Wall Mechanics of the Stented Extracranial Carotid Artery

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Background and Purpose—Abrupt compliance changes and concomitant nonlaminar flow patterns may contribute to endothelial dysfunction and subsequent neointimal thickening. The aim of this study was to test the feasibility of wall mechanics measurement using B-mode ultrasound image analysis by dedicated software in the stented human carotid artery.

Methods—Carotid Wallstents (Schneider) were placed in the extracranial carotid arteries of 15 patients. B-mode ultrasound examination was performed with a 7.5-MHz probe on the carotid artery upstream; at the proximal, mid, and distal stent levels; downstream from the stent; and on the contralateral internal and common carotid arteries. Carotid diameter (d) and systolic and diastolic diameter changes (Δd) were measured with a dedicated image processing system (IÔ version 3.1, IÔDP), while pulse blood pressure (ΔP) was measured. Diameter compliance (Cd) and distensibility coefficient (DC) were calculated as Cd=2Δd/ΔP and DC=2Δd/ΔP/d and compared between measurement sites.

Results—The evaluation could be completed in 8 of 15 patients. Compliance was significantly lower at the proximal, mid, and distal stent levels (27.7±1.11, 27.38±1.08, 27.38±1.09×10⁻³ mm·kPa⁻¹) than upstream (103.3±36.7×10⁻³ mm·kPa⁻¹), downstream (91.5±41.3×10⁻³ mm·kPa⁻¹), or on the contralateral internal (87.6±28×10⁻³ mm·kPa⁻¹) and common (149.3±47.6×10⁻³ mm·kPa⁻¹) carotid arteries.

Conclusions—Stenting of the extracranial carotid artery induces a compliance mismatch between the native and the stented artery. (Stroke. 2003;34:e222-e224.)

Key Words: carotid arteries • compliance • stents

Carotid artery stenting does not avoid the risk of late restenosis, which occurs at 1 year in 4% up to 75% of patients.¹ Compliance mismatch between the native artery and synthetic vascular substitutes is often reported in the literature as the main contributing mechanical factor for graft failure.² As demonstrated in animal studies, stenting induces a long-standing compliance mismatch.³ We found no report in the literature concerning changes in wall mechanics induced by stenting in human peripheral arteries. The aims of our study were (1) to assess the feasibility of the measurement of stent-induced wall mechanics changes in the extracranial carotid artery by B-mode sonography and (2) to evaluate the resulting compliance mismatch.

Materials and Methods

Patients
Fifteen consecutive patients who underwent stent placement in the extracranial carotid artery were investigated. Strict parallel scanning of the carotid artery downstream from the stent could not be achieved in 7 patients, who therefore were excluded. In the remaining 8 patients (7 men, 1 woman; mean age, 65.6 years; range, 49 to 78 years), the underlying pathology responsible for carotid stenosis was atheroma (n=5), dysplasia complicated by dissection and stenosis (n=1), and radiotherapy for pharyngeal cancer (n=2). All patients were symptomatic, 5 were stage 1, and 3 were stage 3. The carotid stenosis was located on the proximal internal carotid artery in all cases. The mean delay between stent placement and ultrasound evaluation was 16.25 months (range, 3 to 46 months).

Stent Placement
The stents were placed according to the ANAES consensus recommendations.⁴ A Carotid Wallstent (Schneider) was used in 7 patients (nominal size, 7×30 mm in 5 patients, 7×40 mm in 2 patients), whereas a Wallstent (Schneider) of nominal size 7×55 mm was placed in the patient with the dissected carotid artery. All stents were placed across the carotid bifurcation with their proximal part located in the common carotid artery and their distal part in the internal carotid artery.

Diameter and Flow Measurements
B-mode ultrasound images were obtained with a real-time ultrasound scanner and a 7.5-MHz convex probe (Sequita, Acuson). B-mode, duplex, and color Doppler sonography was used in search of atherosomatous plaques, homogeneous thickening of the carotid wall, and hemodynamically significant stenoses. Diameter measurement was performed 10 mm upstream (common carotid artery), at the stent level, and 10 mm downstream from the stent (internal carotid artery) on the stented artery and on the contralateral common and internal carotid arteries.

Real-time B-mode sequences were transferred to the image processing system (IÔ version 3.1, IÔDP) through the Y/C video...
output and then digitized, archived, and processed. The ECG signal was simultaneously acquired and used for cardiac cycle recognition. A strict parallel scanning of the artery was required so that the angle of approach was strictly perpendicular to the vessel axis, thus offering the largest carotid diameter and ensuring optimal axial resolution. At least 3 acquisitions were performed at the same anatomic site, and all values obtained during at least 20 cardiac cycles at each site were averaged.

Pulse pressure (systolic minus end-diastolic pressure) was obtained for each subject by monitoring the right brachial artery with an automated blood pressure measurement device.

Processing of B-mode Images and Sequences
On the stored B-mode images, the observer drew a region of interest encompassing the proximal and distal vessel walls on a length of at least 10 mm. The intima-to-lumen interface of the proximal wall and the lumen-to-intima interface of the distal wall were detected automatically by analyzing the mean gray-level profiles of the frame. The relative displacement of these interfaces on consecutive frames was measured (Figure 1). The waveform of vessel diameter and diameter changes over time was automatically generated, and the average systolic and diastolic diameters and diameter changes were calculated (Figure 2). Diameter compliance (Cd) was calculated as

\[ Cd = \frac{2 \Delta d}{\Delta P} \]

and distensibility coefficient (DC) as

\[ DC = Cd/d \]

where d is diastolic diameter, \( \Delta P \) is average systolic-diastolic blood pressure change, and \( \Delta d \) is average systolic-diastolic diameter change.

Statistical Analysis
Measurement sites were compared for diameter, compliance, and distensibility coefficient by use of a paired t test.

Results
Plaques were present in both common carotid arteries in the 4 patients with atheroma. The 2 patients with radiation-induced lesions had diffuse contralateral intimal thickening. The patient with fibroplasia had irregular parietal thickening and lumen dilatation of both internal carotid arteries. No hemodynamically significant stenosis was found.

The diameter was smaller in both the stented (P<0.05) and nonstented (P<0.005) internal carotid artery than in the ipsilateral common carotid artery. Compliance was significantly lower at the 3 measurement sites within the stent than downstream (P<0.0005), upstream (P<0.005), and on the contralateral common (P<0.0001) and internal (P<0.001) carotid arteries. The distensibility coefficient was significantly lower at the 3 measurement sites within the stent than downstream (P<0.0005) and upstream from the stent (P<0.005) and in the contralateral common (P<0.0005) and internal (P<0.0005) carotid arteries. The distensibility coefficient was significantly lower on the common carotid artery upstream from the stent than on the contralateral common carotid artery (P<0.05) and at the proximal stent level than at the distal stent level (P<0.05) (the Table).

Discussion
Our results show that stenting of pathological human carotid arteries induces a compliance mismatch between the native carotid artery and the stented segment. Although this has been previously demonstrated in atheromatous animal models, human atheroma differs from the minimally calcified atheroma induced by diet and intima abrasion in New Zealand rabbits, and subsequent differences in wall mechanics may be expected. Moreover, the spectrum of underlying pathologies accounted for the wide range of wall mechanics values we

| Carotid Diameter, Compliance and Distensibility Coefficient at the 7 Measurement Sites |
|---------------------------------|-----------------|-----------------|-----------------|
|                                | Upstream From the Stent | Stented Segment | Downstream From the Stent | Contralateral Common Carotid Artery | Contralateral Internal Carotid Artery |
| Diameter, mm                   | 7.685±1.504        | 7.301±1.012     | 5.666±0.773     | 5.02±0.687               | 5.058±1               | 8.286±1.383               | 5.079±0.85               |
| Compliance, 10⁻² mm · kPa⁻¹    | 103.3±36.7         | 27.77±1.08      | 27.38±1.08      | 27.38±1.09               | 91.5±41.3              | 149.3±47.6                | 87.5±28.0                |
| Distensibility coefficient, 10⁻³ kPa⁻¹ | 13.8±5.8          | 3.94±1.74       | 4.78±1.44       | 4.44±2.17                | 16.6±4.9               | 18.1±5.4                 | 17.2±5.1                 |

Figure 1. B-mode image of the internal carotid artery downstream from the stent in patient 2 (atheroma). Intima-to-lumen interface of the proximal wall and lumen-to-intima interface of the distal wall are detected automatically in the region of interest.

Figure 2. Waveform of the internal carotid artery diameter and diameter changes over time are automatically generated, and average systolic and diastolic diameters and diameter changes are calculated.
found on nonstented carotid arteries. Although all these states may, to some extent, reduce arterial wall compliance and distensibility, the dramatic additional decrease in compliance and distensibility after stenting always resulted in a marked compliance mismatch.

The main technical limitation of our study is, despite the stent-related stiffness of the carotid artery, the difficulty in accurately analyzing arterial segments that were not perpendicular to the plane section, especially the internal carotid artery downstream from the stent, so 50% of the patients were excluded. Our study demonstrated a marked alteration of wall mechanics in the stented segment, supporting the compliance mismatch hypothesis. Further investigation of the correlation between distensibility and intima thickening is needed to completely demonstrate this physiopathological link.

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References
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