Atherosclerosis is a progressive disease which is usually silent clinically until late in its course. The reasons for the clinical silence are apparent upon study of the pathology of the disease. Early atherosclerosis is an inflammatory disease which destroys the normal anatomy of the arterial wall. The destruction of the elastin in the wall causes arterial dilation. Only late in its course does the thickening and scarring produced by atherosclerosis narrow and finally occlude the arterial lumen, although ulceration of an arteriosclerotic lesion may lead earlier in the course of the disease to thromboembolism, with end-organ damage.

The first sign of advancing atherosclerotic stenosis is often an arterial bruit. This occurs when the arterial lumen is compromised to about 50% of its original lumen diameter, which is equivalent to 25% of the original cross-sectional area. Symptoms usually do not occur until vessel diameter is narrowed by 80% or so, i.e., until the diameter is 20% of its original value (and cross-sectional area only 4% of that of the normal vessel!). In summary, early atherosclerosis is mural, not luminal. Stenosis is a late occurrence in the course of atherosclerosis, and its often apparently rapid progression is the end of a long pathologic process.

Not surprisingly, neither noninvasive methods nor angiography are helpful in detecting early atherosclerosis. Furthermore, very little is known about the natural history of the disease—not only how long it takes for a lesion to progress from its earliest stages to a significant stenosis, but also the rate of progression from a 25% diameter stenosis to an 80% stenosis or a complete occlusion. Nowhere are these questions more pertinent than in the carotid circulation. The carotid bifurcation and the proximal internal carotid arteries are only a few centimeters from the skin surface. The latter vessels supply much of the blood to the brain; an exquisitely sensitive end-organ. Both scientifically and clinically, we would like to know the prevalence of carotid atherosclerosis, how often it progresses to thromboembolism and occlusion, what influence risk factors like age, sex, blood pressure, cigarette smoking, blood lipids, and blood sugar have on the course of the disease, and what risks intervention with angiography and surgery entail. We need to know as well the effects of medical therapy—lowering of blood pressure, treatment of hyperlipidemia and diabetes mellitus, and the use of antiplatelet and anticoagulant drugs, for instance.

It is worthwhile trying to learn from our experience with ischemic heart disease, where these questions were asked, and partially answered, a few years earlier than they have been for cerebrovascular disease. The risk factors for, and clinical concomitants of, atherosclerosis in the coronary and carotid circulations are similar. Better understanding of the natural history of coronary disease has recently shown us that medical therapy may do as well as surgical bypass, and that risk factor reduction, at least for plasma cholesterol concentrations, can arrest and perhaps even reverse the course of atherosclerosis. Carotid disease stands now where coronary disease did a decade ago. Little is known about the effects of medical therapy, and surgical enthusiasm is high.

In this context, the study by Roederer and colleagues, in this issue of Stroke, is a significant step in the right direction. The authors attempt to define the rate of progression of carotid disease, the influence of risk factors, and the consequences of diagnostic tests and therapy. It may be worthwhile to outline some of the advantages and disadvantages of the study as performed. The population was not randomly selected but, rather, included the patients referred to the authors' laboratory for evaluation of previously detected asymptomatic bruits. The disadvantages of this approach are several. There may be a bias in the selection of patients; perhaps those who consult a physician are more likely to have had subclinical or unadmitted symptoms. Therapy was certainly different than it would have been in those patients with bruits found by screening the general population. Furthermore, treatment was not randomized, nor was it selected by the investigators. On the positive side, the population studied had a lot of disease and experienced a sufficiently high rate of progression to allow statistically significant conclusions to be drawn within a realistic time frame. The method used for diagnosis and follow-up was the duplex doppler scan. Its major drawback is that it is not truly quantitative. It is also not very widely used because of the expense of the equipment and the long experience required to use the technique to its fullest potential. On the other hand, duplex doppler carotid scanning is one of the most accurate methods available today for carotid noninvasive diagnosis.
and has been successful in laboratories other than that of the Seattle group. The technique can be used safely and repeatedly for following patients over time.

Unfortunately, the study omits one of the major risk factors, the plasma cholesterol, and a major medical intervention, the use of anticoagulants. However, it does address the effects of age, smoking, the presence of diabetes mellitus and surgical intervention on the patients' outcome.

Despite its shortcomings, the paper by Roederer et al provides some convincing and important data on the natural history of carotid disease. The highlights include:

1. Symptoms and strokes were rare when stenosis diameter was less than 80%.
2. The overall rate of occurrence of symptoms in patients with asymptomatic bruits was remarkably low at 4% per annum.
3. Cigarette smoking and diabetes mellitus were associated with more rapid progression of disease, while hypertension was not.
4. Stopping smoking decreased the likelihood of disease progression.
5. The older the patient, the less rapid was the progression of disease.
6. Treatment with aspirin and/or dipyridamole had no effect.
7. Surgical intervention did not reduce the incidence of symptoms or of stroke in asymptomatic patients; if it were employed only in those with greater than 80% diameter stenosis, however, it might have been of benefit in that group.

The methods, results and conclusions of this study will all be controversial. In a field as important as this one, that is a good outcome which will stimulate the further research necessary to delineate better the natural history of carotid disease and the effects of treatment. Nevertheless, the conclusion by a prominent group of academic surgeons that carotid angiography and surgery should not be performed in asymptomatic patients who do not have evidence, by noninvasive studies, of rapid progression of stenosis, deserves the attention of every physician who deals with patients with carotid disease.

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