that patients with stroke will benefit from raising cerebral blood flow (CBF) by lowering hematocrit (Hct), the major determinant of whole blood viscosity.[1–5] A second benefit might result from raising cardiac output (CO) by reducing peripheral vascular resistance (caused by lowering viscosity)[6] and by expanding left ventricular end diastolic volume in those patients undergoing "hypervolemic" hemodilution.[7] An increase in CBF and CO might result in improved perfusion of potentially viable ischemic tissue which has been identified in some patients with acute stroke using positron emission tomography.[8–10]

Despite the interest in this form of therapy and its basis in cardiovascular physiology, there are no published data on the baseline hemodynamic status of patients entering the hospital with acute strokes. Such data are necessary for designing protocols that will optimize the desired effects on viscosity and cardiac output and that will be safe in patients who often have impaired cardiac function.[11]

This study presents data from 9 patients who under-

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Received February 28, 1985; revision #1 accepted June 5, 1985.