The Origin and Distribution of Vasa Vasorum at the Bifurcation of the Common Carotid Artery With Atherosclerosis

Walter J. Bo, PhD, William M. McKinney, MD, and Robert L. Bowden

The purpose of our study was to determine the origin and relation of vasa vasorum to atherosclerotic plaque at the bifurcation of the common carotid artery. We randomly selected 12 unembalmed adult human cadavers, 40–96 years of age. We prepared luminal casts of the arteries from eight cadavers and cleared the arteries from the remaining four cadavers. A network of vasa vasorum surrounding atherosclerotic plaque was observed in five luminal casts and in two cleared specimens; the vasa vasorum originated from the superior thyroid and ascending pharyngeal arteries. Three of the five luminal casts also demonstrated vasa vasorum arising directly from the internal carotid artery distal to the plaque. An extensive network of vasa vasorum was not observed in specimens from the five cadavers relatively free of gross atherosclerotic plaque. Our findings demonstrate the importance of the external carotid artery in giving rise to the vasa vasorum that supply the areas of atherosclerotic plaque. (Stroke 1989;20:1484–1487)

Materials and Methods

We randomly selected 12 unembalmed adult human cadavers, 40–96 years of age. The common carotid arteries of each cadaver were cannulated and flushed with saline. Each artery was injected with methyl methacrylate resin, which was allowed to solidify. In specimens from eight cadavers, luminal casts of the arteries were prepared by completely corroding the tissue with NaOH. The injected intima exceeded a critical depth (0.5 mm for the aorta and 0.35 mm for the first portion of the anterior descending branch of the left coronary artery). Barger et al4 studied the vasa of carotid arteries by cinematography of silicone polymer-injected, cleared human hearts and observed a dense network of vasa in the area of atherosclerotic injury. However, vasa vasorum were rarely seen in the walls of normal coronary arteries.

The bifurcation of the common carotid artery is predisposed to the development of atherosclerotic lesions. The plaques can exhibit intramural hemorrhage,5 which may give rise to thromboemboli, resulting in transient ischemic attacks and/or stroke. A knowledge of the vascularization of carotid atherosclerotic plaque may assist in the understanding of its sequelae. Therefore, our study was designed to determine the origin of the vasa vasorum and their relation to atherosclerotic plaque at the bifurcation of the common carotid artery. This was accomplished by preparing luminal casts of the arteries and by visualizing vasa vasorum in the walls of cleared arteries.

Nutrients are provided to large arteries by vasa vasorum, which supply the adventitia and outer media; the intima and inner media are supplied by diffusion from the lumen. Following the injection of lead chromate, Higginbotham et al1 described a superficial plexus of vasa in the adventitia and deep plexus in the mediadventitial layer of the monkey aorta; the media and intima were free of vessels. Anastomoses between the plexuses were observed. Song et al2 studied the vasa vasorum of the thoracic aortas of sheep, dogs, and pigs and observed that vasa vasorum extended into the internal elastic lamina. However, it was impossible to determine if the subintimal vessels came from the lumen of those vessels.

With the development of atherosclerotic plaque there is a thickening of the intima, which precipitates a change in the vascular pattern of the area. From histologic studies of the human aorta and coronary arteries, Geiringer3 proposed that vessels from the adventitia and the lumen extend into the plaque. However, vascularization from the lumen was observed only when a portion of the arterial intima exceeded a critical depth (0.5 mm for the aorta and 0.35 mm for the first portion of the anterior descending branch of the left coronary artery). Barger et al4 studied the vasa of carotid arteries by cinematography of silicone polymer-injected, cleared human hearts and observed a dense network of vasa in the area of atherosclerotic injury. However, vasa vasorum were rarely seen in the walls of normal coronary arteries.
arteries from the remaining four cadavers were dissected free from the surrounding tissue, fixed in 10% formalin for 12 hours, dehydrated in a series of graded alcohols, and cleared with a 1:1 mixture of methyl salicylate and methyl benzoate. To trace the distribution of the vasa, the cleared specimens were examined with a dissecting microscope.

### Results

The gross pathology in the region of the bifurcation of the common carotid artery varied from a slight thickening to extensive calcification of the artery wall. Of the 12 cadavers, seven had extensive gross atherosclerotic plaques and five were relatively free of atherosclerotic pathology.

Luminal casts from five and cleared specimens from two cadavers demonstrated a network of vasa vasorum in the area of atherosclerotic plaque (Figures 1 and 2); the vasa vasorum originated from the superior thyroid and ascending pharyngeal arteries in all seven. Luminal casts of three of these cadavers also demonstrated vasa vasorum arising from the internal carotid artery distal to the plaque (Figure 3). In one cast specimen a branch from the external carotid artery was observed contributing to the network of vessels.

No extensive network of vasa vasorum was observed in arteries of the five cadavers that were relatively free of gross atherosclerotic pathology (Figures 4 and 5).

### Discussion

Our data show that the vasa vasorum are prominent in areas of marked atherosclerosis. The vasa vasorum originated from the superior thyroid and ascending pharyngeal arteries as well as directly from the lumen of the internal carotid artery distal to the plaque.

There are four stages in the development of gross atherosclerotic plaque, which have been described by Moossy: 1) fatty streaks, 2) fibrous plaques, 3) fibrous...
fibrous plaques with hemorrhage and ulceration, and 4) calcified lesions. Whether there is a correlation between proliferation of the vasa vasorum and the stage of atherosclerotic plaque development is not clear. We observed vasa vasorum entering the adventitia in arteries relatively free of gross atherosclerotic plaques and from the lumen of internal carotid arteries containing marked atherosclerotic plaques. However, using the alkaline phosphatase technique to demonstrate endothelial cells, Peterson et al reported that vascularization of the intima and media of the abdominal aorta occur prior to the development of gross, recognizable atherosclerotic lesion. According to Geiringer, the intima is vascularized by channels from the lumen only when the intima reaches a critical thickness.

The proliferation of vasa vasorum into atherosclerotic plaque suggests neovascularization and indicates that angiogenic factors may be involved. Macrophages and platelets are involved at some stage during the development of atherosclerotic plaque and have angiogenic properties. Therefore, these cells may have a role in precipitating neovascularization.

The extension of vasa vasorum into atherosclerotic plaque is a mechanism to supply essential nutrients to the area. Our findings demonstrate the importance of branches of the external carotid, superior thyroid, and ascending pharyngeal arteries in giving rise to the vasa vasorum. Compromise of the function of these branches may play an important role in the ischemic and hemorrhagic manifestations that occur at atherosclerotic plaques.

References
FIGURE 5. Vasa vasorum (VV) are few at bifurcation of cleared common carotid artery (CC) relatively free of gross atherosclerotic pathology. EC, external carotid artery; IC, internal carotid artery.


KEY WORDS: arteriosclerosis • carotid artery diseases • vasa vasorum
The origin and distribution of vasa vasorum at the bifurcation of the common carotid artery with atherosclerosis.

W J Bo, W M McKinney and R L Bowden

Stroke. 1989;20:1484-1487
doi: 10.1161/01.STR.20.11.1484

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1989 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/20/11/1484