Role of Carotid Stenosis in Ischemic Stroke
Chang Z. Zhu, MD, and John W. Norris, MD

Using Doppler ultrasonography, we evaluated the frequency and severity of carotid artery stenosis in 261 patients with carotid ischemic strokes, 813 patients with carotid transient ischemic attacks, 500 patients with asymptomatic neck bruits, and 500 controls. Most patients with strokes and transient ischemic attacks had no associated carotid artery disease (55% and 64%, respectively), and such patients without neck bruits were even more likely to be without carotid artery disease (69% and 77%, respectively). Carotid stenosis was more frequent and more likely to be severe in symptomatic than in asymptomatic patients (p < 0.0002), even after adjusting for age and sex. Carotid stenosis is present in only a minority of patients with strokes and transient ischemic attacks, especially if neck bruits are absent, and the cause of the ischemic cerebral events in most of these patients remains unexplained. (Stroke 1990;21:1131-1134)

Carotid ischemic stroke is commonly attributed to atherosclerosis of the extracranial carotid arteries, with subsequent embolic or hemodynamic cerebral ischemia.1,2 However, data from stroke registers indicate that even after accounting for all carotid and cardiac disease, the etiology in most cases remains unexplained.3

Neck bruits are commonly associated with underlying extracranial carotid artery disease and therefore are associated with an increased risk of stroke.4,5 Although community studies of patients with neck bruits indicated that subsequent strokes were unrelated to the side of the bruit,6,7 later reports including data on noninvasive carotid imaging with clinical follow-up suggest that the more severely stenosed carotid artery, which does not necessarily correspond to the side of the neck bruit, carries the greater stroke risk.5,8 We previously evaluated the frequency of carotid stenosis in 336 patients with asymptomatic neck bruits and found that 61% had ipsilateral and 28% had contralateral carotid artery stenosis.9 We also have investigated the laterality of ischemic cerebral events during follow-up in patients with asymptomatic carotid stenosis and found that 80% of the events occurred ipsilateral to the more stenosed artery; in symptomatic patients, this number was 72%.10 Risk of stroke therefore relates to both the presence of neck bruits and to the underlying carotid lesion, but the relation is still uncertain.

We compared the frequencies of neck bruits and carotid stenosis (determined by Doppler imaging) in asymptomatic patients with the frequencies in patients with transient ischemic attacks (TIAs) and ischemic strokes to determine the role of carotid artery disease in subsequent cerebral ischemic events.

Subjects and Methods
Data were collected between July 1982 and July 1986. The two symptomatic groups comprised 261 consecutive patients admitted with carotid ischemic stroke (in whom carotid Doppler ultrasonography is routinely performed) and 813 patients with carotid TIAs referred to our Doppler laboratory. Patients with vertebrobasilar TIAs were excluded. The two symptomatic groups were divided in subgroups of patients with and without neck bruits. The asymptomatic group consisted of 500 patients referred to our Doppler laboratory with asymptomatic neck bruits. A control group of 500 patients without neck bruits referred to the same vascular laboratory with nonvascular diagnoses had epilepsy, headache, or dizzy attacks.

Neck bruits are recorded in all patients referred to our Doppler laboratory after auscultation by a trained technician or the reporting physician. A previous comparison in our laboratory of the accuracy and consistency of neck bruit auscultation indicated a high concordance among observers.9

The diagnoses of stroke were all made by referring neurologists from the admitting hospitals. The diagnoses of TIAs were screened by the neurologist or neurological fellow attending the Doppler laboratory. Patients with insufficient evidence or, in our opinion, incorrect diagnoses of TIAs were excluded. Carotid Doppler ultrasonography was performed in all patients with the continuous-wave method (Model 1050, Carolina Medical Electronics, Inc., King, N.C.) without B-mode imaging. We previously documented the accuracy, sensitivity, and specificity of this tech-
TABLE 1. Frequency and Severity of Carotid Artery Stenosis in Symptomatic and Asymptomatic Patients

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<tr>
<th>Group</th>
<th>Stroke With Bruit</th>
<th>Stroke Without Bruit</th>
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% Patients

FIGURE 1. Frequency and severity of carotid artery stenosis in symptomatic and asymptomatic patient groups. Dotted areas, 35–75% stenosis; shaded areas, >75% stenosis.

FIGURE 2. Frequency and severity of carotid artery stenosis in symptomatic and asymptomatic patients with neck bruits. White areas, <35% stenosis; dotted areas, 35–75% stenosis; shaded areas, >75% stenosis.
of stroke in patients with atrial fibrillation implies a
only mural thrombosis, but also ventricular aneu-
previously believed. At least 15% of ischemic strokes
are documented to be due to cardiac disease, not
hypertension, and dilated cardiomyopathy. Present
laboratory methods for detecting potential cardiac
sources of emboli are still imprecise, but they are
improving.12

There was a significant difference in the severity of
carotid stenosis among the patients with neck bruises
by group. This was not due simply to differing sex
ratios or mean ages among the groups and therefore
must indicate that more severe carotid artery disease
relates directly to more severe cerebral ischemic
disease.

Our finding that only 1.2% of controls had severe
stenosis is similar to that of other community studies,
and carotid artery disease is clearly uncommon in the
asymptomatic population. For instance, screening
348 unselected volunteers at a “health fair” using a
clinical questionnaire and carotid Doppler ultrasono-
graphy, Colgan et al19 found that only 5% of
patients over age 60 years had any detectable carotid
stenosis and that only 1–3% had stenosis of >80%
diameter reduction. Two similar studies20,21 revealed
detectable carotid artery disease in 5% and 5.9% of
normal populations.

Carotid stenosis is clearly not a major cause of
brain ischemia, especially in patients without neck
bruits. Rational therapy for stroke cannot be insti-
tuted until the cause or causes of stroke are deter-
mined in this large population with unexplained
cryptogenic stroke.

Discussion

Most symptomatic patients with neck bruits in our
study population had an associated carotid artery
stenosis detected by carotid Doppler ultrasonogra-
phy, but carotid stenosis was present in only a
minority of those without neck bruits, so the causes
of their strokes and TIAs remain unexplained. Cardiac
investigations, such as echocardiography, were not
performed systematically in these patients so we
cannot comment on the frequency of cardioembo-
lish. Our data support the observations made in
stroke data banks: our understanding of the basic
mechanisms of stroke is still grossly deficient. Mohr12
appropriately dubbed this idiopathic entity “crypto-
genic stroke.”

Since the carotid Doppler technique in our labo-
atory is inaccurate in diagnosing stenoses of <35%
diameter reduction,10 nonstenosing or only mildly
stenosing carotid sources of potential emboli might
escape detection. However, although nonstenosing
carotid ulcers may occasionally cause cerebral
embolism,13 this point remains controversial,14 and if
it occurs it must be unusual. Also, in studies in which
stroke risk is correlated with the degree of carotid
stenosis, lesions of <50% diameter reduction carry a
very low risk of stroke.8,9 Therefore, even if minor
degrees of carotid stenosis account for a few cases of
cryptogenic stroke, they are unlikely to explain the
majority.

Information from the National Institute of Neuro-
logical and Communicative Disorders and Stroke
stroke data bank indicates that after neurologic
investigation, approximately 22% of strokes can be
attributed to carotid artery disease while 11.6% are
considered lacunar;15 embolism of uncertain origin is
the commonest cause of stroke. Recent advances in
cardiographic imaging, such as the “bubble” echocardi-
ographic technique for detecting a patent foramen
e,15 demonstrate that cardioembolic stroke from
parasadoxical sources is not uncommon. The recent
introduction of transesophageal echocardiography
reveals cardiac sources of emboli missed by standard
echocardiographic techniques.16 Also, the high risk
of stroke in patients with atrial fibrillation17 implies a
more important role for cardiogenic embolism than
previously believed. At least 15% of ischemic strokes
are documented to be due to cardiac disease, not
only mural thrombosis, but also ventricular aneu-
rysms, hypertension, and dilated cardiomyopathy.18

The real percentage is clearly much larger. Present

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KEY WORDS • bruit • carotid artery diseases • cerebral ischemia
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