Quantitative Analysis of Intracranial Circulation Using Rapid-Sequence DSA

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Quantitative Analysis of Intracranial Circulation Using Rapid-Sequence DSA

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Using a high-frame-rate technique, intravenous digital subtraction angiography (IVDSA) of the intracranial circulation was performed in patients with transient ischemic attacks and asymptomatic bruits. Twenty patients with normal carotid arteries or mild stenosis of no hemodynamic significance were selected as a control group to evaluate the effect of carotid stenosis on the difference between hemispheres in the peak arrival time (ΔTMAX) of the contrast bolus. Data were obtained for the anterior (ACA), middle (MCA), and posterior (PCA) cerebral artery distributions. Raw data of the time-density curve in a region of interest were analyzed by polynomial curve-fitting techniques to obtain the peak arrival time (TMAX). The ΔTMAXs for normal middle, anterior, and posterior distributions were 0.140 ± 0.119 sec, 0.152 ± 0.146 sec, and 0.189 ± 0.187 sec, respectively. Eleven patients with tight carotid stenosis or occlusion whose ΔTMAX fell outside the normal range as established from the 20 control patients were analyzed with regard to ischemic symptoms. The ΔTMAXs of the MCA, ACA distributions in the asymptomatic patients with tight carotid stenosis or occlusion were 0.653 ± 0.379 sec and 0.118 ± 0.159 sec, respectively; $p < 0.001$ in the MCA when compared with controls. The ΔTMAXs of the MCA, ACA distribution in the symptomatic patients with tight carotid stenosis or occlusion were 1.31 ± 0.13 sec and 0.525 ± 0.079 sec, respectively; $p < 0.001$ in the MCA and ACA distributions when compared with controls. Quantitative analysis of this type of physiologic data may enable the detection of patients with carotid stenosis who are well compensated by collateral flow from those who are poorly compensated and at risk for possible infarction on a hemodynamic basis. Serial follow-up DSA studies in patients with asymptomatic bruits may help to correlate the progression of extracranial carotid stenosis and the status of intracranial collateral reserves.

There are no well accepted criteria in the literature as to what constitutes a hemodynamically significant stenosis [1–3]. A lesion is certainly hemodynamically significant if there is a decrease in cerebral tissue perfusion distal to the stenosis. Because of the many potential collateral pathways via the circle of Willis, ophthalmic artery, and leptomeninges, the hemodynamic effect of a carotid stenotic lesion may not be accurately reflected by the morphologic information provided by selective injections on conventional angiography. Currently, a battery of noninvasive tests [4], conventional angiography, and intravenous digital subtraction angiography (IVDSA) are used in the evaluation of carotid stenosis; each method has its merits and limitations. Asymptomatic patients with major extracranial occlusive disease are occasionally encountered in clinical practice; the lack of ischemic symptoms presumably reflects an adequate collateral reserve. The surgical management of patients with carotid stenosis would be facilitated if patients with carotid stenosis could be separated into those who are well compensated by collateral flow and those who are not. The purpose of this study was to establish a normal baseline for the absolute difference between hemispheres of the peak arrival times (ΔTMAX) of the intravenous contrast bolus and to evaluate this measurement in the assessment of cerebral collateral reserves.

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TABLE 1: **Gender, Age, Reasons for Referral, and DSA Findings in Control Patients**

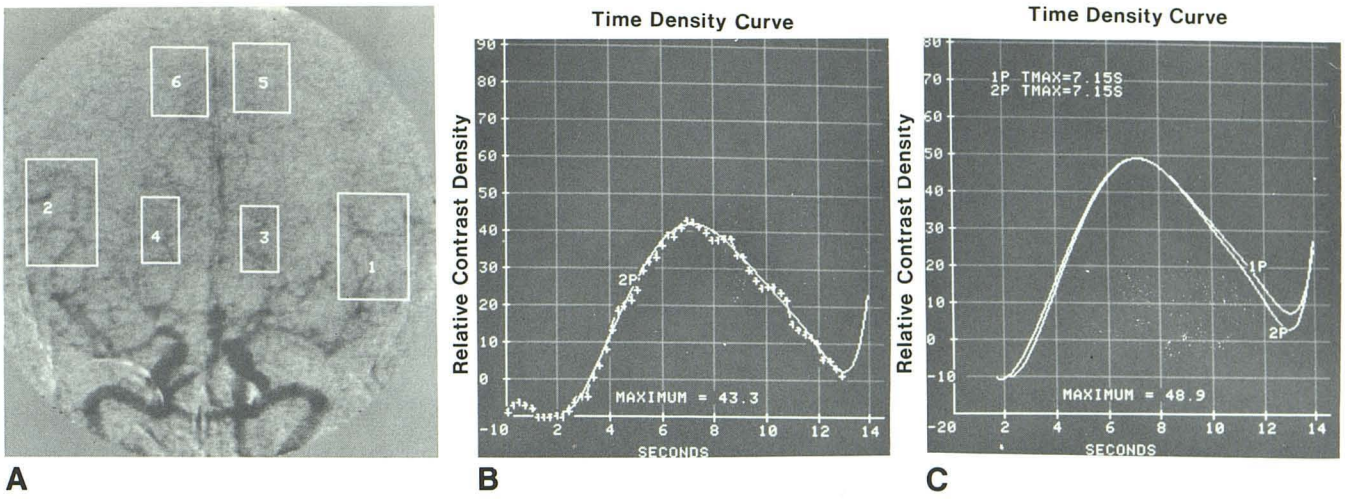
Case No. (age, gender)	Reason for Referral	History of TIAs	DSA Findings
1 (60,F)	L amaurosis fugax; L jaw numbness	R hemisphere	Normal
2 (56,M)	Difficulty writing during TIAs	L hemisphere	30% stenosis, origin R ICA
3 (71,F)	Vertigo; dizziness	R hemisphere	Minimal plaque, R carotid bifurcation
4 (57,M)	Migraine; L arm and leg weakness; R hand weakness	Both hemispheres	Normal
5 (70,F)	Vertigo, ataxia, tinnitus	Vertebrobasilar	Normal
6 (87,M)	3 yr history intermittent ataxia and unsteady gait	L hemisphere	Minimal plaque, both carotid bifurcations
7 (65,F)	Transient numbness, R upper extremity	L hemisphere	Normal
8 (28,M)	R arm and facial numbness; transient dysarthria	L hemisphere	Normal
9 (56,M)	Aortic stenosis; dizziness and blurred vision	Vertebrobasilar	Normal
10 (60,F)	Migraine headache; equivocal findings on noninvasive carotid evaluation	No	Normal
11 (56,M)	Asymptomatic bilateral carotid bruits	No	20% stenosis, origin R ICA
12 (62,M)	Asymptomatic L neck bruits	No	Normal
13 (43,F)	Blurring of L visual field; equivocal findings on noninvasive carotid evaluation	Equivocal history	Normal
14 (88,M)	S/P 6000 rad (60 Gy) irradiation of neck for squamous cell CA of parotid; equivocal findings on noninvasive carotid evaluation	No	Mild circumferential plaque at both carotid bifurcations
15 (63,F)	Vertigo; ataxia	Vertebrobasilar	Normal
16 (55,M)	Asymptomatic R neck bruits	No	Normal
17 (59,F)	Asymptomatic R neck bruits	No	Normal
18 (60,F)	Asymptomatic L neck bruits	No	Normal
19 (60,M)	Transient weakness, R arm	Yes	Normal
20 (48,M)	S/P ligation of R ECA for prior trauma; asymptomatic R neck bruit	No	Ligated R ECA; normal R ICA

Note.—DSA = digital subtraction angiography; TIA = transient ischemic attack; L = left; R = right; ICA = internal carotid artery; ECA = external carotid artery; yr = year; S/P = status post; CA = carcinoma.

TABLE 2: **Gender, Age, Clinical History, and DSA Findings in Patients with High-Grade Stenosis or Occlusion of Internal Carotid Artery**

Case No. (age, gender)	Reasons for DSA Study	DSA Findings
1 (59,M)	Bilateral carotid bruits; left upper and lower extremity weakness	90% stenosis in origins of right CCA and right ICA
2 (62,F)	Asymptomatic bruits	Total occlusion of left ICA at origin
3 (74,M)	Asymptomatic bruits; preoperative for aorto-femoral bypass surgery	Occlusion of right ICA
4 (62,M)	Recurrent carotid stenosis; transient aphasia	>90% stenosis of left ICA origin
5 (62,F)	Recent Broca and Wernicke aphasia	99% stenosis of left ICA; 20% stenosis of right ICA
6 (58,M)	Asymptomatic bruits; preoperative for coronary artery bypass surgery	Complete occlusion of left ICA at origin
7 (67,M)	Asymptomatic bruits; history of retinal emboli	>90% stenosis at origin of right ICA
8 (68,M)	Asymptomatic bruits; recurrent carotid stenosis; S/P endarterectomy	90% stenosis in right CCA; <50% stenosis in left ICA origin
9 (60,M)	1-month history of right amaurosis fugax	Occlusion of right ICA
10 (58,F)	Asymptomatic bruits	95% stenosis of right ICA
11 (65,M)	Asymptomatic bruits	95% stenosis of right ICA

Note.—DSA = digital subtraction angiography; CCA = common carotid artery; ICA = internal carotid artery; S/P = status post.



A B C

Fig. 1.—**A**, ROIs for MCA (1 and 2), PCA (3 and 4), and ACA (5 and 6) distributions are outlined. This AP image has been integrated from four frames for better demonstration of intracranial vascular anatomy. **B**, Raw data (*pluses*) and polynomial fit (*solid line*) of transit of contrast bolus in right MCA distribution in case 15 (table 1) with normal carotid artery bifurcation and normal cardiac output. Final upswing of curve beyond raw data points is function of curve-fitting technique; it does not represent recirculation. **C**, Polynomial fit comparing right (2P) and left (1P) MCA territory in same patient. Discrepancy in maximum on *Y* axis between **B** and **C** is from computer normalization of region 1P and 2P to same maximum value for easier visual comparison.

Subjects and Methods

Twenty patients with normal carotid arteries or mild stenosis of no hemodynamic significance as determined by right (RAO) and left (LAO) anterior oblique views of the extracranial and siphon portions of the carotid arteries were selected as a control group. These patients were referred for evaluation of transient ischemic attacks (TIAs) (11 patients), asymptomatic bruits, or equivocal findings on noninvasive carotid evaluation (nine patients). The gender, age, pertinent clinical history, and DSA findings are listed in table 1. Similar information on 11 patients with high-grade extracranial carotid stenosis or occlusion is listed in table 2; of these, seven were asymptomatic and four symptomatic. There was no history of stroke or significant stenosis in the contralateral carotid artery in this group of patients.

DSA studies were performed with a General Electric DF 3000 digital Fluorocon system mounted on LU/A angiographic arm. All DSA studies were performed with superior vena cava injection through a catheter introduced via the antecubital vein. Renografin 76 was injected at 20–25 ml/sec for a total of 40 ml. Technical factors used in the low-dose, high-frame-rate intracranial runs were 75–90 kVp, 300 mA, and exposure times measuring 12–24 msec; the exposure at the image intensifier was set at 200 μR (0.052 μC/kg)/frame [5]. The patient was positioned supine, with the canthomeatal line perpendicular to the examination table. Anteroposterior (AP) runs were performed with 10°–15° caudocranial angulation. The caudocranial angulation was used to ensure that the proximal portion of the posterior cerebral arteries was projected below the transverse sinus. The run was terminated when washout of contrast bolus from the venous sinus was nearly complete. In a patient with normal cardiac output, a run would typically require 60–65 frames at four frames/sec. For morphologic information, the intracranial vascular anatomy was optimized by remasking and integration of images. The image data after digitization were transferred from disk to magnetic tape for off-line processing. Regions of interest (ROIs) were outlined on the CRT screen for defining the anterior (ACA), middle (MCA), and posterior (PCA) cerebral vascular distribution (fig. 1A). Precautions were taken to avoid inclusion of a major venous sinus from the ROI. The time-density curves of each ROI encompassing the distribution of each major cerebral artery territory were obtained to generate the peak arrival time of the contrast bolus (TMAX) in that vascular distribution. Raw data of the time-density curves were analyzed by both gamma variate [6] and the polynomial curve-fitting techniques.

Time parameters describing the arrival of intravenous contrast bolus included TMAX, time to half peak (THMX), and mean transit time (MTT) and were generated by gamma variate fit. The polynomial fit only generated TMAX and THMX parameters. Recent work in our laboratory has shown that all these time parameters are sensitive to vessel stenosis. The time-density curves were normalized when side-to-side comparisons were made. Scaling constants were not used so negative values appear on the *Y* axis. The difference between time to peak (ΔTMAX) was used rather than absolute numbers to minimize the effect of veiling glare, geometric distortion, and image lag in the image intensifier. Of 20 patients in the control group, the gamma variate fit and the polynomial fit yielded comparable results by visual inspection of the curves in 12 patients, while the polynomial fit was superior to the gamma variate fit in the other eight patients. Since the TMAX from the polynomial fit approximated the peak from the raw data better than the gamma variate fit, TMAX generated by polynomial fit was used throughout this study for side-to-side comparison and for all statistical analyses. The raw data and the polynomial fit for the transit of the contrast bolus in the MCA distribution of a patient with normal carotid bifurcation and normal cardiac output have the configuration of a typical indicator dilution curve (fig. 1B). Recirculation had not occurred during the time interval of data acquisition. The polynomial fit comparing the right and left MCA territory in the same patient is illustrated on figure 1C. The angiogram, IVDSA, and the polynomial fit of a patient with tight extracranial carotid stenosis are illustrated in figure 2. Statistical analyses were performed using the Student *t* test.

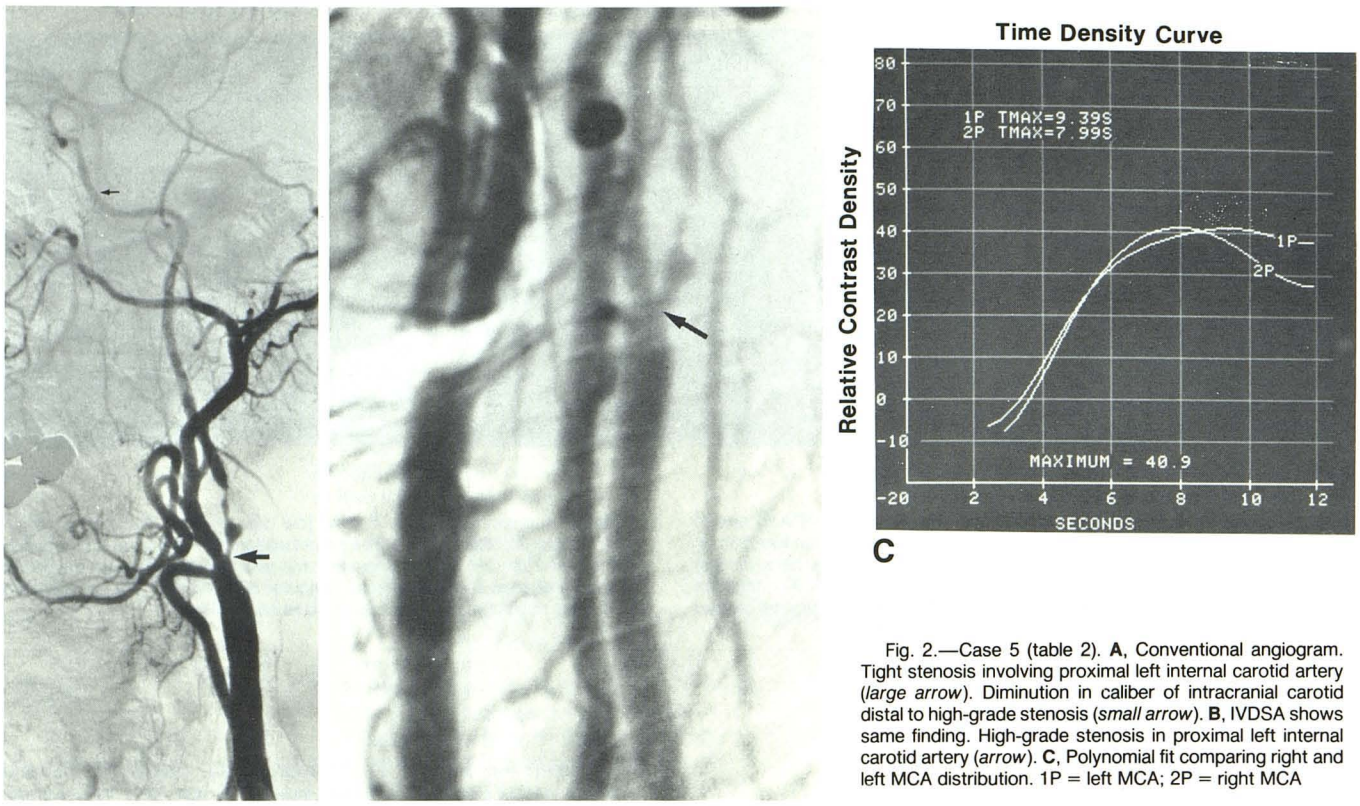


Fig. 2.—Case 5 (table 2). **A**, Conventional angiogram. Tight stenosis involving proximal left internal carotid artery (*large arrow*). Diminution in caliber of intracranial carotid distal to high-grade stenosis (*small arrow*). **B**, IVDSA shows same finding. High-grade stenosis in proximal left internal carotid artery (*arrow*). **C**, Polynomial fit comparing right and left MCA distribution. 1P = left MCA; 2P = right MCA

TABLE 3: **ΔTMAX for Each of the Three Major Vascular Territories in Asymptomatic and Symptomatic Control Groups**

Group	No. of Patients	Mean ΔTMAX in sec (SD)		
		Middle Cerebral Artery	Anterior Cerebral Artery	Posterior Cerebral Artery
Asymptomatic	9	0.093 (0.079)	0.097 (0.084)	0.126 (0.059)
Symptomatic	11	0.186 (0.133)	0.191 (0.171)	0.230 (0.229)
Combined	20	0.140 (0.119)	0.152 (0.146)	0.189 (0.187)

Note.—ΔTMAX = absolute difference between hemispheres of peak arrival times of intravenous bolus of contrast material.

TABLE 4: **ΔTMAX for Each of the Three Major Vascular Territories in Asymptomatic and Symptomatic Patients with Unilateral Tight Extracranial Carotid Stenosis or Occlusion**

Group	No. of Patients	Mean ΔTMAX in sec (SD)		
		Middle Cerebral Artery	Anterior Cerebral Artery	Posterior Cerebral Artery
Asymptomatic	7	0.653 (0.379)	0.118 (0.159)	0.235 (0.265)
Symptomatic	4	1.31 (0.137)	0.525 (0.079)	0.245 (0.06)

Note.—ΔTMAX = absolute difference between hemispheres of peak arrival times of intravenous bolus of contrast material.

Results

In the normal control group, ΔTMAX and its standard deviation for each of the three major vascular territories as derived from polynomial curve-fitting technique are shown in table 3. The ΔTMAX in each of the three major vascular territories demonstrated no significant statistical difference between the symptomatic and asymptomatic subgroups for MCA, PCA, and ACA distributions. These two subgroups, therefore, were combined for subsequent statistical analysis. For this analysis, ΔTMAX was defined to be abnormal if it was greater than 2 SD from the mean in these 20 controls. The ΔTMAX and its standard deviation between the two hemispheres for the MCA, ACA, and PCA distributions in 11 patients with high-grade stenosis or occlusion are shown in table 4. While there is no significant difference in the ΔTMAX in the PCA distribution for the symptomatic and asymptomatic abnormal subgroups, a significant difference was demon-

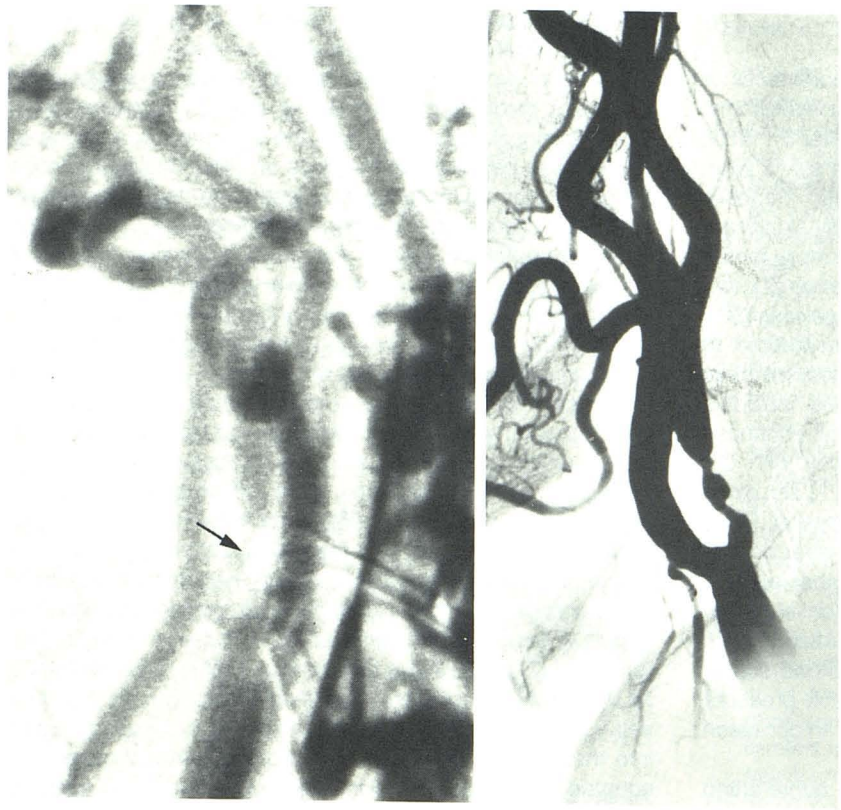


Fig. 3.—Case 8 (table 2). **A**, Preoperative IVDSA. High-grade stenosis in proximal right internal carotid artery (*arrow*) in patient with asymptomatic bruit. **B**, Finding confirmed on conventional angiogram. **C**, Gamma variate fit demonstrating ΔTMAX of 1.15 sec on preoperative study between two MCA territories, right slower than left. 1:T(PK) = TMAX of left MCA distribution; 2:T(PK) = TMAX of right MCA distribution; MTT = mean transit time; T(HMX) = time required for contrast bolus to reach one-half peak value. **D**, Postoperative gamma variate fit in MCA territory. ΔTMAX is now 0.18 sec. Discrepancy of data along *Y* axis between pre- and postoperative studies was from difference in performance characteristic of image intensifier between two studies. An identical volume of contrast material was used for these studies. Using similar window and centering, vascular structures in preoperative study appeared darker on CRT screen than in postoperative study. Absolute difference between peak and troughs is not that dissimilar between two studies.

strated for the MCA and ACA distributions ($p < 0.01$). The ΔTMAX of the MCA territory in patients with carotid occlusive disease was significantly different from the control group whether the patient was symptomatic or asymptomatic ($p < 0.001$). The ΔTMAX of the ACA territory showed a significant difference only between symptomatic abnormal and control groups ($p < 0.001$), not between asymptomatic abnormal and control groups. The ΔTMAX of the PCA showed no significant difference between any of the groups. Expressed slightly differently, the symptomatic abnormal patients demonstrated significant ΔTMAX in both the MCA and ACA distributions when compared with control patients, whereas the asymptomatic abnormal patients demonstrated significant ΔTMAX only in the MCA distribution. The mean ΔTMAX in the MCA and ACA distributions of the symptomatic patients with carotid occlusive disease were about 9 and 3.5 times that of the normal control, respectively.

Serial pre- and postendarterectomy studies were available for analysis in two patients. One patient (case 8) had a history of an asymptomatic bruit on the right side, and both preoperative IVDSA and standard intraarterial angiography demonstrated high-grade stenosis on the right (figs. 3A and 3B). The preoperative ΔTMAX in the MCA distribution was 1.15 sec, right delayed compared with left (fig. 3C). The postoperative study demonstrated a widely patent proximal right internal carotid artery. ΔTMAX in the MCA distribution in the postoperative study decreased to 0.18 sec, which is in the normal range (fig. 3D). This indicated the hemodynamic effect of the stenosis had been corrected. One patient (case 9) had a symptomatic bruit caused by a 95% stenosis of the right internal carotid artery. ΔTMAX in the MCA distribution was 1.42 sec, with the right side delayed compared with the left. After a superficial temporal artery–MCA bypass, the ΔTMAX in the MCA decreased to 0.55 sec, indicating the hemodynamic effect of carotid stenosis had been partially compensated by surgery.

Discussion

Cerebral ischemic symptoms result from hemodynamic or embolic processes; the latter account for most symptoms, although the two processes can operate concomitantly. While there is no general agreement in the literature as to what constitutes a hemodynamically significant carotid stenosis, one may consider a lesion to be hemodynamically significant in the cerebral circulation if it results in compensatory measures such as the recruitment of collateral circulation [7], low regional cerebral blood flow (rCBF), or an impaired rCBF response to induced hypercapnia [8]. The ideal method to estimate the functional tissue perfusion is direct determination of cerebral blood flow in vivo. Various techniques, including rCBF with xenon inhalation techniques [8], xenon-enhanced CT brain scan [9], positron emission tomography with O^{15} [10], and single-photon emission tomography [11] are being investigated with the potential of fulfilling this function. However, even a normal rCBF does not indicate to what degree collateral reserve has been used. There is also poor correlation between morphologic information obtained from standard intraarterial angiography and functional tissue perfusion [12]. A gross estimate of the adequacy of collateral blood supply can be obtained by measurement of carotid back pressure either intraoperatively [13] or preoperatively with an occlusion balloon at the time of carotid angiography [14].

Rapid-sequence IVDSA complemented by curve-fitting techniques is capable of providing accurate TMAX for side-to-side comparison of intracranial perfusion. This technique holds promise for providing physiologic information about carotid stenosis and the adequacy of collateral cerebral blood flow. Comparison of ΔTMAX between the two hemispheres provides a rough but global estimate on the effects of collateral circulation distal to an occluded artery because this technique simultaneously delivers contrast material into the territory distal to the occlusion from all possible collateral sources. In our normal controls, the ΔTMAXs were 0.14 sec, 0.15 sec, and 0.19 sec in the MCA, ACA, and PCA distributions, respectively. Without computer analysis and curve-fitting techniques, it would be extremely difficult to diagnose accurately side-to-side differences since visual comparison of raw data is inadequate. In patients with carotid occlusive disease, direct demonstration often can detect the discrepancy between the two hemispheres, but accurate documentation of the TMAX can still only be determined graphically. The circle of Willis provides the most efficient collaterals and may not significantly increase ΔTMAX. Anatomic anomalies [12] or acquired disease in the circle of Willis can potentially decrease this collateral pathway, resulting in an increased ΔTMAX. Sequential increases in ΔTMAX between the two hemispheres on follow-up examinations provides an index of depletion of the more efficient collateral pathways, and patients with this trend may eventually be at risk for infarction on a hemodynamic basis. In patients with symptoms of cerebral ischemia, identification of this hemodynamic factor does not eliminate a possible embolic etiology since these two etiologies are not mutually exclusive.

The "intracranial determinants" that affect the configuration of the time-density curves include but are not limited to several factors: functional capacity or the presence of an anatomic anomaly in the circle of Willis, the existence of collateral pathways through the ophthalmic artery, the presence of leptomeningeal collaterals, and the peripheral resistance of the vascular bed distal to the circle of Willis. Clinical evidence suggests that asymptomatic patients with hemodynamically significant internal carotid stenoses (defined as 87% reduction in the diameter or greater than 30% reduction in internal carotid blood flow) [7] have a lower cerebrovascular resistance than their symptomatic cohorts, due to autoregulation. Delay in the transit of contrast bolus and perhaps ischemic symptoms would occur after the maximal dilatation and recruitment of the more efficient collaterals had been exhausted. This situation probably occurred in our four symptomatic patients with tight carotid stenosis or occlusion where both MCA and ACA distributions were affected. In the seven asymptomatic patients, significant delay in the transit of contrast bolus only occurred in the MCA distribution. There are two implications of these findings: (1) inability to redistribute blood across the anterior communicating artery between the two ACA territories is a contributing factor to ischemic symptoms and (2) a significant reduction in TMAX of the ipsilateral MCA and ACA territories combined represents an absolute delay in the transit of contrast material (and therefore blood) into that hemisphere. Transit of contrast material cannot be equated with tissue perfusion, but our limited data do suggest a correlation between ischemic symptoms and significant delay in the transit of contrast bolus through the entire hemisphere.

Our data showed a clear separation in the transit of contrast material in the MCA distribution between the control population and those patients with stenotic or occlusive carotid disease whether or not they were symptomatic. The asymptomatic patients with unilateral stenosis or occlusion have clearly drawn on the more circuitous collaterals as indicated by about four times greater MCA ΔTMAX between the hemispheres. In addition, the symptomatic patients with stenotic or occlusive carotid disease showed a further increase in the ΔTMAX by a factor of 2 compared with similar asymptomatic patients. At what level the collateral supply is exhausted and cerebral ischemia and infarction result has not been determined. In patients with cerebral ischemia but without blood-brain barrier disruption, the difference in the mean parenchymal transit time of iodinated contrast material in the MCA territory of the two hemispheres obtained by dynamic CT scanning [15] was very similar to our results. Of note is that in the series of Takahashi et al. [15], all patients who suffered cerebral infarction had an interhemispheric difference in the MCA territory exceeding 1.6 sec. The ΔTMAX measurement may provide a valuable index of the collateral status, especially in asymptomatic patients with tight carotid stenosis. Serial ΔTMAX determinations may prove valuable in following asymptomatic patients. In this manner, each patient serves as his own control for longitudinal follow-up. A pattern of progressively increasing ΔTMAX would perhaps indicate either an absolute decrease in the potential collateral blood supply or increased dependence on the more circuitous col-

laterals. This may be a rationale for prophylactic surgery.

There are several important limitations in this technique. The methodology involves comparison of the two hemispheres; therefore, detection of an abnormality depends on a difference between them. For example, if both carotid arteries have significant stenosis, the side-to-side comparison is less useful and may not detect a significant delay in TMAX. A second major limitation is related to poor cardiac output or large central blood volume; this leads to a prolongation or flattening of the peak of the contrast bolus [16], reducing the sensitivity of TMAX in detecting interhemispheric differences. The third major limitation is from the phenomenon of "interhemispheric steal," resulting in interhemispheric redistribution of cerebral blood flow [17, 18]. This phenomenon could result in no significant difference in the ΔTMAX in a patient with a unilateral high-grade stenosis. Since there is experimental evidence to support the fact that the MCA distribution is protected at the expense of other vascular territories by the phenomenon of interhemispheric redistribution, a significant ΔTMAX in the MCA would suggest that the redistribution potential from collateral sources may be approaching its limit. It should be reiterated that this technique does not yield quantitative blood flow. At the expense of being more invasive, this technique could be used in aortic arch injections, which would decrease the effect of poor cardiac output and large central blood volume.

In summary, there are several potential clinical applications of this technique: (1) Patients with carotid stenosis could be divided into those who are well compensated by collateral flow (i.e., with symmetric TMAX) and those who are poorly compensated (i.e., with large ΔTMAX). The latter may be at risk for possible infarction on a hemodynamic basis. The identification of this subgroup could be important if the patients are being evaluated preoperatively for major vascular reconstructive procedure with a potential of intraoperative hypotension. (2) Serial follow-up studies could be conducted of patients with asymptomatic bruits to correlate the progression of extracranial stenosis with the status of intracranial collateral blood supply. (3) The hemodynamic effects of carotid endarterectomy or extraintracranial bypass surgery could be evaluated. It is important to emphasize that the presence of an abnormally increased ΔTMAX does not mean a cerebral vascular accident is imminent, nor does it provide blood flow data. It simply gives an index of diminished collateral cerebral blood supply or increased dependency on more circuitous collaterals. Documentation of serial prolongation of ΔTMAX in the MCA distribution may be an indication for prophylactic surgery in patients with asymptomatic bruits.

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