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Race, Sex and Occlusive Cerebrovascular Disease: A Review

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IDEALLY, treatment of a patient with cerebral ischemia should be guided by knowledge of the nature, location, and severity of the occlusive disease within the extracranial and intracranial vessels.[1] To plan efficient evaluation of the individual patient, the responsible clinician must be familiar with the relative probability of finding occlusive lesions at various sites within the vascular tree since individual imaging and non-invasive tests have different capabilities with regard to various vascular loci.[2] A growing body of data suggests that there are important differences in the distribution of occlusive vascular disease in Blacks and Whites and in men and women. This review summarizes the presently available data.

Early Pathological Studies in Predominantly White Patient Populations

In 1951, Fisher reviewed the autopsy results of 200 patients with cerebrovascular disease and did not find a single example of occlusion of the middle cerebral artery (MCA).[3] Previously, most patients with anterior circulation infarcts were given the clinical diagnosis of MCA occlusion. In Fisher's experience, internal and carotid artery (ICA) occlusion was more common.[3,4] Adams and Vander Eecken[5] found that the commonest sites of occlusive cerebrovascular lesions were the origins of the ICA, the sigmoid portions of the intracranial ICA, the first 3 to 4 cm. of the proximal MCAs, the anterior cerebral arteries (ACA) just proximal and distal to the anterior communicating artery, and the proximal vertebral, basilar, and posterior cerebral arteries (PCA).

Using a dissecting technique that involved the removal of the vertebral arteries with the cerebral vertebrae en bloc, Hutchinson and Yates noted that atherosclerosis of the vertebral artery was most prevalent in the proximal portion of the vertebral artery and often was contiguous with atheroma within the parent subclavian artery.[6] The severity of atheroma within the proximal vertebral artery often paralleled that within the proximal ICA.[7] Baker and Iannone described the location and severity of atherosclerosis in 173 consecutive autopsies.[8] Lesion grading was based on fatty deposits and calcification; luminal stenosis was only one factor studied. The most common sites of involvement were the ICA origin and distal basilar artery; the proximal and mid-portion of the basilar artery, and the MCA were next most frequently involved, followed by the vertebral arteries and PCAs. The posterior inferior cerebellar arteries (PICA), superior cerebellar arteries (SCA), and the distal ACA were frequently spared from atherosclerosis.[8]

Martin, Whisnant, and Sayre studied the pathological changes in the extracranial carotid and vertebral circulations in 100 autopsied patients 50 years of age or older.[9] The most severe and frequent atherosclerotic lesions were found in the proximal 2 cm. of the ICA. The common carotid arteries (CCA) were less severely involved, and the innominate artery was seldom the site of severe atherosclerosis. Whisnant and colleagues confirmed the frequency of proximal ICA atheroma.[10] Swartz and Mitchell in England studied 93 consecutive autopsies, and Torvik and Jorgenson[12] in a Norwegian study of 994 autopsy cases, corroborated the frequency and importance of occlusive disease at the ICA origin and the intracranial ICA. Fisher and colleagues studied extracranial and intracranial atherosclerosis in 178 routine autopsies.[13] All 15 ICA occlusions were located within the proximal portion of this artery. No example of thrombotic occlusion of an ICA branch was found. There were also 5 vertebral artery occlusions within the neck, and 6 intracranial posterior circulation occlusions usually within the intracranial veretebral and basilar arteries.[13] Lhermitte and colleagues[14] and Blackwood et al[15] noted that intracranial occlusions of the MCA were usually of embolic origin. Castaigne and colleagues studied the extracranial carotid arteries at post-mortem in 29 patients with cerebrovascular disease and 21 other neurological patients.[16] The ICA was occluded unilaterally by atherosclerosis and thrombosis in 25 cases; 16 at the ICA origin, 3 at the carotid siphon, and in 6, the origin of the thrombus could not be determined. Twelve patients had bilateral ICA occlusions most often within the carotid sinus.[16] Castaigne and colleagues also studied 44 patients with occlusive disease within the posterior circulation.[17] Twenty-two of 25 vertebral artery occlusions were caused by atherosclerosis; 17 of these resulted from a thrombus forming at a site of severe atherosclerotic stenosis. The primary sites of thrombosis were the intracranial vertebral artery in 12 and the proximal vertebral artery in 4. Seventeen occlusions of the basilar artery were due to atherosclerosis and one to embolism. Among 30 PCA territory occlusions, the main cause was embolism originating from either the heart or a more proximal vascular occlusive lesion, or direct extension of an occlusion from within the basilar artery.[17]

The vascular lesions described so far could be noted by gross inspection of the larger extracranial and intracranial arteries. Interest and data was also accumulating about disease of penetrating arteries visible only under the microscope. Fisher, in a series of articles spanning 2 decades, analyzed the pathological lesions within these penetrating arteries in patients with lacu-

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nar infarcts.[18-21] The microscopic pathology of these vessels, less than 200 micra in diameter, included fibrinoid degeneration of the vessel walls with hyaline material within the vascular media, and small microaneurysms. Fisher called these lesions "lipohyalinosis" and, using serial sections, documented that lacunar infarcts resulted from ischemia distal to arterial disorganization and disruption of the vessel wall secondary to the fibrinoid degeneration. The arteries involved were deep penetrating branches of the major cerebral arteries including: medial striate penetrators from the ACA, lateral lenticulostriate branches of the MCA, capsular penetrating branches from the anterior choroidal artery (AChA), thalamic perforating branches of the basilar and communicating arteries, and paramedian penetrators to the medullary and pontine bases from the vertebral and basilar arteries. These penetrating arteries are distinct anatomically from the larger circumferential arteries and are especially vulnerable to damage secondary to hypertension. Others have also described and analyzed these changes in small intracerebral arteries.[22-24] The same pathology, lipohyalinosis, fibrinoid degeneration, and microaneurysms leads to intraparenchymatous hemorrhages (ICH) when leakage from these vessels occurs.[25-26] Lacunar infarcts and ICH share similar locations within the brain.

Fisher's Schema of Ischemic Infarction

Using data from his own autopsy studies, and those from England and France, Fisher devised a construct which he taught to succeeding neurology residents and stroke fellows including the senior authors. (LRC & DBH) In this system, patients with ischemic infarcts are classified into 1 of 3 categories.

Group 1. Atherosclerosis of larger arteries. These lesions preferentially affect the ICA and vertebral artery origins extracranially. The ICA siphon, intracranial vertebral and basilar arteries are also often diseased but intracranial stenosis of the MCA, ACA, and PCA is less common.

Group 2. Fibrinoid degeneration — Lipohyalinosis of smaller arteries causes lacunar infarcts in deep penetrating vessel territories (basal ganglia, internal capsule, thalamus, and pons.)

Group 3. Thrombi within circumferential arteries on the brain's surface and the medium sized arteries (MCA, ACA, PCA and their major superficial branches) is most often caused by embolism from the heart, proximal arterial system or from direct extension of thrombi within the proximal arterial system. Medium sized vessel occlusion also results from nonatheromatous causes such as coagulopathies, oral contraceptives etc. and is commoner in women.[27]

Clinical and Angiographic Data from Predominantly White Patient Populations

Angiographic studies corroborated the distribution of atherosclerotic lesions found at autopsy. Callow and colleagues analyzed the findings from 4-vessel angiography in 100 white patients;[28] 341 stenotic lesions were noted. The proximal ICAs (38% of lesions) and proximal vertebral arteries (22%) most often harbored atheromatous lesions while intracranial sites favored the basilar artery and ICA siphon. Extracranial ICA origin lesions were very common in the Joint Study of Extracranial Arterial Occlusion.[29-31] Toole and colleagues also found a high incidence of atherosclerotic lesions of the extracranial ICA origin among 160 patients with transient ischemic attacks (TIAs) studied angiographically.[32]

The Framingham Study[33-36] and Harvard Stroke Registry[37] added epidemiological and clinical data in large groups of almost exclusively white patients. Hypertension was very common in patients with atherosclerotic brain infarctions. Patients with severe ICA origin stenosis or occlusions had a high incidence of accompanying coronary and peripheral vascular disease, hypercholesterolemia, and TIAs.[37] Other studies documented the striking frequency of coexistent coronary and extracranial ICA disease, and the high death rate from coronary artery disease in patients with TIA or stroke due to extracranial ICA disease. Angiographic studies in the HSR also corroborated the distribution of angiographic lesions predicted by Fisher's schema.[37] This data helped to further characterize Fisher's group 1 atherosclerotic patients. White patients with atherosclerotic lesions of the ICA origin had a high frequency of coronary and peripheral vascular disease, hypertension, hyperlipidemia, and TIAs. In one study, 32.8% of patients with symptomatic peripheral vascular disease had significant associated extracranial arterial occlusive disease.[41]

Our Own Data in Mixed but Predominantly Black Patient Populations

In a racially mixed population at the Michael Reese Hospital in Chicago, Fisher's schema was not accurate in predicting the distribution of vascular occlusive disease in Blacks. Formal studies were devised to study these observed racial differences in angiographically detected lesions in a mixed but predominantly Black urban population.

Clinical and angiographic features of 26 White and 45 Black patients with symptomatic anterior circulation occlusive disease were studied.[42] Angiograms were examined blinded to clinical and racial data. White patients had more TIAs ($p < 0.01$) more carotid bruits ($p < 0.05$), more ICA origin angiographic lesions ($p < 0.05$), more high grade ICA origin lesions ($p < 0.05$), a greater mean degree of stenosis of the ICA origin ($p < 0.05$) and more symptomatic lesions at the ICA origin ($p < 0.001$). Black patients had more angiographic lesions of the supraclinoid ICA ($p < 0.05$) and MCA stem ($p < 0.05$), a higher degree of mean stenosis of the supraclinoid ICA ($p < 0.05$) and middle cerebral artery stem ($p < 0.05$) and more symptomatic lesions of the main stem MCA ($p < 0.02$).[42] Race was the only significant factor that predicted the location of vascular lesions. Other risk factors including hypertension were not predictive of lesion site. In general, White patients had more lesions, more symptomatic lesions, and more high grade le-

sions along the extracranial ICA, while Blacks had a predominance of intracranial vascular lesions.

Next, the question of racial distribution and clinical features of anterior circulation occlusive disease was studied by reviewing personal cases and Michael Reese Stroke Registry patients selected because of severe ICA or MCA occlusive disease.[43] MCA disease patients were predominantly Black, and female whereas ICA patients were usually White and male. ICA disease patients more often had TIAs and had a higher frequency of coexisting systemic coronary and peripheral vascular disease and cardiac death.[43]

Clinical and angiographic features among 27 White and 24 Black patients with symptomatic posterior circulation disease and abnormal angiography were analyzed.[44] We found Whites had significantly more angina pectoris ($p = 0.05$) more lesions of the left vertebral artery origin ($p = 0.02$) and more tight stenosis of the extracranial vertebral artery origins ($p = 0.06$). Blacks had higher mean diastolic blood pressure ($p = 0.04$), more lesions of the distal basilar artery ($p = 0.05$) and more tight ($p = .04$) and symptomatic ($p = 0.05$) lesions of the intracranial posterior circulation branch vessels (PICA, AICA, SCA and proximal portions of the FCA). Logistic regression analysis, odds ratio determinations, and Mantel-Haenszel statistics revealed that race, was the only factor that increased the risk of intracranial posterior circulation occlusive disease.[44]

Results from the Pilot Stroke Data Bank (PSDB)[45] and the Stroke Data Bank (SDB)[46] also corroborated racial and gender differences among patients with angiographically documented occlusive vascular lesions. There were 1144 patients in the PSDB; 43.8% were Black and 43% were female. This series did not include patients from Michael Reese Hospital. Four hundred and eight had angiography. Forty-four patients had greater than 50% stenosis of the ICA origin of whom 86.4% were White and 59.1% were male. Only 12 patients had greater than 50% stenosis of the MCA documenting the relative rarity of that lesion. In the group with MCA lesions 9 (75%) were White and 9 were women. The frequency of hypertension, diabetes and heart disease is outlined in table 1. Hypertension and heart disease were more common in patients with ICA origin disease while diabetes was more often noted in patients with intracranial ICA or MCA disease.

Preliminary data were also analyzed from the main phase SDB.[46] Though there were 816 patients, only 275 had angiography. Again the number of patients with MCA occlusive disease was small. Two patients had greater than 50% stenosis of both the ICA siphon and MCA and are noted in each of those groups in table 2. In this study, patients with MCA disease or with intracranial disease (MCA + ICA siphon) were predominantly Black and female while ICA origin patients were White and male. In this study a history of hypertension was more common in the patients with intracranial disease. TIA was more common in patients with lesions of the ICA origin as has been noted in our previous studies.[42, 43]

None of the studies cited in mixed racial groups can even remotely be considered epidemiologically valid. Michael Reese Hospital and the other Data Bank centers are tertiary referral centers that receive selected groups of patients because of their known interest in stroke and their geographical locations. Bias on the part of the neurologists might have affected selection of cases for angiography and so inclusion in the studies cited. Analysis of this possible angiographic selection bias argues against preselection as the explanation for the observed racial differences.[42] Socioeconomic, dietary, smoking, exercise and other factors were not analyzed systematically. The inner city urban population served by Michael Reese and other participating hospitals was predominantly poor and Black, while the White population was more often suburban, older, and wealthier. Life styles, occupations, diet and many factors other than race separate these groups. The data is purely observational and must be studied prospectively in other populations using valid epidemiological techniques to be validated.

Angiographic and Pathologic Data From Other Studies Regarding Cerebrovascular Lesions in Blacks and Whites

Bauer and colleagues studied the distribution of occlusive sites by cerebral angiography in a racially mixed population of patients with symptomatic cerebrovascular disease.[47] Diffuse vascular tortuosity and dilatations of vessels were more frequent in Blacks, while large atherosclerotic plaques in proximal cerebral arteries were more frequent in Whites. In the Joint Study of Extracranial Arterial Occlusion, surgically

TABLE 1 *Pilot Stroke Data Bank (PSDB)*

	n	Blacks (%)	Female (%)	Hypertension (%)	Diabetes (%)	Vascular or heart disease (%)	Average age (yrs.)
All patients	1144	43.8	43	63.5	21.2	23	63
All angiogrammed patients	408	33.7	36	61.3	21.7	23.6	64
ICA-B > 50% stenosis	44	13.6	40.9	72.7	25.6	28.6	60
ICA-S > 50% stenosis	15	33	40	73	46.7	23	55.2
MCA > 50% stenosis	12	25	66.7	45	41.7	18.2	57.5

ICA-B = internal carotid artery bifurcation; ICA-S = internal carotid artery; MCA = middle cerebral artery; hypertension = history of BP > 150/90; vascular or heart disease = angina, MI, claudication.

TABLE 2 *Stroke Data Bank (SDB)*

	n	Blacks (%)	Female (%)	History Hypertension (%)	Diabetes (%)	Vascular or heart disease (%)	Average age (yrs.)	TIA* (%)
All patients	816	60.8	55.5	63	20.8	21	63.9	14.7
All angiogrammed patients	275	53	55	51.3	15.3	15.3	54	17
ICA-B > 50% stenosis	25	40	32	56	28	36	59.6	43
ICA-S > 50% stenosis	7	85.7	57	100	28.6	0	59.7	40
MCA > 50% stenosis	18	83.3	55.5	83.3	27.7	27.7	60.2	17
Intracranial > 50% stenosis (ICA-S + MCA)	23	82.6	56.5	86.4	30.4	21.7	60	25

*All patients in SDB had stroke — none just TIA.
ICA-B = internal carotid artery bifurcation; ICA-S = internal carotid artery siphon; MCA = middle cerebral artery; hypertension = history of BP > 150/90; vascular or heart disease = angina, MI, claudication.

accessible extracranial disease was much more common in white patients.[30] In this study there were 956 examples of unilateral ICA occlusion and 88 bilateral ICA occlusions.[31] Ninety-one percent of patients with ICA occlusions were White, while only 9% were Black.[31] Racial differences were even more striking in the group of patients with subclavian steal due to occlusive disease of the proximal subclavian artery. Ninety-eight percent of patients with subclavian steal were White.[48] Blacks in the Joint Study, however, had a higher frequency of occlusive disease in the intracranial arteries.[49]

Heyden et al analyzed a group of patients with angiographically documented non-embolic occlusion of the ICA and MCA and noted a preponderance of blacks in the group with intracranial disease.[50] Extracranial disease patients also had a high frequency of associated ischemic heart disease, intermittent claudication, and hypercholesterolemia. The ratio of MCA to ICA occlusive disease in Whites was 13/84 whereas in Blacks it was 10/1.[50] Eighty-four of the 85 patients with only ICA disease were White while among 23 MCA lesions 43% were Black.[50] Russo also found a disproportionately small number of Black TIA patients had proximal ICA disease, and race was the only factor that correlated with the presence of extracranial disease documented by angiography.[51]

Autopsy studies of mixed racial populations have also shown differences between Blacks and Whites. The largest bank of data is the International Atherosclerosis Project.[52-54] Twenty-one hundred and sixty-six autopsies were performed on "unselected" patients whose death was a result of a diversity of causes including Blacks and Whites from New Orleans, Blacks from Jamaica, and Whites from Oslo, Norway. Investigators noted the presence of fatty streaks, the percentage of vascular surface area involved with fatty streaks and the number of raised lesions. Stenosis was not prominently weighted in judging the severity of atherosclerosis. The extracranial carotid and vertebral arteries, and the intracranial MCA and basilar arteries were routinely studied as well as the aorta, coronary arteries, and systemic vessels. When Blacks and Whites from New Orleans were compared, Blacks had significantly more intracranial arterosclerosis. In the 65–69 year age group 43% of Blacks had raised atherosclerotic intracranial lesions compared to only 8.5% among Whites.[53] Whites had more coronary and aortic atheromas but the cerebral arteries were about equally affected in Whites and Blacks.

Resch,[55] Williams[56] and Baker and colleagues[57] also studied the racial and geographic distribution of atherosclerosis on several continents. These authors graded atherosclerotic plaques within the Circle of Willis on a scale of 1–4 but only grade 4 included severe luminal stenosis. The prevalence and severity of cerebral atherosclerosis was less in the Nigerian Negro than in the Minnesota population. Blacks from Alabama had comparable findings to American Whites. In a study of systemic and cerebral arteries in Bantu Blacks, intracranial lesions were not common but the MCA was more affected than any other intracranial vessel. Disease of the carotid arteries was rare.[58]

These angiographic and autopsy studies all confirm that Blacks were more prone to develop intracranial atherosclerosis while Whites had more extracranial disease. Blacks from different geographical localities had, however, varied incidences of cerebral vascular disease.

Japanese, like Blacks, also have a predilection for occlusive lesions of the MCA, and a low prevalence of occlusive extracranial ICA disease.[59-65] Recent data from the International Study of Extracranial to Intracranial Bypass also corroborated, that, in Japan, MCA occlusive disease was a more common indication for STA-MCA bypass than in predominantly White North American patients.[66] When atherosclerosis of the Circle of Willis was compared in patients from Japan and Minnesota, occlusive lesions were usually found in the large caliber vessels of the circulation in the Minnesota populace, while small caliber vessels were more often involved in Japan.[67] Though we could not find definitive published data that formally documented the distribution of cerebrovascular occlusive lesions in China, neurologists there believe that angiography of patients with ischemic stroke frequency reveals MCA occlusive disease, and ICA disease is unusual.[68] Japa-

nese, Chinese, and Blacks also share a low incidence of coronary artery disease, and a high incidence of hypertension and intracerebral hemorrhage.

Sex Differences in the Distribution of Occlusive Vascular Lesions

Nearly all series of referred patients with well documented occlusive disease of the extracranial arteries show a male preponderance. Table 3 depicts 12 series of patients with extracranial occlusive disease. The selection criteria vary in the individual series from slight degrees of stenosis to complete occlusion to a lesion felt by the surgeon to warrant endarterectomy.

These are not population studies. Men have a known higher incidence of coronary artery disease. Detection of bruits or occlusive lesions by non-invasive studies or angiography could have been biased by the referral patterns for coronary artery bypass surgery in these centers. In the series of Toole et al, the presence of TIA was the criterion for admission to the group and 88% of these patients had significant extracranial occlusive disease (those without extracranial occlusive disease were not specifically broken down by race or sex.) Though criteria for selection of patients for carotid endarterectomy clearly varied from center to center and among surgeons in the same center, it is unlikely that sex had an important independent effect on the decision to operate. Each series shows a male preponderance of patients with significant extracranial occlusive disease of the ICA. The cumulative figures show that 66% of 8,314 patients with carotid lesions in these 12 series were male. In a recent study of patients presenting for evaluation of carotid bruits, Ford et al found that, among symptomatic patients who also had TIA, 56% were male. Twice as many men as women had ICA stenosis as determined by non-invasive tests or angiography, while asymptomatic bruit without stenosis occurred in 38 men and 63 women (62% female predominance).[77] Data regarding other extracranial arteries is scanty. In the Joint Study of Extracranial Occlusion, 113 (67%) of 168 patients with subclavian artery occlusive disease and subclavian steal were male.[48]

Data regarding intracranial occlusive disease is quite meager when contrasted to extracranial disease. When MCA lesions secondary to intrinsic occlusive disease were studied, 4 of 16,[78] 13 of 20,[43] 8 of 12,[45] and 10 of 18,[46] were female. Though these numbers are small, they do show a relative female preponderance for intracranial disease that is in striking contrast to the male preponderance of extracranial disease. Sex differences have also been noted in autopsy materials. Flora, Baker and colleagues using grading criteria already commented upon, analyzed 5,033 consecutive autopsies.[77] From the 4th to 6th decades of life the percentage of females with no cerebral atherosclerosis was higher than males, but beyond age 65 the frequency of atherosclerotic lesions was the same in the two sexes. Of interest, diabetic women had more cerebral atherosclerosis than non-diabetic men, and after the 4th decade they had at least as much cerebral atherosclerotis as diabetic males.[79] In the International Atherosclerosis Project, consistent sex differences in carotid and vertebral artery atherosclerosis were found with males having more significant disease.[52-54] Raised atherosclerotic lesions were also more common among males in the intracranial arteries (this included the vertebral and basilar arteries as well as the MCA.)

Race and Sex Differences in Other Systemic Vessels

Racial and sex differences in the distribution of extracranial and cerebral vascular occlusive disease are not unique findings restricted to the cerebral circulation. Volumes of literature also note important race and sex differences in the incidence of atherosclerosis of the aorta, coronary, renal, and peripheral arteries.[80] Prevalence of hypertension also varies by sex and race. Space will not allow full review of this data so that we have chosen only selected important studies to give the reader a brief introduction to this vast subject.

Clinical and pathological studies note a relatively

TABLE 3 *Gender in ICA Disease*

Author	Criteria	n	Men (%)	Women (%)
1. Cote, Barnett, Taylor[69]	occlusion	47	83	17
2. Toole, Janeway, Choi[32]	TIA (88% significant extracranial disease)	160	67	33
3. Fields, Lemak[31]	occlusion	1044	77	23
4. Pilot SDB[45]	> 50% stenosis (angio)	44	59	41
5. SDB[46]	> 50% stenosis (angio)	25	68	32
6. Brott, Thalinger[70]	carotid endarterectomy	371	60	40
7. Slavish, Nichols, Gee[71]	carotid endarterectomy	43	63	37
8. Rowed, Noris[72]	carotid endarterectomy	276	70	30
9. Mayo Clinic (Sundt)[74]	carotid endarterectomy	2036	69	31
10. Am. Assoc. N.S. (Sundt)[75]	carotid endarterectomy	3328	62	38
11. Norris[73]	> 75% stenosis (non-invasive)	110	59	41
12. Ackerman[76]	< 2 mm lumen (non-invasive)	130	68	32
Totals		8314	66	34

low frequency of coronary artery disease in Blacks. In the International Atherosclerosis Project, 23,207 sets of coronary arteries and aortas collected from a variety of geographical locales were studied.[52, 81] White patients from New Orleans and Oslo had the highest frequency of raised coronary occlusive lesions and Blacks had consistently less atheromatous coronary artery disease.[81] Men had more severe coronary artery disease than woman but in Blacks, men and women had nearly equal severity of coronary artery disease.[81]

In series of patients with myocardial infarction studied in racially mixed populations in the southern United States,[82-85] myocardial infarction was much less common in Blacks than Whites. In Africa, though hypertension was common among urban, native Africans, among 434 autopsied black hypertensive patients at one hospital there was only a 2.2% incidence of myocardial infarction.[86] In this same group of patients, there were 41.9% "cerebrovascular complications" of which 95% were intracerebral hemorrhages.[86]

Every study of hypertension has documented a strikingly high prevalence of this condition in Black men and women. In the national health survey of the years 1960–1962, 27% of Blacks were hypertensive as compared to 14% of Whites.[87] In a 1971 survey that screened 1,759 Black Baltimore residents, 41% of men and 33% of women had diastolic blood pressures over 95 torr.[88] Other studies have documented higher average blood pressure readings in Blacks when compared with Whites throughout the United States.[89-92] Black women seem to have more hypertension than Black men. In White hypertensive patients the level of blood pressure elevation is approximately equal in men and women until age 45 when it becomes greater in women.[89, 90]

There are also prominent differences in renal vascular disease in Blacks and Whites.[93-98] Rostand et al noted a striking preponderance of Blacks over Whites among patients with end-stage renal failure and hypertension in Alabama.[93] Despite equal severity of hypertension and similar degrees of hypertensive cardiac changes, Blacks in one study had less renal blood flow (390 + 35 mm/min/m Blacks vs. 473 + 17 mm/min/m in Whites) and more severe disease of the intrarenal medium sized arteries determined angiographically.[94] Renal vascular hypertension due to stenosis of a renal artery is also much less common among Blacks.[89] In a Dallas clinic population, less than 10% of cases of renal vascular hypertension were in Blacks even though Blacks made up 75% of the population evaluated.[98]

Conclusion

The data available to date and herein reviewed allows modification of the 3 groups in Fisher's original schema and the important addition of a 4th, previously under–recognized group (table 4). Group 1, atherosclerosis of the larger extracranial arteries is more prevalent in Whites and men and is commonly accompanied by hypercholesterolemia and occlusive disease of the coronary arteries and arteries to the limbs. Groups 2 and 3 are unchanged though presently there have been no studies to compare the morphology, severity, or distribution of penetrating small vessel disease by race or sex. Group 4 consists of patients with occlusive disease of the intracranial arteries. Medium sized intracranial arteries and their major branches, ACA, MCA, MCA upper division branch, PCA, PICA, AICA, and SCA, and the distal basilar artery are most often affected. Morphological details of these intracranial lesions and comparison with patients in groups 1 and 3 are not presently available. Group 4

TABLE 4 *New Schema of Occlusive Cerebrovascular Disease*

	Vascular Locale	Race	Gender	Serum Lipids	Other Vascular Disease	Hypertension
Group 1	extracranial large arteries ICA and vertebral origins intracranial basilar artery	White	M>F	+ + +	coronary subclavian iliac renal	+ +
Group 2	microscopic deep penetrating branches of medium sized arteries lenticulostriate thalamostriate penetrating pontine	—	—	+	no	+ + + +
Group 3	medium sized intracranial arteries and main branches MCA, ACA, PCA	—	—	—	cardiac disease as embolic source Also Group 1 patients with artery to artery emboli from proximal extracranial arteries	
Group 4	superficial branches of medium-sized intracranial arteries and vessels of Group 3 and PICA, AICA, SCA	Black Oriental	F≥M	—	—	+ + +

MCA = middle cerebral artery; ACA = anterior cerebral artery; PCA = posterior cerebral artery; PICA = posterior inferior cerebellar artery; AICA = anterior inferior cerebellar artery; SCA = superior cerebral artery.

patients are more often of Black or oriental origin and are probably disproportionately more often female when compared to Group 1. There is a low incidence of accompanying coronary and peripheral vascular disease and renal vascular hypertension. Hypertension is also very common in Group 4 patients but hypercholestrolemia is not.

The information available, though voluminous, is still very inadequate. Several vessel sites, especially the ICA siphon and intracranial vertebral artery, do not, as yet, fall clearly into any of these groups. Comparative morphological data, analyses using newer genetic techniques, biochemical lipid analysis, and platelet studies are meager. We have not even alluded to possible explanations for these racial and sex differences because of space limitations, but suffice it to say that the reasons for these racial and sex differences in the epidemiology and distribution of occlusive vascular disease are unknown. Perhaps therein are hidden clues to help unlock the puzzle of the disorder we loosely call "atherosclerosis."

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