The Asymptomatic Carotid Bruit — Operate or Not?

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THE QUESTION of how to manage the patient with an asymptomatic carotid bruit is one which is still unsolved and the subject of considerable controversy. By definition a cervical carotid bruit is one which is localized to the midcervical region just behind and below the angle of the mandible. The differential diagnosis of cervical bruit includes physiological murmurs of no significance; venous hum; arteriovenous fistula; angiomatosmal malformations; atherosclerosis of the brachiophallic, subclavian, vertebral, or carotid arteries; loops, kinks, and fibromuscular dysplasia of the carotid artery; and murmurs transmitted from the upper mediastinum. The midcarotid bruit is usually localized and either disappears or diminishes in intensity as one moves the stethoscope down the neck. The bruit tends to become more apparent when stenosis is 50% or greater but may actually disappear when stenosis reaches 85% to 90%. The degree of correlation of internal carotid artery stenosis with an audible bruit is somewhere between 75% and 85%, according to Thompson.1 On the other hand, over-all correlation between demonstrable carotid disease and bruits is about 60% since stenotic lesions may be demonstrated on the arteriogram when an audible bruit is not present. There is no doubt that when present in a patient with symptoms related to the ipsilateral eye or cerebral hemisphere, carotid bruit is a significant clinical finding. The controversy arises with respect to the significance of the bruit in the absence of any such symptoms.

Asymptomatic bruits have been described in three different and perhaps somewhat unrelated contexts; however, information about each one will shed some light on the total picture. First, there is the bruit heard over the carotid artery in a patient whose symptoms relate to the opposite carotid artery and in whom arteriography reveals bilateral disease. Second, there is the patient without cerebral ischemic symptoms who is about to undergo major thoracic or abdominal surgery for another reason and in whom severe carotid stenosis was detected on one or both sides during preparation for other elective surgical procedures or prior to the medical management of hypertension. Third, there is a group of patients, perhaps the largest of all, in whom bruits are detected on routine physical examination but who are otherwise in good health except possibly for a mild to moderate degree of hypertension.

Let us consider first those persons with symptoms lateralized to one eye or hemisphere in whom arteriography demonstrates bilateral carotid bifurcation lesions. The neurological symptoms are localized to one side and the appropriate lesion is successfully treated by surgical repair, what is the significance of the lesion detected in the contralateral carotid artery in the now asymptomatic patient? There are those who would say unequivocally that the presence of such a lesion is a hazard to the patient, and therefore, a second operation should be staged one to two weeks after the first one. Unfortunately, there is no clear-cut evidence to suggest that such a lesion is necessarily a "prestroke lesion" or to support the assertion by some that to leave such a lesion untreated would be a breach of good medical practice. On the other hand, Levine and Sondheimer reported a personal series of 250 patients who underwent surgical reconstruction of the carotid arteries between 1961 and 1963. In 60 of these patients, stenosis was greater than 50% in both internal carotid arteries, but reconstructive surgery of the carotid artery was performed on the symptomatic side only. All of these patients were followed for a minimum of two years, and there were no further strokes. The second side was operated on only if the patient became symptomatic after the first operation, and in their series subsequent surgery was necessary in only two patients.

The second group to be considered includes individuals who enter the hospital for major arterial reconstructive operations in the thorax or abdomen who are noted to have carotid bruits at the time of the admission physical examination. In a few of these, a careful and detailed history may detect subtle evidence of cerebral ischemia, but the vast majority will not provide a history of disordered function of the central nervous system. I first pointed out in the early 1960's that when the attending physician or the operating surgeon who noted the presence of carotid bruits in patients in this group first performed aortocranial arteriography and then operated on one or both carotid arteries, the frequency of intraoperative or postoperative stroke was dramatically reduced.2 On the particular service with which I was associated at that time, 3 to 5% of such patients suffered serious postoperative neurological deficits, many of them permanent. After these precautions were observed, this figure was reduced to an almost negligible morbidity from neurological disturbance. Javid et al.3 in 1971 reported 56 patients who were asymptomatic for cerebral ischemia but in whom severe carotid stenosis was detected on one or both sides during preparation for other elective surgical procedures or prior to the medical management of hypertension. In their group of patients in whom carotid arteries were operated on first, there was one surgical death and two postoperative strokes. These authors were of the opinion that endarterectomy for asymptomatic carotid bruits in this setting was ill advised in hypertensive patients over 65 years

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of age who have a history of myocardial infarction. In younger patients without several risk factors, however, they recommended carotid endarterectomy for severe internal carotid stenosis. This problem was further explored by Lefrak and Guinn in 1974, and their conclusions were essentially the same. However, they stated, "The ultimate answer to the question of whether or not this type of protective surgery is really beneficial in preventing strokes will depend upon the initiation and results of a randomized prospective study of surgical and nonsurgical treatment of matched groups of asymptomatic patients with carotid artery stenosis."

The third and perhaps the most controversial group of all includes those persons who have unilateral or bilateral bruits detected during routine annual physical examinations or at the time of examination for additional life insurance. It is a very difficult decision indeed to recommend to a patient who has a bruit detected under such circumstances that he should undergo an operation which, like all major operations, has a significant, serious morbidity and mortality. I have personally long held the opinion that such procedures should be done only by surgeons who are doing these operations frequently and who have demonstrated that in their hands the procedure can be performed with a morbidity and mortality of 1% or less. It is not a procedure to be undertaken by someone who does this type of arterial surgery only occasionally. If we examine the record of Thompson, who is well known throughout this country as a vascular surgeon highly experienced in dealing with this type of problem, we find that he reports 149 carotid operations in 114 patients classified by him as having asymptomatic carotid bruits. In this group of patients, there was no operative mortality, but there were two permanent deficits, both described as mild. During the long-term follow-up period there were no fatal cerebral infarcts, but one patient had a major stroke and another one a moderately severe stroke. What Thompson uses for comparison as a "control series" is a group of 102 of his patients not operated on for a variety of reasons: (1) the patient did not choose to have arteriography or surgery; (2) the referring physician did not want the studies done; and (3) the bruit was unilateral and soft. He indicates that in his opinion a soft bruit, particularly one that is unilateral, is less significant. It is difficult to be sure what evidence he has to support this latter contention. During a ten-year follow-up period in this group of patients, 28 developed transient ischemic attacks and were operated on, and 48 had what he referred to as "frank strokes" from two days to four years after detection of the bruit. In the summary of his report, he states, "If hazardous lesions are demonstrated, carotid endarterectomy may be recommended for selected patients without multiple risk factors to prevent the occurrence of ischemic cerebral episodes." Unfortunately, we have no evidence which enables us to state unequivocally which lesion is hazardous.

Thompson and others, including Humphries et al. and Moore et al., suggest that some recently developed and noninvasive screening techniques might be helpful in determining the hemodynamic significance of these bruits and thereby assist in the decision as to which patients require arteriography and operation. Unfortunately, hemodynamic alterations do not provide all of the information necessary since many nonobstructing lesions which are ulcerated may be responsible for the downstream embolization of fibrinoplatelet aggregates or other atherosclerotic debris. Some of the ultrasonic imaging techniques now under development may provide additional valuable information. In spite of the potential benefits which might be derived from the use of such noninvasive methods, I would certainly support the position taken by both Humphries and Moore that a randomized multicentered study should be undertaken to shed further light on this matter. The solution to the problem is of no small importance since every day across this country more and more patients with asymptomatic carotid bruits are being operated on by surgeons with varying degrees of experience and competence. It is difficult to justify to a patient who was previously working regularly and otherwise functioning normally or to the family of such a patient the occurrence of a paralyzing and perhaps permanent stroke.

In the past several years there has been considerable anecdotal evidence that pharmaceutical agents, particularly aspirin, which suppress platelet functions may influence the occurrence of transient cerebral ischemic attacks. Support for this form of medical therapy has now been published in a report from the Joint Study of Cerebral Ischemia (AITIA study), supported by the National Heart, Lung, and Blood Institute. The reduction or cessation of TIA's by aspirin was most clearly demonstrated in patients with lesions appropriate to their symptoms. I firmly believe that patients in the third group of asymptomatic bruits described in the foregoing review should be placed on two aspirin twice daily after meals rather than subjecting them to a surgical operation for which the need is still unproven. It is necessary, of course, to eliminate from consideration of such medical management those patients who are suffering from blood dyscrasia or active peptic ulcer. Furthermore, another drug shown to have similar properties should be used if the patient has a clearly demonstrated idiosyncrasy to aspirin.

**Conclusions**

The indications as well as the contraindications for arterial reconstructive operations in patients with transient ischemic attacks, progressing stroke, or completed stroke have been reasonably well defined. The "risk-benefit ratio" of prophylactic surgery for patients with asymptomatic carotid artery stenosis is unknown. Furthermore, the natural history of asymptomatic carotid artery stenosis is also unknown.

A major controversy still exists with regard to indications even for angiography in individuals with asymptomatic carotid bruit. Some surgeons take the position that while it is not possible to make the asymptomatic patient feel better, it is certainly feasible to keep him or her from getting worse by preventing stroke. If one were sure that this statement was in all respects valid, then there would be no controversy, but none of the operated series thus far reported has been from controlled studies. An adequate answer to the question posed in the title of this review will be forthcoming only when the natural history of asymptomatic carotid artery stenosis is studied and the opportunity provided for evaluating the results of prophylactic operation in altering this natural history. A proposal describing the clinical material and methods of procedure for such a controlled
study have been provided by Moore et al. in a recently published paper. It is earnestly hoped that there are physicians and surgeons in this country with experience in performing these operations who would be willing to undertake such a controlled trial.

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Editor’s Note

As Dr. Fields points out in this paper, there is controversy about the management of patients with asymptomatic carotid bruits. I agree substantially with his opinions. Carotid endarterectomy, among other surgical procedures, should be done only by those surgeons who are experienced, who have an opportunity to perform the procedure frequently, and who have demonstrated ability by meeting the criteria stated by Dr. Fields. It is of little consequence whether a neurological surgeon or a vascular surgeon does the procedure; experience and ability are the criteria. Obviously, the same situation holds for angiography: for patients with known or suspected cervicocranial atherosclerotic disease, angiography should be done only by an angiographer with demonstrated experience and ability. This opinion is not based on theory or a desire to restrict certain medical or surgical procedures to specific institutions or persons but on experience gained from preoperative and postoperative evaluations of patients.

Dr. Fields correctly points out that there is no strong evidence that carotid endarterectomy is beneficial for patients with asymptomatic carotid bruits. Unfortunately, there also is no strong evidence that platelet antiaggregating agents are beneficial. If a bruit is caused by an atherosclerotic plaque, it seems reasonable that prevention of a stroke would best be achieved by endarterectomy. Accurate data are difficult to acquire, but most physicians who deal extensively with cerebrovascular disease probably have seen patients with untreated asymptomatic carotid bruits who have developed ischemic cerebral infarcts. If carotid endarterectomy is not feasible because of specific contraindications or because a suitable angiographer and surgeon are not available, platelet antiaggregating agents may be useful.

There has been considerable trepidation about suggesting in print or in public discussion that carotid endarterectomy might be a suitable treatment for a patient with an asymptomatic carotid bruit because of a fear of exposing patients to needless risk at the hands of inexperienced angiographers and surgeons. This need not be so if priority is given to the patient’s best interests in decisions about treatment or referral.

Undoubtedly, many readers of this issue will have different opinions, some of which will be strongly held. Comments are welcome and may be published in a later issue. Suggestions about other controversial topics that could be considered also are welcome. — A. G. Waltz
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Stroke. 1978;9:269-271
doi: 10.1161/01.STR.9.3.269

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