Pressures were measured in the carotid arteries of 61 patients proximal and distal to atherosclerotic plaques which were carefully studied by angiography and anatomical dissection.

1. An atherosclerotic plaque causing a constriction of less than 47% luminal diameter leaving a lumen greater than 3.0 mm in diameter never caused pressure drops of greater than 10 mm Hg. Stenoses of greater than 63% luminal diameter leaving lumens less than 1.0 mm in diameter always caused pressure drops.

2. Atherosclerotic plaques producing defects which narrowed the lumen fell into a distinct pattern:
   - Type 1 lesions—This basic lesion filled the bulb of the internal carotid artery near its origin, causing a 1 to 2 cm smooth elliptical encroachment on the lumen.
   - Type 2 lesions—Short localized areas of thickening in addition to the basic lesion caused bar-like defects of the lumen at the origin of the internal carotid artery or near the distal end of the lesion.
   - Type 3 lesions—Multiple bar-like defects were sometimes seen.
   - Type 4 lesions—The areas of increased thickening of the lesion were sometimes quite narrow, producing diaphragm-like defects on the lumen.

Although theoretically these various types of stenoses should produce different hemodynamic changes, insufficient numbers of observations were made to corroborate these presumptions.

3. Angiograms in general mimicked the gross appearance of the plaques and predicted the actual degree of stenosis produced but did not identify many diaphragm defects, ulcerations, or small thrombi.

ADDITIONAL KEY WORDS
- angiography
- embolism
- atherosclerotic plaques
- pressure changes
- endarterectomy
- thrombosis
- blood flow

It is well known that an arterial stenosis can decrease distal blood flow and arterial pressure. It is less well known that a very marked constriction may be required before any hemodynamic changes occur. When a “critical stenosis” is present, small increases in constriction cause significant reductions in flow and pressure. The atherosclerotic lesion at the bifurcation of the common carotid artery commonly associated with symptoms of cerebrovascular insufficiency is a good example of a localized arterial stenosis. The immediately adjacent vessels are usually of normal caliber. Previous studies on small groups of patients indicated that atherosclerotic stenoses causing occlusions of less than 47% luminal diameter (72% luminal area) did not decrease distal carotid artery pressure. Stenoses producing constrictions of greater than 60% luminal diameter (84% luminal area) almost always caused decreases in pressures and flows.
DEWEESE, MAY, LIPCHIK, ROB

A decrease in arterial pressure distal to a stenosis reflects a comparable percentage decrease in blood flow through the narrowed vessel. The measurement of pressure drop across a 0% stenosis, and occlusion of the vessel was a 100% stenosis.

\[
\text{\% stenosis} = \frac{(\text{internal diameter of vessel} - \text{diameter of lumen in plaque}) \times 100}{\text{internal diameter of vessel}}
\]

Constriction is more practical than measuring blood flow and provides direct evidence of hemodynamic changes.

It was felt desirable to extend these observations to a larger group of patients in hopes of determining more accurately what degree of carotid artery stenosis was hemodynamically significant. Careful dissections of thromboendarterectomy specimens were planned to determine how accurately the arteriogram mimicked the anatomical specimens. The patterns of narrowing produced by the atherosclerotic plaque and their possible hemodynamic significance were additional objects of the study.

**Method**

The 61 patients in this series underwent carotid endarterectomy for symptomatic atherosclerotic occlusive disease at the University of Rochester Medical Center. Angiograms, carotid arterial pressures, and the anatomy of the resected atherosclerotic plaque from the carotid artery bifurcation were studied.

**ANGIOGRAPHS**

Angiograms of carotid artery bifurcations in two planes were obtained by direct carotid arteriography in five instances and arch aortography in the remaining 56 patients.

Four-vessel angiography was obtained by injecting 40 to 50 cc of sodium iothalamate (Angio-CONRAY) into the ascending aorta through a catheter inserted percutaneously via the femoral or axillary artery. Simultaneous biplane views of the arteries were obtained in each study for visualization of the two diameters of the vessels.

The degree of stenosis in percent was determined by measuring the diameter of the radiopaque column at its narrowest point in the internal carotid artery and relating it to the normal luminal diameter of the vessel at this point. The wall of the artery was not visible and therefore its limits were estimated by drawing lines between the radiopaque border of the distal common carotid artery and the radiopaque borders of the first normal-appearing portion of the internal carotid artery distal to the plaque. If there was no narrowing of the vessel, it was called recorded. All measurements were made directly on the radiographs. These measurements were corrected for magnification to give the absolute diameter in millimeters. In our laboratory, the vessels are enlarged between 20 and 25% on the radiographs.

Careful observations of the site of narrowing and shapes of the radiopaque column in the region of the carotid artery bifurcation were recorded. It was possible to make accurate measurements in 53 of 61 angiograms. Inadequate visualization in eight angiograms was due to poor technique or overlapping structures.

**ARTERIAL PRESSURES**

Arterial pressures were obtained at the time of the operation after mobilization of the carotid artery. Pressures were measured with a mercury manometer connected by a short length of plastic tubing to a No. 22 needle inserted into the artery. Mean pressures were determined by adding to the diastolic pressure one-third of the difference between the diastolic and systolic pressures. Proximal pressures were measured in the common carotid artery. The needle was then inserted into the internal carotid artery beyond the plaque and the distal pressure measured. Then the carotid artery was occluded and a second distal pressure recorded as the backflow pressure.

**ANATOMY OF THE ATHEROSCLEROTIC PLAQUE**

Most of the endarterectomies were performed by making a longitudinal incision through the adventitia of the artery and establishing the correct plane of dissection between the plaque and outer media before occluding the carotid artery. It was then usually possible to remove the entire specimen intact. The diameter of the central lumen was measured with steel rods at varying diameters in increments of 0.5 mm. The diameter of the vessel was determined by measuring the diameter of the plaque at the point of maximal narrowing. The degree of stenosis was determined as before by the formula given previously.

The plaques were carefully examined and in different specimens tangential, longitudinal or transverse cuts were made and the anatomy drawn. It was not possible to remove all of the specimens intact. Careful measurements of the lumen were possible in 41 of 61 patients.
CAROTID ARTERY STENOSIS

Results
ANATOMY OF THE ATHEROSCLEROTIC PLAQUE
The extent of the atherosclerotic plaquing was remarkably similar in all specimens (figs. 1-3). The medial and intimal thickening and degeneration began about 1 cm proximal to the carotid bifurcation, extended only a few millimeters into the external carotid artery, and extended about 1.5 cm into the internal carotid artery to a point just beyond the bulbous portion of the proximal internal carotid artery. The plaque became very thin near its proximal and distal ends where it appeared to melt into more normal vessel walls (figs. 1A and 2A). It was quite easy to separate the atherosclerotic core from the media over the central portions of the lesion, but it became more difficult to separate the media from the adventitia and intima near the thinner ends of the lesion.

The atherosclerotic plaque was characteristically thickest at the origins of the internal and external carotid arteries and in the bulbous portions of the proximal internal carotid artery. This produced a smooth elliptical encroachment on the lumen of the internal carotid artery as was best demonstrated by cutting the carotid bifurcation gross specimen tangentially or on the oblique view of the angiogram (type 1) (fig. 1B).

Frequently, the plaque was quite thick at
A. Atherosclerotic plaque from carotid bifurcation which has been cut tangentially. There is marked thickening at the origin of the internal carotid artery (type 2). A metal rod with a diameter of 1 mm is through the lumen at a point where the diameter of the plaque is 6 mm and, therefore, there is an 83% constriction of the lumen. The pressure drop was 65 mm Hg. B. Oblique view of the angiogram of the same patient demonstrates a bar-like encroachment on the lumen of the vessel (type 2). The lumen measured 1 mm on the angiogram and the stenosis was calculated to be 86%. Note narrowing also of the external carotid.

The difference between the mean arterial pressure in the common carotid artery and internal carotid artery was related to percent constriction of the luminal diameter measured in 41 specimens (table 1). Stenoses of 0 to 47% never produced significant pressure drops greater than 10 mm Hg, whereas all 20 plaques removed intact. Twenty percent (eight of 40) who were examined demonstrated either ulceration of the plaque with small particles of loose calcium in the lumen or thrombi of varying ages loosely or tightly adherent to the endothelial surface (fig. 3). Neither ulcerations nor thrombi were recognized accurately on angiograms, even in retrospect.

**Pressure Changes Distal to Atherosclerotic Plaques**

It was possible to examine carefully the endothelial surface of the plaques which were
CAROTID ARTERY STENOSES

Atherosclerotic plaque from internal carotid artery demonstrating bar-like narrowing at the distal end of the plaque (type 2). Metal pin is through the lumen. The endothelial surface of the plaque is lined with fresh and old thrombus, some of which is easily dislodged by the metal pin. The lesion produced an 83% stenosis and a pressure drop of 50 mm Hg.

Producing stenoses greater than 63% caused pressure drops of greater than 10 mm Hg (figs. 2A and 3). Stenoses producing 48 to 62% constriction of the luminal diameter might or might not produce significant pressure drops. A plaque producing a 58% stenosis which did not result in any pressure drop is illustrated in figure 1A.

The differences in pressures were related to percent constriction as measured on 53 angiograms (table 2). It was again observed that stenosis of 0 to 47% never produced pressure drops greater than 10 mm Hg. In all instances where there was greater than a 63% stenosis there was some pressure drop (figs. 2B, 4 and 5). Stenoses of 48 to 62% might or might not cause pressure drops (fig. 1B).

In general, there was close agreement between the measured percent stenosis in gross specimens and on angiograms. There was less than a 10% difference in these measurements in 28 of 34 patients where the comparison could be made. The angiographical measurements were 11 to 25% greater than the

<table>
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<tr>
<th>Gross specimen stenosis, %</th>
<th>0 mm Hg</th>
<th>1-10 mm Hg</th>
<th>&gt;10 mm Hg</th>
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<tr>
<td>0-47</td>
<td>4</td>
<td>1</td>
<td>0</td>
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<tr>
<td>48-62</td>
<td>4</td>
<td>9</td>
<td>3</td>
</tr>
<tr>
<td>63-90</td>
<td>0</td>
<td>0</td>
<td>20</td>
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Angiogram of carotid artery bifurcation. The atherosclerotic plaque is of varying thicknesses with bar-like defects on the lumen near both the proximal and distal ends of the plaque (type 3). There was an 86% stenosis causing a pressure drop of 100 mm Hg. The lumen was 1 mm in diameter at its narrowest.

specimen measurements in four patients and 13 to 20% less than the specimen in two patients (figs. 1 and 2).

In most instances it was quite easy to obtain an accurate measurement of the remaining lumen in the carotid artery on angiograms. It was more difficult to estimate the internal diameter of the vessel without a plaque. Therefore, the pressure drop across the stenosis was related to the absolute luminal diameter as measured on angiograms in 43 patients (table 3). If the lumen was 0 to 1.5 mm in diameter there was always a greater than 10 mm Hg pressure drop (figs. 2, 4 and 5). If the lumen was greater than 1.5 to 3.0 mm there might or might not be a pressure drop (fig. 1). If the lumen was greater than 3.0 mm, there was never a pressure drop greater than 10 mm Hg.

### Discussion

The results of the pressure drops occurring across varying degrees of stenosis were remarkably similar to previous studies of smaller groups of patients. A stenosis causing constriction of less than 47% luminal diameter (72% luminal area) never caused a significant decrease in distal pressure of greater than 10 mm Hg. All stenoses causing a greater occlusion than 63% of luminal diameter (87% luminal area) produced some pressure drop. In the range of stenoses between 47% and 63%, the results of the pressure drops were not consistent. It was not possible, therefore, to assign a specific degree of stenosis as being the dividing line between an insignificant and significant stenosis as was possible in experimental models. From a practical viewpoint, however, one can state that if an angiogram demonstrates less than a 47% narrowing of the luminal diameter of the carotid artery or the patent lumen is greater than 3.0 mm in diameter, the stenosis is not hemodynamically significant. A 63% constriction of luminal diameter or a patent lumen less than 1.0 mm in diameter is always hemodynamically significant.

The arteriogram, in general, accurately described the gross appearance of the atherosclerotic lesions at the carotid bifurcation. In four instances, however, the arteriogram indicated the stenosis to be 11 to 25% more severe

### TABLE 2

<table>
<thead>
<tr>
<th>Arteriogram stenosis, %</th>
<th>0 mm Hg</th>
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<th>&gt;10 mm Hg</th>
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<tr>
<td>0-47</td>
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<td>4</td>
<td>0</td>
</tr>
<tr>
<td>48-62</td>
<td>6</td>
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<td>3</td>
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### TABLE 3

<table>
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<th>Luminal diameter, mm</th>
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<tr>
<td>0-1.5</td>
<td>0</td>
<td>0</td>
<td>19</td>
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<tr>
<td>1.5-3.0</td>
<td>3</td>
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<td>3</td>
</tr>
<tr>
<td>&gt;3.0</td>
<td>5</td>
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CAROTID ARTERY STENOSIS

than it actually was. In only two cases did the arteriogram indicate that the stenosis was 13 to 20% less than when the specimen itself was measured. Unfortunately, it was not possible to identify small thrombi or ulcerations on angiograms in the 20% of specimens demonstrating such potential sources of emboli.

Blood flow through and pressure drop beyond an arterial stenosis result from the interaction of numerous local and systemic variables. The systemic variables include cardiac output, peripheral vascular resistance, velocity of blood flow, collateral circulation, luminal size of the artery, viscosity of blood, and heart rate. The local variables having hemodynamic influence include the cross-sectional luminal area remaining patent within the stenosis and, possibly, the shape of the stenosis. Many of these variables are interdependent, a change in one resulting in simultaneous changes in others.

Several investigators have shown that a certain degree of arterial constriction, termed “critical stenosis,” must be exceeded before any appreciable hemodynamic alteration occurs. At first there was a lack of consistency among investigators concerning the magnitude of this “critical stenosis.” But this apparent lack of agreement disappeared when the anatomy of the arterial stenosis was reduced to its component parts and the hemodynamic effects of these parts were individually analyzed. The hemodynamically dominant component of an arterial stenosis is its outflow tract, i.e., the expansion to normal distal lumen. Of considerably less significance is the length of the constriction, and of negligible importance is the proximal luminal contraction. The hemodynamic effect of an arterial stenosis may be expressed mathematically as:

The above equations were derived for conditions of laminar and steady flow. Nevertheless, when they have been applied to experimental observation on flow through stenoses in blood vessels the fit has been very close. In addition the formula has been used to predict the degree of stenosis that might be necessary to decrease flow in arteries of different caliber and with different velocity of flow; these predictions have been accurate.

Assuming that the hemodynamic contribution of stenosis shape occurs predominantly at the distal luminal expansion, then Equation 2 may be modified to read:

Equation 2. \[ \Delta P = \frac{8\mu}{R^2} \times L \times V_1 \left( \frac{A_1}{A_2} \right)^2 + \rho \times V_1^2 \times \left( \frac{A_1}{A_2} \right)^2 \]

where \( f(\psi) \) = the function representing the hemodynamic effect of stenosis shape.

The magnitude of the contribution of \( f(\psi) \) to \( \Delta P \) has already been the object of experimental investigation. In the considera-
TABLE 4

<table>
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<tr>
<th>Pressure drop</th>
<th>Shape of stenosis*</th>
<th>Percent stenosis</th>
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<td>33</td>
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<tr>
<td>0</td>
<td>2</td>
<td>50</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>48</td>
</tr>
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</table>

*Type 1: gradual, elliptical encroachment on lumen; type 2: bar defect; type 3: multiple bar defects; type 4: diaphragm.

... tion of stenosis shape on hemodynamic effects, the length of stenosis, the presence of multiple stenoses in series, and the configuration of the outflow tract may be profitably examined. There is agreement that length has a measurable, although small, hemodynamic effect.7 8 The influence of length becomes much larger when the degree of luminal constriction is severe.2 VonRuden et al.8 found that the addition of identical but distinct arterial stenoses in series causes hemodynamic changes essentially the same as those of increased length. A series of stenoses causing different degrees of luminal narrowing was found to have the same effect as the most constricting stenosis alone. The effect of differences in configuration of the distal luminal expansion of the stenosis was studied by Holen and Schwartz.9 They found that gradual distal expansion diminished the hemodynamic influence of a stenosis in an in vitro system whereas it had no perceptible influence in an in vivo canine aorta preparation. Thus, given stenoses of the same degree of luminal constriction the shapes that might be expected to differ in hemodynamic influence may be categorized as long and short. In terms of the carotid atheromas encountered in this series of patients, the lesions may be grouped as follows in order of hemodynamic importance:

Multiple bars > single bar >> diaphragms or smooth stenosis, or

Type 3 > Type 2 >> Type 4 or Type 1

In attempting to correlate the hemodynamic contribution of shape of the atheromas, it is necessary to realize that lesions producing modest degrees of luminal constriction will be predominantly of type 1. Indeed, of the atheromas producing less than 48% luminal constriction, 50% were type 1 shape, whereas those producing more than 62% constriction, only 14% were of type 1 shape. Therefore, if hemodynamic differences due to shape are to become apparent, one must analyze the atheromas which constrict the lumen from 48% to 63%, i.e., which are not so disparate in degree of luminal constriction and which are at the threshold of critical stenosis. Table 4 ranks 20 carotid atheromas in order of degree of luminal constriction and correlates the shape of the atheromas and the pressure drop. There is no statistically significant distribution of the shape of atheromas with regard to pressure drop. There is, however, a positive correlation between the magnitude of pressure drop and the degree of luminal constriction. The shape of carotid atheromas did not seem to influence the hemodynamic effects of the resulting arterial stenosis in this study, but the possibility should be considered in evaluation of larger series of lesions.

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