Pattern of Cerebral Atherosclerosis in Hong Kong Chinese
Severity in Intracranial and Extracranial Vessels

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Background and Purpose: The clinical pattern of stroke and the angiographic distribution of cerebral atherosclerosis in Chinese are different from those in Caucasians. Pathological data from autopsy studies are lacking.

Methods: The intracranial and extracranial arteries supplying the brains of 114 consecutive Chinese patients undergoing autopsy in a regional general hospital were examined by computer-assisted morphometric analysis under a microscope as well as by macroscopic grading for atherosclerotic narrowing. The severity was correlated with various atherosclerosis-related factors.

Results: Atherosclerosis of the intracranial cerebral vessels was more severe than that of the extracranial vessels. The distal branches of the intracranial vessels were also commonly involved. Hypertension and diabetes mellitus were identified as factors associated only with intracranial atherosclerosis ($p<0.001$), whereas ischemic heart disease was associated with atherosclerosis in both the intracranial ($p<0.001$) and extracranial ($p=0.012$) vessels. Smoking was associated with narrowing of the extracranial vessels only ($p=0.0054$).

Conclusions: Compared with figures from Caucasian and Japanese populations, the extent of intracranial atherosclerosis is much more severe in Hong Kong Chinese, whereas atherosclerotic narrowing of the extracranial carotid artery is less severe in Hong Kong Chinese than in Caucasians. (Stroke 1993;24:779–786)

KEY WORDS • atherosclerosis • carotid arteries • cerebral arteries • Chinese • epidemiology

Racial differences in the distribution of cerebral atherosclerosis has long been a subject of interest. Angiographic and autopsy studies in stroke patients have shown that African-Americans$^{1-5}$ and Japanese$^{6-14}$ tend to have intracranial vascular occlusion, whereas Caucasians$^{15-18}$ tend to have more extracranial lesions. Autopsy studies of the distribution of atheroma in the general population have shown similar racial differences. African-Americans and Japanese have more intracranial atherosclerosis, whereas Caucasians have more extracranial disease.$^{19-27}$ Data in the Chinese population are scarce. A recent angiographic study involving a small series of Chinese stroke patients suggested a pattern similar to that in Japanese patients and different from that in Caucasian patients.$^{28}$ Pathological study is lacking.

Although the mortality rate of patients with ischemic heart disease and acute myocardial infarction in Hong Kong is only one fourth that of Caucasians, the mortality rate of cerebrovascular disease is comparable.$^{29,30}$ The incidence of stroke in Hong Kong is not well recorded, but a recent epidemiological study in China found an overall stroke incidence higher than that in Rochester, Minn., but lower than that in Hisayama, Japan.$^{31}$ A recent clinical study of the pattern of stroke in Hong Kong Chinese showed a different proportion of stroke subtypes compared with that in Caucasian studies, with a high proportion of cerebral hemorrhage and lacunar infarction. Carotid bruits, transient ischemic attacks, and other accompanying features of atherosclerosis such as ischemic heart disease were far less common in Hong Kong Chinese than in Caucasians.$^{32}$

As atherosclerotic narrowing of vessels is a main pathological determinant of ischemic cerebral vascular disease, the distribution of atherosclerosis in cerebral vessels in a general autopsy population serves as useful background information. In this study, we attempted to define the pattern of cerebral atherosclerotic narrowing in a general autopsy population of Hong Kong Chinese and to examine its relation to various known risk factors of systemic atherosclerosis. The relation between the clinical evidence of stroke, the presence of cerebral infarction, and atherosclerosis of the extracranial, intracranial, and intraparenchymatous arteries will be dealt with separately in another report.

Subjects and Methods

One hundred fourteen consecutive autopsies of Chinese patients who died at Queen Mary Hospital, Hong Kong (a regional general hospital) during the period from August to November of 1988 were included in the

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study. The incidence of atherosclerosis-related diseases in this group of patients was obtained by reviewing the case notes and interviewing the deceased patients' relatives and was supplemented by autopsy findings. Ischemic heart disease was defined either by history of angina or previous infarction or by postmortem finding of acute or old myocardial infarct. Hypertension was defined as a history of elevated blood pressure with or without previous antihypertensive therapy or persistent elevated blood pressure (>160 mm Hg systolic or 95 mm Hg diastolic) during hospitalization. The presence of diabetes mellitus and smoking was based on the clinical history. A history of stroke was recorded. The brain was examined for the presence of recent and old cerebral infarction and hemorrhage.

The bodies were routinely opened by a U-shaped cervical incision, thus facilitating removal of the anterior extracranial vessels. The cervical portion of the internal carotid artery was cut as close to the skull as possible and removed with the carotid sinus, the common carotid artery down to its origin in the aortic arch, and the innominate artery. The vessels were fixed in 10% formalin, and cross-sections were cut at 5-mm intervals. Blocks were taken from the narrowest parts affected by atherosclerosis, with the sites recorded in predrawn diagrams. Caution was taken to ensure perpendicular cutting and embedding. Sections 6 μm thick were prepared and stained with hematoxylin and eosin, Mason's trichrome, and elastic–van Gieson stains for microscopic histology and videoplan analysis.

The brains were removed and fixed in 20% formalin for 2 weeks before cutting. The vessels in the circle of Willis were graded by gross examination using the method of Baker et al19 (by which we divided them into 22 zones, taking into account the luminal narrowing and circumferential involvement) as follows: grade 1, opacity involving a small part of vessel circumference, with no luminal narrowing; grade 2, a diffuse, thin plaque not involving the entire vessel circumference, with minimal luminal narrowing, or a small, thick plaque producing <25% luminal narrowing; grade 3, a diffuse, thin plaque with circumferential mild luminal narrowing or a thick plaque producing 25–50% narrowing; and grade 4, a thick plaque with moderate or marked circumferential narrowing or localized narrowing of >50%. The sum of scores from the 22 zones was taken as the Baker score. During grading, the vessels were cross-sectioned at 5-mm intervals; the narrowest parts in the middle cerebral stem and the anterior cerebral, posterior cerebral, basilar, and vertebral arteries were then taken for histological sectioning and analyzed as for the extracranial vessels. The presence of thrombotic or embolic occlusion and ulceration in the intracranial vessels was also recorded separately, and blocks were taken for histological confirmation.

From the 114 autopsied cases, a total of 103 pairs of extracranial carotid arteries and 102 brains were found to be suitable for analysis. After examination of the vessels, the brains were sectioned and blocks taken for histological examination.

For morphometric analysis, the Kontron Videoplan 2 image processing system was used, and the method described by Cheung et al30 was used to measure the degree of luminal narrowing (Figure 1). The internal elastic lamina was taken as equivalent to the original luminal border because the endothelium is thin and closely apposed to it in normal vessels. The internal elastic lamina was traced, and the original lumen was determined by the formula $A = \frac{p^2}{4\pi}$, where $A$ was the original luminal area and $p$ was the perimeter of the area bounded by the internal elastic lamina. The area of atheroma was then traced and determined by the videoplan machine, and the percentage of luminal narrowing was determined by the formula $\frac{A}{A} \times 100\%$, where $A$ was the area occupied by the atheroma. This method eliminated errors due to luminal collapse that might occur during processing.

By the above method, the maximal narrowing of the right and left extracranial carotid arteries was obtained in each case, and the mean percent narrowing of the extracranial vessels (MEC) was calculated. The mean percent narrowing of the intracranial vessels (MIC) was similarly obtained by averaging the maximal narrowing of the nine intracranial vessels measured.

Multiple linear regression analysis was used to assess the effects of age, ischemic heart disease, diabetes mellitus, hypertension, and smoking on mean intracranial and extracranial narrowing as measured by MEC and MEC. The SAS package34 was used for computation.
TABLE 1. Age and Sex Distribution of Subjects

<table>
<thead>
<tr>
<th>Age range (years)</th>
<th>Sex</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>1–10</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>11–20</td>
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<td>5</td>
</tr>
<tr>
<td>81–90</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td>71</td>
<td>43</td>
</tr>
</tbody>
</table>

Combinations of variables were included in stepwise fashion, and the improvement in prediction was assessed by the partial F test.35

Results

Table 1 shows the age and sex distribution of our subjects. Table 2 shows the cause of death of the 114 subjects and presents a comparison with the mortality statistics of the local population.29 In our subjects, there are more deaths that fall under the category of injury and poisoning and that of pneumonia, as expected in an autopsy series that includes coroners' autopsies. The percentage of death due to ischemic heart disease in our study is similar to that in the population, whereas the percentage of death due to cerebrovascular disease is lower. The incidence of intracranial hemorrhage and cerebral infarction cannot be compared because many undefined cases, in which no computed tomography or postmortem examination is performed, are entered into the “other cerebrovascular disease” category in the mortality statistics.

The percent incidence of various atherosclerosis-related diseases is shown in Table 3. Nine patients had a history of stroke, and small, old cystic infarcts were found in the brain. One patient developed superimposed acute cerebral infarct secondary to bacterial endocarditis. There were four patients with acute cerebral infarcts, two of embolic causes due to bacterial endocarditis and disseminated malignancy and two due to severe atheromatous narrowing of intracranial vessels (with superimposed thrombus formation in one). The distribution and relation of cerebral infarcts with extracranial, intracranial, and intraparenchymal vascular occlusion will be dealt with separately in another report.

Atherosclerosis of Intracranial Vessels

We found a severe degree of atherosclerosis in the intracranial vessels of our population. If we take >50% luminal narrowing as determined by morphometric measurement as evidence of severe atherosclerosis (which corresponds to grade 4 of the Baker's scoring system), 31.4% of the subjects had at least one of the nine intracranial main cerebral arteries affected by severe atherosclerosis. Figure 2 presents the relative frequency of this severe degree of intracranial atherosclerosis in relation to age. The percentage of subjects with severe intracranial atherosclerosis in at least one vessel rose from approximately 30% in the sixth and seventh decades to approximately 50% in the eighth and ninth decades. Moreover, in these affected subjects usually more than one vessel was involved. The percent-

FIGURE 2. Graph showing percentage of patients with severe intracranial atherosclerosis (defined as >50% luminal narrowing) in a varying number of vessels.
age of subjects with involvement of at least two, three, and four vessels is also shown in Figure 2.

Table 4 shows the mean Baker score of the 102 subjects in relation to age. The mean score increased with advancing age, reaching a maximum of 46.1 in the ninth decade. The grading in general showed good correlation to the luminal narrowing as determined by morphometric measurement. Because of the intrinsic design of Baker's grading method, when there is circumferential narrowing of the vessels the grade may be higher than is reflected by the morphometric measurement. The distinction between circumferential and localized narrowing is reasonable because a circumferential lesion is more likely to cause a severe degree of impairment of blood flow due to loss of distensibility than is an eccentric lesion of the same size. Baker's scoring system also adds information to the state of the medium-sized vessels present in the more distal part of the circulation, in which morphometric measurement becomes more difficult.

The distribution of severe atherosclerotic narrowing (grade 4 by the Baker method) in the intracranial vessels is shown in Figure 3. Commonly affected sites were the posterior cerebral artery, middle cerebral artery stem, vertebral artery, and basilar artery. Medium-sized branches such as the distal part of the anterior cerebral artery, the middle cerebral artery after bifurcation, the superior cerebellar artery, and the posterior inferior cerebellar artery were also commonly affected. Moreover, many of these grade 4 lesions were located in the very distal branches in the leptomeningeal surfaces.

Although severe atheromatous narrowing of intracranial vessels was common, the plaques were usually covered by an intact fibromuscular cap, and ulceration and fissuring were rarely detected. Complete occlusion of intracranial vessel was noted in 11 vessels belonging to 10 subjects. In seven of these vessels, there was evidence of severe atheroma, with thrombosis or emboli. Ulceration was not detected in any. It must be noted, however, that in four vessels the lesions were old; therefore, whether ulceration or fissuring might have been an initiating event for thrombosis could not be discerned. The other four vessels showed embolic occlusion with no atheroma.

Atherosclerosis of Extracranial Vessels

Eighteen percent of the subjects had at least one of their two extracranial carotid arteries narrowed by >50%; only 2% had complete occlusion. In 70% of these cases, the sites of maximum narrowing occurred in the carotid sinus and the origin of the internal carotid artery. Most of these plaques had a thick, fibrous cap and smooth intimal lining. Ulceration and fissuring of the plaque were demonstrated in only three cases in our study, two of them with superimposed thrombosis resulting in complete luminal occlusion. All three were associated with severe generalized atherosclerosis. In two cases there was a ruptured atherosclerotic saccular aneurysm in the arch of the aorta. The patient in the other case had severe coronary atherosclerosis and died of acute myocardial infarction. None showed a cerebral infarct related to the carotid artery occlusion.

Intracranial Versus Extracranial Vessels

Figure 4 shows the percent incidence of severe (left panel) and extreme (right panel) atherosclerosis in the intracranial and extracranial vessels in different age groups. As age advanced, the incidence of severe extracranial atherosclerosis leveled off, while that of the intracranial vessels continued to increase. For the percent incidence increase of extreme atherosclerotic narrowing (defined as >75% luminal narrowing) in the two groups of vessels, the intracranial vessels had a much higher incidence of extreme narrowing than did the extracranial vessels. In the sixth, seventh, and eighth decades, approximately 20% of subjects studied had at least one vessel that showed extreme involvement of atherosclerosis. Interestingly, the incidence of extreme atherosclerotic narrowing in both the intracranial and extracranial vessels dropped in the ninth decade.

Relation to Risk Factors

Intracranial vessels. Table 5 shows that age, ischemic heart disease, diabetes mellitus, and hypertension had a positive effect on vessel narrowing. Age was the most

<table>
<thead>
<tr>
<th>Age range (years)</th>
<th>Cases (n)</th>
<th>Baker's score (mean±SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-10</td>
<td>1</td>
<td>13.0±0.0</td>
</tr>
<tr>
<td>11-20</td>
<td>4</td>
<td>5.1±2.4</td>
</tr>
<tr>
<td>21-30</td>
<td>3</td>
<td>12.1±3.3</td>
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<td>31-40</td>
<td>6</td>
<td>15.2±3.6</td>
</tr>
<tr>
<td>41-50</td>
<td>9</td>
<td>16.0±3.3</td>
</tr>
<tr>
<td>51-60</td>
<td>15</td>
<td>26.2±4.3</td>
</tr>
<tr>
<td>61-70</td>
<td>29</td>
<td>33.7±3.8</td>
</tr>
<tr>
<td>71-80</td>
<td>20</td>
<td>44.5±4.8</td>
</tr>
<tr>
<td>81-90</td>
<td>15</td>
<td>46.1±5.4</td>
</tr>
</tbody>
</table>
influential variable, followed by ischemic heart disease. Significant improvement in correlation was obtained at each stage when the number of variables was increased to two, then three, and then by all four. Age and ischemic heart disease achieved the best bivariate prediction, followed closely by age and diabetes mellitus or age and hypertension.

**TABLE 5. Multiple Linear Regression Analysis for Mean Intracranial Narrowing**

<table>
<thead>
<tr>
<th>Regression variables</th>
<th>$R^2$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>0.13</td>
<td>0.0001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.15</td>
<td>0.0001</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>0.21</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age</td>
<td>0.27</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, diabetes mellitus</td>
<td>0.36</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, hypertension</td>
<td>0.36</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, ischemic heart disease</td>
<td>0.40</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diabetes mellitus, hypertension, ischemic heart disease</td>
<td>0.44</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diabetes mellitus, hypertension, ischemic heart disease, age</td>
<td>0.56</td>
<td>0.0001</td>
</tr>
<tr>
<td>Smoking</td>
<td>...</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Fitted Model**

\[
\text{Regression equation: mean intracranial narrowing = } -8.6 + 11.8 \times \text{diabetes mellitus} + 11.23 \times \text{hypertension} + 16.96 \times \text{ischemic heart disease} + 0.34 \times \text{age}. \text{NS, not significant.}
\]

The final combination of all four variables was a significant improvement over any three-variable combination, accounting for 55% of the observed variation in mean intracranial narrowing.

Smoking did not contribute significantly either on its own or in multiple regressions for mean intracranial narrowing.

**Extracranial vessels.** For mean extracranial narrowing, age was the best single predictive variable (Table 6). Age and ischemic heart disease were the best pair, with significant improvement in fit over age alone. The fit for the three variables age, ischemic heart disease, and smoking represented a significant improvement in correlation over age and ischemic heart disease.

**TABLE 6. Multiple Linear Regression Analysis for Mean Extracranial Narrowing**

<table>
<thead>
<tr>
<th>Regression variables</th>
<th>$R^2$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>0.1</td>
<td>0.0023</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>0.15</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age</td>
<td>0.23</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, ischemic heart disease</td>
<td>0.32</td>
<td>0.0001</td>
</tr>
<tr>
<td>Age, ischemic heart disease, smoking</td>
<td>0.37</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>...</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>...</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Fitted Model**

\[
\text{Regression equation: mean extracranial narrowing = } -2.312 + 8.58 \times \text{smoking} + 10.56 \times \text{ischemic heart disease} + 0.43 \times \text{age}. \text{NS, not significant.}
\]
Diabetes mellitus and hypertension were not found to be associated significantly with mean extracranial narrowing individually nor did they improve the fits of other combinations of age, ischemic heart disease, and smoking when included.

Discussion

The Baker scoring system provides a quick and comprehensive grading of a large number of vessels in the cerebral circulation although it has some drawbacks. It is especially valuable in reflecting the state of the medium-sized vessels in the more distal parts of the circulation. The main problem with such a gross grading system is interobserver variability. We overcame this problem by supplementing the system with objective morphometric measurement. Our method of assessing atherosclerosis by measuring the luminal narrowing is particularly satisfactory for cerebral vessels because physiological remodeling processes such as diffuse intimal thickening in coronary vessels are not seen in the cerebral arteries, and any lesion that causes thickening of intima in cerebral vessels can be attributed to the atheromatous process. We also avoided observation errors due to luminal collapse. Errors can occur as a result of tangential cutting or excessive trimming of the block, which results in underestimation of the narrowing. However, these errors can be minimized by careful handling.

The main problem of data from an autopsy population is the possibility of bias toward certain diseases. A comparison with population mortality statistics shows that more subjects in our study died of injury and poisoning and pneumonia, whereas cardiovascular and cerebrovascular diseases were not overrepresented. As there is no direct correlation between cerebral atherosclerosis and accidents and pneumonia, we conclude that our studied subjects are representative of the general population in the severity of cerebral atherosclerosis.

We confirm the angiographic findings that the pattern of cerebral atherosclerosis in a Chinese population is similar to that in Japanese and African-American populations and different from that in a Caucasian population. Martin et al.20 examined extracranial vessels from 100 random autopsy cases and found that 11% had complete occlusion of at least one extracranial vessel, the majority of which were carotid arteries; 40% of the cases had >50% luminal narrowing. Fisher et al.21 studied 178 routine autopsies and found in 8.4% occlusion of internal carotid artery origin. Only 2% of our cases showed complete occlusion of extracranial carotid arteries, and 18% showed >50% narrowing. This is a much lower figure than in the above series. Moreover, ulcerated plaques were seen in only 3% of our cases, and all were related to severe systemic atherosclerosis. The finding is consistent with the clinical observation that carotid bruits and transient ischemic attacks, which are related to extracranial cerebral atherosclerosis, are rare in our stroke patients.32 It seems that the incidence of severe systemic atherosclerosis is less common in a Chinese population, and it is in those cases that the extracranial cerebral vessels are involved.

For the intracranial vessels, the Baker scores of our subjects are much higher than those of Caucasians, indicating a more severe degree of intracranial atherosclerosis. We have a higher Baker score than that reported in the Japanese23 as well, but the temporal difference of three decades between the two series may account for some of the differences.

Figure 5 shows the percentages of cases with severe intracranial atherosclerosis (as defined by at least one vessel graded 4+ by Baker’s system) in different populations. Compared with the Caucasian series of Fisher et al.22 and the Japanese series of Nakamura et al.23 our incidence of severe intracranial atherosclerosis is again much higher. Some may argue that observer error may account for our high Baker score. This is unlikely because the results of morphometric measurements in our population, which give a lower percentage of severe intracranial atherosclerosis due to the intrinsic design of Baker’s system as mentioned, are still much higher than those in the Caucasian and Japanese series.

We observed a high Baker score in the youngest subject in our series. He was an 8-year-old boy with aplastic anemia, treated with steroids, who died of meningitis and ventriculitis. There were several atheromas in the intracranial vessels at his early age; one was well formed and produced 12% narrowing of the left vertebral artery. Whether this early onset of intracranial atherosclerosis was related to the underlying disease process and treatment or reflected a high vulnerability of our population to develop intracranial atherosclerosis is uncertain.

Based on data from whites, Fisher divided stroke patients into three groups: those with atherosclerosis involving mainly the larger extracranial arteries and the intracranial basilar and vertebral arteries; those with lacunar infarcts due to lipohyalinosis; and those with medium-sized vessel obstruction, usually due to embolism. Caplan et al.17 reviewed the data in African-Americans and Orientals and added in a fourth group, which is common in these two races, with occlusion of the medium-sized intracranial arteries and their major branches. Morphological details of these occlusions are not known. We confirm their observation in our population that atherosclerotic narrowing of the medium-

![Figure 5. Graph showing percent incidence of severe intracranial atherosclerosis (defined as at least one vessel graded 4+ by Baker's system) in different populations.](http://stroke.ahajournals.org/DownloadedromStroke.ahajournals.org)
sized intracranial arteries is common in Chinese. Grade 4 lesions are common in the posterior inferior cerebellar artery, superior cerebellar artery, middle cerebral artery and its branches, and the anterior and posterior cerebral arteries. According to our observation, distal branches from these vessels in the leptomeningeal surfaces are also commonly affected by atheromatous plaques. We believe that these are the lesions that give rise to ischemic stroke. In a way, obstruction of these distal branches contributes to the development of small cortical infarcts, which are not uncommon in our locality. Moreover, the circumferential thick and rigid atheromas in the middle cerebral artery and the basilar artery not only cause decreased flow through the lumen, but also severely obstruct the origins of small perforating branches, which contributes significantly to the development of lacunar infarction. This is supported by the high incidence of lacunar infarction in our stroke patients. As noted in some of the cases, the narrowed intracranial arteries are also favored sites of embolic obstruction, and depending on the state of collateral circulation, result in the formation of both cortical and lacunar infarcts. However, autopsy studies in a larger number of Chinese stroke patients are needed to further clarify the pathology of the intracranial vascular narrowing and its relation to cerebral infarction.

Relation to Risk Factors

Coronary heart disease, stroke, hypertension, and diabetes mellitus were factors shown to be associated with more extensive cerebral atherosclerosis in numerous studies in the past two decades. Smoking is also a risk factor for stroke in Caucasians. We found smoking only to be related to extracranial atherosclerosis, which is not an important cause of stroke in the Hong Kong Chinese population. This may explain why it cannot be demonstrated that smoking is a risk factor for stroke in a local study. An angiographic study of stroke patients in a mixed Caucasian and African-American population showed that ischemic heart disease is more common in patients with disease involving the origin of the internal carotid artery, whereas diabetes mellitus is more often noted in patients with intracranial internal carotid artery or middle cerebral artery disease. Our finding that diabetes mellitus is associated only with more severe intracranial atherosclerosis is in accordance with the above observation.

McGarry et al studied the pattern of cerebral atherosclerosis in New Orleans, La., and found a higher incidence of intracranial atherosclerosis in African-Americans compared with Caucasians. He drew attention to the higher incidence of hypertension and cerebrovascular diseases in African-Americans in relation to his observation. Moreover, he found that hypertension was associated with more severe intracranial atherosclerosis while such a relation was not found in the extracranial cerebral vessels. Several studies also stressed the importance of blood pressure in intracranial atherosclerosis. We also confirmed the importance of hypertension in intracranial atherosclerosis.

Analysis of risk factors from the Oslo, Norway, study has shown blood pressure, and to a lesser extent serum lipid level, to be important risk factors in intracranial atherosclerosis. Other authors suggested that extracranial carotid atherosclerosis and coronary atherosclerosis might be more closely related to serum lipid level than to blood pressure, whereas atherosclerosis in the large intracranial arteries was related more closely to blood pressure than to serum lipid level. Unfortunately, the serum lipid level was not measured in our patients. Ischemic heart disease, which is closely related to coronary atherosclerosis and serum lipid levels, was the factor analyzed in our study. We found ischemic heart disease to be related to both intracranial and extracranial atherosclerosis.

It seems that in our locality, extracranial atherosclerosis follows the risk factors associated with coronary atherosclerosis, and the low incidence of both may partly be explained by the generally lower serum lipid level in the Chinese population. The high incidence of intracranial atherosclerosis may reflect the contribution of other factors such as diabetes mellitus and hypertension. The prevalence of hypertension is 20% in the local population over 60 years of age. The prevalence of diabetes mellitus is 9.8% for those over 60 and 17.1% for those over 75 years of age. This is not high compared with Caucasian figures. However, in this local survey, 76% of the diabetics were previously undiagnosed; two thirds of the diagnosed diabetics defaulted on follow-up, and in the remaining one third, blood sugar levels were poorly controlled. The impression gained from clinical practice indicates that this poor control may also apply to patients with hypertension, although firm data are not available. Thus, the cause of the high level of intracranial atherosclerosis cannot be explained by a higher prevalence of the two diseases in our population. Rather, we suggest that poor control of the disease may be a more important factor. This also partly explains the high incidence of stroke in our locality in a population with a relatively low level of systemic atherosclerosis. With increasing health awareness and the improvement in the health care system in recent years, we may expect better control of the above two diseases and a drop in stroke incidence. However, with improved economic status and change in dietary habits, the local population may in turn be threatened by hyperlipidemia and its complications.

In a comparative study of the locations of vascular lesions in Caucasian and African-American stroke patients, race was found to be the only significant factor. The factors contributing to racial differences are many; among them are socioeconomic and dietary factors, exercise, and smoking. Recently, the levels of apolipoproteins and lipoprotein(a) are being recognized as important risk factors for stroke. As there is genetic variation in the apolipoprotein genes and certain phenotypes are associated with atherosclerotic diseases, racial differences in the apolipoprotein genes may be a contributing factor in the observed racial differences in cerebral atherosclerosis. Data regarding this aspect are lacking and await further study.

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Stroke. 1993;24:779-786
doi: 10.1161/01.STR.24.6.779

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