The Rate Of Progression Of Carotid Atherosclerosis

To the Editor:

One does not often see angiographic evidence of the progression of arteriosclerosis in the carotid or brachiocephalic vessels. The following case report shows the dramatic reduction in the lumen of a common carotid artery over a period of seven months.

A right handed housewife born in 1925 began to complain of pain in the right forearm in March 1982. The pain was present with use of the arm and occasionally on first waking in the morning. Peeling potatoes and ordinary housework brought it on. It sometimes radiated proximally as high as the shoulder. When painful the arm was white and uncomfortable.

There was no history of Raynaud’s phenomena, chest, or leg pain. There were no cerebral symptoms.

Examination in May 1982 revealed strong and equal bilateral carotid and subclavian pulses without murmurs. Brachial and radial pulses were strong and normal on the left and the blood pressure was 125/70 in this arm.

On the right these pulses were barely discernible and no blood pressure could be measured. There was no evidence of digital occlusive disease in either hand, heart sounds were normal, and leg pulses and the aorta were normal.

Her family history revealed that a sister had died at age 19 of a brain hemorrhage. Mother was aged 78, father 80 and both were alive and free of cardiac and cerebral vascular symptoms. She was on no medication, was not a diabetic, but had smoked 20 cigarettes a day for 35 to 40 years.

Abnormal investigations included a hemoglobin of 173 gms/L, a hematocrit of 0.494 L/L, mean corpuscular hemoglobin of 35 pg, and an erythrocyte sedimentation rate of 26 mm per hour. Borderline abnormalities included mean corpuscular volume of 100 fl and mean corpuscular hemoglobin concentration of 350 g/L. Her cholesterol was 8.01 m mol/L and triglycerides 2.29 m mol/L. Her lipoprotein profile was not done. Her chest x-ray, ECG, blood gases, white cell count, differential and platelet count were normal.

A diagnosis of right axillary artery occlusion was made. An arch aortogram revealed occlusion of the axillary artery distal to the origin of the subscapular branch. The left common carotid was diffusely, but mildly, narrowed and irregular in its mid portion. The left internal carotid showed a small stenotic lesion about 1.0 cm distal to its origin. Marked stenosis was seen in the third portion of the left subclavian artery.

A right axillary to subclavian dacron (R) bypass was performed with complete relief of her right arm signs and symptoms (Figure 1B shows the bypass prosthesis in place).

In October 1982 she noticed loss of all the vision in the left eye. The vision loss came and went lasting 5—8 minutes each time and occurred...
up to 12 times each day. It was present while she was sitting, standing, or lying, at rest or at work, was always all the vision of one eye. There were no complaints related to her speech, face, arms, or legs. She thought she might have had an occasional day free of visual symptoms but these were rare.

She was treated with warfarin with no change in her complaints. Continuous intravenous heparin during her pre-operative investigations did not stop the intermittent blindness.

In January 1983 she was re-admitted. Her right arm had normal pulses and blood pressure, there were no carotid murmurs and her hematological abnormalities were unchanged. A repeat arch arteriogram and selective left carotid angiogram showed a marked increase in the stenosis of the left common carotid artery. In addition, the internal carotid artery was seen to be critically stenosed 1.0 to 1.5 cm distal to its origin (Figure 1B). An endarterectomy of the left common and internal carotid was performed. The removed tissue consisted of atheromatous plaques with some calcification, and no evidence of intra mural hemorrhage. Attached mural thrombi in various stages of organization were present. She has been free of symptoms since then.

In the seven months between the two angiograms the disease in the left internal carotid has progressed from less than 50% occlusion to about 90% occlusion. Over the same interval the left internal carotid artery disease has also progressed, although not to the same extent. The left subclavian artery which is abnormal doesn’t seem to have changed.

The rate of progression of atheromatous disease has been studied by Javid1 and Javid et al2 and the relative importance of hydraulic forces, hypertension, genetic tendencies, hyperlipidemia and tobacco smoke evaluated. The natural history of intracranial internal carotid artery stenosis has been illustrated by Craig et al.3 They followed 58 patients for 30 months.

The risk/accelerating factors in this present case were the cigarette smoking and the high hematocrit and hemoglobin. The latter were a double hazard by accelerating atherogenesis4,5 and increasing viscosity. The hyperviscous state produces reduced cerebral blood flow and a predisposition to stroke and transient ischemic attacks.6

Sincerely Yours,

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References

An Easy Technique For Catheterization Of The Internal Jugular Bulb

To the Editor:

Many techniques utilizing various anatomical landmarks have been published about cannulating the internal jugular bulb.1,2 However, the catheterization has been difficult and some complication, such as inadvertent carotid puncture, facial palsy and nerve injuries may occur. We describe a new technique for the percutaneous catheterization of the internal jugular bulb, using an ultrasound Doppler blood flow detector.

The location of the internal jugular vein was detected by an ultrasound pencil-shaped Doppler blood flow detector (Ultrasonic Doppler Flow Detector, Model 811, Parks Electronics Laboratory, Beaverton, Oregon, U.S.A.), following Ullman's procedure, which was used for the central venous catheterization.3 Briefly, the patient was placed in the head-down position with his head turned maximally to the opposite side of the catheterization, and the Doppler detector was applied vertically to the skin. A continuous low-frequency signal, called "wind-storm," was audible 1 to 2 cm apart laterally and parallel to the carotid artery, which was detected by the characteristic pulsatile sound. The procedure was repeated at least three times at the following levels: the mandibular angle, the thyroid cartilage, and 2 cm above the clavicle. The vascular points [X] and [●] were marked on the skin in ink.

A 22 gauge needle with syringe was vertically inserted, by which the position of the internal jugular vein is made sure. Then a 16 gauge central venous catheter introducer (Intramedic catheter kit, Nippon Sherwood Co. Ltd, Japan) fitted with a three-way stop cock and a syringe was inserted from 1 cm below the 22 gauge needle at an angle of 30° to the skin, along the line corresponding to the internal jugular vein (fig. 1). After the removal of the inner metallic needle, a radio-opaque

![Diagram of the right neck. The internal jugular vein is indicated by the shaded areas. The symbols [X] and [●] are the vascular points of the internal jugular vein and the carotid artery, respectively, detected by the Doppler detector. 'a' represents a needle inserted, by which the position of the internal jugular vein is made sure. 'b' is the catheter introducer inserted 1 centimeter below 'a' and at a 30 degree angle to the jugular vein.](http://stroke.ahajournals.org/figure/fig1.png)
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