Clinical Review

Asymptomatic Carotid Bruit

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The asymptomatic carotid bruit is not an uncommon finding in patients aged 45 years and older. The presence of such a finding has been associated with an increased incidence of stroke and cardiovascular death. The optimal evaluation and management of such patients are still unresolved. Noninvasive carotid evaluation appears to be useful for defining the degree of carotid artery stenosis and possibly in identifying a particular subset of patients at high risk for stroke. Whether patients with asymptomatic carotid bruits are best managed medically or surgically remains controversial and will require further investigation.

Although it has been reported that the incidence of stroke has been on the decline since 1945,[1-3] stroke remains the third leading cause of death in the United States.[4-7] There are a reported 400,000 new cases of stroke each year in the United States, and 27 to 50 percent of these patients die within 30 days of the stroke.[8-10] In 1972 the total cost to the nation was $6.2 billion. It is a disease not confined to the elderly, with up to two thirds of strokes occurring in patients less than 65 years old.[11]

Approximately 60 to 84 percent of strokes are related to thromboembolic phenomenon,[8,10,12] and the majority of these are due to atherosclerosis of the carotid and vertebral-basilar arteries. Forty percent of patients with ischemic stroke have the principle obstructive lesion confined to the extracranial vasculature[9,13] with surgically accessible carotid artery disease accounting for up to 15 percent of all strokes.[14]

Stroke has been called one of the "most devastating of human illnesses."[15] Thus far, there is no known medical or surgical therapy effective in limiting or reversing a stroke once it has occurred. Efforts, therefore, have been focused on prevention. Identification and treatment of known risk factors for stroke, such as hypertension, diabetes, hyperlipidemia, cardiac abnormalities (eg, mitral stenosis, atrial fibrillation, mural thrombus post-myocardial infarction), and smoking, has been one approach. In addition, much attention has been directed toward two entities felt to be precursors of ischemic stroke, the asymptomatic carotid bruit and the transient ischemic attack.[9] This paper will focus on the most controversial of the two, the asymptomatic carotid bruit.

Prevalence and Incidence

Cervical bruits, of which carotid bruits are the most common, can be found in 4.4 to 10 percent of people older than 45 years, with a prevalence of 7 to 11 percent reported in a California retirement community.[16,17] The Framingham Study found an eight-year incidence of asymptomatic carotid bruit

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0094-3509/82/090521-03$00.75

Table 1. Natural History of the Asymptomatic Carotid Bruit

Series	Number	Follow-Up	Stroke No. (%)	Death No. (%)
Cooperman et al[26]	60	2 to 7 yr	9 (15)	21 (35)
Dorazio et al[27]	97	5 to 13 yr	18 (19)	35 (37)
Thompson et al[16]	138	45 mo(mean)	24 (17)	50 (36)
Heyman et al[17]	72	6 yr	10 (14)	—
Wolf et al[18]	171	2 yr	21 (12)	47 (27)
Humphries et al[28]	168	32 mo(avg)	4 (2)	—
Kartchner and McRae[29]	1,150	24 mo(avg)	38 (3.4)	—

of 4 to 5 percent in a population between 45 and 80 years old.[18]

Pathogenesis

The most frequent cause of a carotid bruit is an atherosclerotic plaque at the bifurcation of the common carotid artery.[16] Ninety percent of carotid bruits are related to disease in the internal carotid artery and 10 percent to disease involving the external carotid artery.[8,9]

Carotid bruits can be produced by the turbulence of blood flowing through a vessel with a narrowed lumen. A carotid bruit usually becomes audible when the responsible plaque has produced a 50 percent narrowing of the artery's diameter.[19] It has been found to be present in 77 to 88 percent of patients with a carotid artery stenosis exceeding 50 percent.[20,21] Javid et al[22] reported that 60 percent of carotid bifurcation atheromata progressed significantly in size when followed angiographically over time.

The auscultatory quality of the carotid bruit is often helpful to the skilled auscultator in estimating the degree of stenosis,[19] although it is by no means infallible.[20,23] A nonobstructive lesion will most likely produce a rumbling bruit of short duration, whereas a high-grade obstructive lesion more likely will produce a high-pitched bruit of long duration with possible extension into diastole.[8,19] A severely stenotic lesion (greater than 90 to 95 percent stenosis), however, is likely to be faint or even inaudible.[8]

Natural History

Carotid bruits are estimated to be present in 60 to 88 percent of patients with significant carotid lesions (50 percent or greater stenosis and/or ulcerative atheromatous plaque)[16,24] with a false-positive rate of 10 to 28 percent.[21,23] In view of this association with carotid artery atherosclerosis, the patient with an asymptomatic bruit is felt to be at increased risk for stroke. Reports in the literature dealing with asymptomatic carotid bruits, however, have provided conflicting information, with the estimated incidence of stroke ranging from as low as 2 percent to as high as 19 percent.[25]

The frequently quoted data regarding the natural history of the asymptomatic bruit are based on the series listed in Table 1 and should be compared with the expected stroke incidence of 0.2 to 1 percent per year.[2] However, whether these figures are totally applicable to the patient found to have an asymptomatic carotid bruit on routine examination in a private practice is controversial. The first two series in the table were composed of patients referred to a vascular surgery clinic and found to have an asymptomatic carotid bruit[26,27]; thus, a tertiary care center bias may have been present. Thompson et al[16] followed patients referred to a private vascular surgeon who were not subjected to carotid endarterectomy for numerous reasons, including medical conditions taking precedence over the bruit, multiple medical risk factors, patient or referring physician refusal, and the presence of a soft, unilateral bruit. His group of patients not operated on may very well have been a group at extremely high risk for stroke; 15 of the 24 (62.5 percent) patients who subsequently had a stroke did so in the first 12 months of follow-up.[16] The studies by Heyman et al[17] and Wolf et al,[18] although composed of patients most similar to those likely to be seen in a private practice, suffer from a rather small population at risk. Humphries et al[28] followed 168 patients with both asymptomatic carotid bruits and greater than 50 percent

stenosis of the ipsilateral carotid artery on angiography. It should be noted that 111 of his patients had already undergone carotid endarterectomy on the opposite side prior to the study. Additionally, during the follow-up period, 26 (16 percent) patients developed transient ischemic attacks and underwent successful carotid endarterectomy. Of the 1,130 patients followed by Kartchner and McRae,[29] only 590 were truly "completely asymptomatic," with the remainder having "nonhemispheric" transient ischemic attacks. In addition, only 53 percent of the 1,130 were found to have carotid bifurcation bruits (the remainder had sounds transmitted from the aortic arch or the heart), and 119 (10 percent) patients underwent carotid endarterectomy during the follow-up period.

In view of these data regarding cerebrovascular disease and the increased mortality (most of which were attributed to cardiovascular disease) seen in Table 1, most investigators agree that a carotid bruit implies an increased risk of stroke and is evidence of generalized atherosclerotic cardiovascular disease. Whether the carotid bruit is causally linked to the subsequent stroke, however, is debatable. This has important therapeutic implications with respect to the efficacy of prophylactic carotid endarterectomy in preventing stroke. As few as 30 percent of patients with asymptomatic carotid bruits who have a stroke will have a cerebral infarction ipsilateral to their carotid bruit. The remainder have been found to have strokes that are due to causes either not amenable to carotid endarterectomy (such as cardiac emboli, lacunar infarction, cerebral hemorrhage, subarachnoid hemorrhage related to ruptured aneurysm, and vertebral-basilar infarction) or due to an atherosclerotic lesion on the side opposite of the bruit.[17,18]

Physical Examination

It has been recommended that all patients over the age of 40 years should have cervical auscultation performed routinely utilizing the standard 3-cm bell of the stethoscope.[16] In light of the significant cardiovascular implications associated with the carotid bruit, it is important for the physician to be able to differentiate a carotid bruit from the other causes of cervical bruits.

Fifty to sixty percent of cervical bruits are carotid bruits.[8,29,30] Carotid bruits are well localized to the midcervical region just behind and below the mandible[16,24] and decrease in intensity when the stethoscope is "walked" down the neck away from the angle of the mandible. A decreased carotid pulse on palpation and a prominent, enlarged ipsilateral superficial temporal artery are supportive findings, if present.[8] It is rarely possible to distinguish whether the bruit is caused by internal carotid stenosis or external carotid stenosis, although Reed and Toole[31] have suggested that an external carotid bruit will decrease in intensity on compression of the ipsilateral superficial temporal and facial arteries.

A hyperdynamic circulation can produce cervical bruits due to augmentation of blood flow producing turbulence. Anemia, hyperthyroidism, pregnancy, fever, and occlusion of one carotid artery with augmented flow to the opposite carotid artery can all produce cervical bruits via this mechanism.[7,16]

A venous hum, present in up to 75 percent of young adults and children, can usually be easily differentiated from a carotid bruit. Venous hums are generally continuous and heard best at the medial end of the clavicle along the anterior sternocleidomastoid muscle. They are accentuated in an upright position. They can be diminished by numerous maneuvers including pressing the thumb across the cervical veins impeding venous return, having the patient recline, and having the patient perform a Valsalva maneuver.[32]

Heart murmurs, particularly valvular aortic stenosis, can be transmitted into the neck and be mistaken for a carotid bruit.[32] Cardiac murmurs will generally decrease in amplitude as the stethoscope is walked up the neck toward the angle of the mandible.

Atherosclerosis of arteries other than the carotid can also produce cervical bruits. A subclavian artery bruit is usually heard best lateral of the sternocleidomastoid muscle at the root of the neck and can be associated with a decreased ipsilateral brachial blood pressure with a normal ipsilateral carotid pulse.[8] A vertebral artery bruit is heard in the supraclavicular fossa and is transmitted toward the posterior triangle of the neck.[19]

Noninvasive Carotid Evaluation

Noninvasive cerebrovascular studies have been recommended by many investigators in the initial workup of the patient with the asymptomatic ca-

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rotid bruit.[8,16,29,33,34] First of all, they are useful to provide confirmation that a cervical bruit is indeed a carotid bruit, since the overall correlation between angiographically demonstrated carotid disease and bruits is no better than 50 to 60 percent.[25,29] In addition, they provide a means to determine whether a hemodynamically significant carotid stenosis (greater than 50 percent stenosis) is present and whether a stenosis is progressing in size over time.

Numerous noninvasive techniques are currently available, most of which are very sensitive for detecting an internal carotid artery stenosis greater than 50 percent. They are inferior to cerebral angiography in that they are unable to detect nonstenotic ulcerative carotid atheromatous plaques, nor are they able to distinguish between severe stenosis and complete occlusion.[7,35]

Ultrasonic Arteriography

This study is based on a Doppler signal and its oscilloscopic image. Bodily et al[6] found the sensitivity and specificity were both 96 percent in detecting a 50 percent or greater stenosis.

Real-time B-mode Scanner

The B-scanner registers echos related to the acoustical impedance of tissue to produce an instantaneous image of the vessel wall. Ackerman[36] has found the B-scanner to be an extremely valuable component of his noninvasive diagnostic armamentarium at Massachusetts General Hospital.

Carotid Phonoangiogram

This test gives information regarding the amplitude and timing of carotid bruits and is useful to differentiate those bruits at the carotid bifurcation from those transmitted from below this area.[29] A bruit extending throughout systole is consistent with a hemodynamically significant lesion.

Oculoplethysmography

Oculoplethysmography determines the relative arrival time of the ocular pulse wave resulting from the pulsatile arterial flow of the ophthalmic artery, the first branch of the internal carotid artery. Combining oculoplethysmography with the carotid phonoangiogram, Kartchner and McRae[29] reported an accuracy of 89 percent in determining greater than 40 percent carotid stenosis. In addition, they found the studies useful in predicting which patients with asymptomatic carotid bruits were at increased risk for stroke. An incidence of stroke of 1.9 percent was found for 877 patients with a negative oculoplethysmography and carotid phonoangiogram compared with an incidence of 11.9 percent in 147 patients with positive studies when followed two to seven years.

Doppler Ophthalmic Test

This test utilizes a small ultrasound flow probe to detect changes in the flow of the supraorbital artery (a branch of the ophthalmic artery) in response to compression of branches of the external carotid artery. It gives an indirect measure of the status of the ipsilateral internal carotid artery by assessing its capability of providing collateral blood flow. McDonald et al[35] found an overall accuracy of 74 percent in patients with 60 percent or greater stenosis.

Oculopneumoplethysmography

Oculopneumoplethysmography measures systolic ophthalmic artery pressure indirectly by determining the intraocular pressure required to cause ophthalmic artery flow to cease. McDonald et al[35] found an overall accuracy of 97 percent in arteries with 60 percent or more stenosis. Busuttil et al[34] reported that patients with asymptomatic carotid bruits with a positive oculopneumoplethysmography had a significantly increased incidence of subsequent transient ischemic attacks and a "trend for greater stroke incidence."

Digital Subtraction Angiography

Digital subtraction angiography utilizes computer enhancement of intravenous carotid arteriograms. This study permits outpatient arteriography without the risks associated with catheterization and is available at only selected medical centers in the country.[37] A diagnostic accuracy of 89 percent had been reported.[38]

In summary, noninvasive carotid testing appears useful in confirming the presence of a hemodynamically significant carotid stenosis in a patient with an asymptomatic carotid bruit and for serial monitoring to ascertain if the stenosis is progressing. In addition, there is some evidence that it might also be useful in selecting that subset of patients who may have an increased risk of subsequent ischemic neurological events.[6]

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Management

The family physician is faced with a therapeutic dilemma when confronted with a patient found to have an asymptomatic carotid bruit on routine physical examination. There are no definitive, randomized, prospective studies available currently to dictate an optimal course of evaluation and management. Numerous diagnostic and therapeutic options have been proposed by various investigators. These options are not mutually exclusive.

No Specific Treatment Indicated

Some authorities recommend no treatment for patients with asymptomatic internal carotid stenosis.[39]

Risk Factor Management

Because a carotid bruit implies an increased risk for cardiovascular disease, risk factors for stroke (hypertension, cigarette smoking, diabetes, hyperlipidemia, etc) should be identified and treated appropriately.[1]

Patient Education

A patient with a carotid bruit is at increased risk for the development of transient ischemic attacks and should be educated regarding the recognition of such an attack and the need to report the symptom promptly to his physician.[28,40] A patient with the initial onset of transient ischemic attacks is at high risk for stroke,[41] and it is at this time that more aggressive diagnostic and therapeutic maneuvers, including anticoagulant therapy, cerebral angiography, and carotid endarterectomy, should be considered.[42] However, it has been reported that as few as 10 to 30 percent of patients with stroke will have antecedent, "warning," transient ischemic attacks.[13]

Noninvasive Carotid Testing

It has been suggested that these studies be utilized to either (1) confirm the presence and degree of an internal carotid artery stenosis solely for the purpose of assessing the patient's risk for a subsequent ischemic neurologic event so that appropriate patient education can be carried out[6]; or (2) detect those patients who would benefit from angiography and possible prophylactic carotid endarterectomy.[8,16,29,34]

Aspirin Therapy

Fields[24] suggested that patients with asymptomatic carotid bruits should be placed on "two aspirins twice daily after meals." Although there are data to support the usage of aspirin in decreasing the frequency of transient ischemic attacks and the incidence of stroke in certain subsets of patients with cerebral ischemia,[43,44] there is no evidence that antiplatelet agents, including aspirin, are efficacious in patients with solely asymptomatic carotid bruits.

Cerebral Angiography with Possible Prophylactic Carotid Endarterectomy

The most controversial of the therapeutic and diagnostic options, this is based on the premise that stroke due to atheromatous disease of the carotid artery is preventable by carotid endarterectomy if the causative lesion is identified early enough.[36] Many investigators are reluctant to subject an asymptomatic patient to the significant, albeit small, risks associated with angiography and endarterectomy in the face of data felt insufficient to judge whether carotid endarterectomy is efficacious in this situation.[3,6,17,18,38] However, other investigators have reported a markedly decreased incidence of stroke in patients with asymptomatic carotid bruits treated with endarterectomy compared with patients not operated on.[16,45,46] For example, Thompson et al[16] found a stroke incidence of 4.6 percent in 120 patients treated surgically (mean follow-up 55 months) vs a stroke incidence of 17.4 percent in his nonoperated group (mean follow-up 45 months). This study, as well as the other surgical studies, has been criticized because it was not properly randomized and did not indicate whether the strokes that occurred during the follow-up period could be directly attributed to the lesion producing the carotid bruit. Javid et al[46] recommended that angiography and prophylactic carotid endarterectomy not be performed in patients older than 65 years with a history of myocardial ischemia and hypertension because of their high (66 percent) early and late operative mortality.

Conclusion

Considerable controversy exists regarding the management of the patient with the asymptomatic carotid bruit. Studies investigating the natural his-

tory of the patient with an asymptomatic bruit describe an increased incidence of stroke. Noninvasive carotid evaluation appears useful for defining the degree of carotid artery stenosis and possibly in identifying a particular subset of patients with bruits at high risk for stroke. Although the optimal management of the patient is debatable, identification and treatment of risk factors predisposing to atherosclerosis, as well as patient education regarding transient ischemic attacks and stroke, are recommended. Although aspirin and antiplatelet therapy may prove useful in the future, there are still insufficient data to recommend its routine usage. The risk-benefit ratio of cerebral angiography and prophylactic carotid endarterectomy in the management of the patient with an asymptomatic carotid bruit has not been clearly determined. Proof of its efficacy by a well-controlled, randomized prospective study is required before it can be routinely recommended.

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