Arterial remodeling and hemodynamics in carotid stents: A prospective duplex ultrasound study over 2 years

Andrea Willfort-Ehringer, MD,a Ramazanali Ahmadi, MD,a Diego Gruber,b Michael E. Gschwandtner, MD,a Angelika Haumer,a Markus Haumer, MD,a and Herbert Ehringer, MD,a Vienna, Austria

Objectives: This study was undertaken to study negative and positive arterial remodeling processes within self-expanding carotid stents, their interaction, and the resulting changes in hemodynamics over 2 years, with duplex ultrasound scanning.

Subjects and methods: One hundred twelve consecutive patients with 121 successfully stented carotid arteries were examined with color-coded duplex ultrasound scanning the day after the stent procedure and at 1, 3, 6, 12, and 24 months of follow-up. The stent diameters at the proximal, middle, and distal regions, and the maximal neointimal thickness (B-mode) and hemodynamic parameters were recorded. Pre-interventional plaques were assigned to three types: soft, fibrous, and largely calcified.

Results: The diameters of the self-expanding stents steadily increased over 2 years (positive arterial remodeling), from (mean ± SD) 5.80 ± 0.89 mm to 6.77 ± 0.98 mm in the proximal stent area, from 3.51 ± 0.76 mm to 4.92 ± 0.89 mm in the middle stent area, and from 3.7 ± 0.5 mm to 4.68 ± 0.61 mm in the distal stent area (P < .001). Stent expansion was most marked in the middle stent area, depending on the type of pre-interventional plaque. The extent in stent expansion was more in soft than in fibrous and calcified plaques (P < .001). Neointimal thickness increased up to 12 months, and stabilized thereafter. The mean (± SD) neointimal thickness at 3, 6, 12, and 24 months was 0.61 ± 0.28 mm, 0.97 ± 0.39 mm, 1.06 ± 0.36 mm, and 1.12 ± 0.38 mm, respectively. These complex interactions resulted in the dominance of negative remodeling secondary to neointimal proliferation, with an increased flow ratio during the first year, from 1.16 ± 0.37 at day 1 to 1.23 ± 0.46 at 3 months, 1.67 ± 1.37 at 6 months, and 1.57 ± 0.70 at 12 months (P < .001), followed by a tendency to decrease as a result of stent expansion thereafter (flow ratio at 24 months, 1.49 ± .70). Two of 121 stents (1.6%) had recurrent stenosis that required a secondary procedure.

Conclusions: Neointimal proliferation or negative arterial remodeling prevails up to 12 months, and may give rise to rare stent recurrent stenosis. Stent expansion reduces this effect in the first year, and dominates in the second year. This might contribute to the good mid-term outcome of carotid stenting. Poor stent expansion in heavily calcified plaques calls for primary surgical management. (J Vasc Surg 2004;39:728-34.)

In terms of both safety and efficacy, carotid artery stenting has emerged as a potential alternative to surgical treatment in the last few years.1 In general, recurrent stenosis continues to be the “Achilles heel” of catheter interventions. The incidence of carotid stent recurrent stenosis, however, has been low in several single-center studies2-5 and in a global questionnaire survey,6 in which the rate of recurrent stenosis reported was 3.46% in 12 months.

Color-coded duplex ultrasound scanning of the carotid artery is an established method to assess the outcome of surgery, and has been used in large surgical trials.7-9 Its diagnostic accuracy with respect to degree of stenosis has been published in a consensus document.10 Comparing angiograms with Doppler ultrasound scan hemodynamic parameters, Robbin et al11 were the first to show that duplex ultrasound scanning may be an acceptable method for evaluation of patency or recurrent stenosis in carotid stents.

Recurrence in the course of wound healing after endovascular treatment is considered the biologic overresponse to vascular injury secondary to endovascular intervention.12 Two major mechanisms may contribute to lumen loss. Neointimal hyperplasia may occur in an early phase of vascular response, and changes in the local constituents of the arterial wall may lead to arterial shrinking in the chronic wound-healing phase. Stents provide a scaffold to maintain the arterial lumen and to prevent the artery from shrinking in the late phase of stent healing.12 Therefore recurrent stenosis after stent deployment is mainly due to neointimal hyperplasia.13

Recently the neointimal proliferation within carotid Wallstents was studied in a prospective ultrasound-based study comprising more than a hundred successfully stented carotid arteries over 24 months.14 The typical neointimal structure was clearly visualized at the 3-month follow-up and after, increasing in thickness to the 12-month follow-up, while no further relevant changes in the neointima were
observed during the second year. Further systematic reports on dynamic changes in the ultrasonic anatomy of carotid stents over time are lacking so far.

The purpose of the present prospective study, comprising 121 carotid arteries in 112 consecutive patients, was to answer the following questions by means of color-coded duplex ultrasound scanning over a 24-month follow-up:

- What is the balance between neointimal proliferation within the carotid stent (negative remodeling) and the expansion of the diameter of the self-expanding carotid stent (positive remodeling) over time?
- Does the type of plaque before the intervention have a role in positive remodeling of carotid Wallstents?
- How is the resultant development of hemodynamic parameters within carotid stents, the result of negative or positive remodeling over 2 years?

**METHODS**

**Patients.** Between January 1997 and November 1998, 121 carotid arteries in 112 consecutive patients (77 men) were successfully stented. The study complied with the Declaration of Helsinki and was approved by the institutional ethics committee, and written informed consent was obtained from all patients. Bilateral stents were placed in 9 patients, in the same session in 6 patients.

The indication for carotid stenting was based on angiographically documented stenosis greater than 70%. In patients with symptoms (39 of 112), there also had to be high clinical suspicion that the neurologic manifestations were due to microembolization from the carotid plaque, and the interval between onset of hemispheric symptoms and the procedure had to be less than 6 months. In patients without symptoms, fulfillment of any one of several criteria supported stent treatment, including rapid lesion progression within 6 months, documented on duplex ultrasound scans; silent cerebral infarction on a CT scan, consistent with thromboembolism from the carotid plaque; very high-grade stenosis (>90%); contralateral occlusion or severe stenosis; or surgeon request before major surgery.

Carotid artery stent placement was performed by one interventionist, without a protective device, in this early series, in accordance with a previously described technical protocol. Self-expanding Wallstents (Boston Scientific, Natick, Mass) with a rolling membrane were used throughout, which had been sized to the common carotid segment measured from the selective angiogram. Diameters of the stents used were 7 mm (n = 5), 8 mm (n = 31), 9 mm (n = 36), and 10 mm (n = 49). Deployment was followed by dilation within the stent with a balloon catheter that was 5 to 6 mm in diameter. Inasmuch as most arteriosclerotic lesions were located at the carotid bifurcation, the stents were placed across the bifurcation from the common carotid artery into the internal carotid artery.

**Study design.** The prospective follow-up examination included duplex ultrasound scanning and clinical examination. Follow-up visits were scheduled the day before, the day after, and 3, 6, 12, and 24 months after stent placement. Negative remodeling due to neointimal proliferation and positive remodeling secondary to expansion of the stented carotid artery were correlated with the hemodynamic parameters.

Concomitant anti-platelet therapy was started in this early series 3 days before the intervention, and consisted of ticlopidine 250 mg twice a day plus acetylsalicylic acid (ASA) 100 mg/day. ASA was discontinued after 4 weeks. Ticlopidine therapy was continued for 1 year, then converted to ASA 100 mg/day. Similar or identical regimens have been successfully applied in carotid stenting.

**Angiographic evaluation.** Angiographic grading of primary stenoses and residual stenoses after carotid artery stenting was done analogous to the North American Symptomatic Carotid Endarterectomy Trial. Selective angiograms were obtained in at least two planes. Measurements were made on the angiographic view that showed the most severe narrowing. The diameter for calculation was measured with manual caliper placement.

**Color-coded duplex ultrasound scanning.** All examinations were performed with a Vingmed System 5 (VINGMED SOUND AIS; General Electric, Horton, Norway) with a 10-MHz linear array transducer (axial resolution, 0.2 mm); the Doppler frequency of the duplex transducer was 5 MHz. Two experienced sonographers performed all ultrasound measurements. Strict duplex settings were applied. Depth gain compensation, dynamic range, frame rate, and persistence values were held constant. Depth and magnification were adjusted to patient anatomy. The gain was turned so that the intima media of the native common carotid artery proximal to the stent was clearly visualized. The angle of insonation used to obtain B-mode images for morphologic assessment was near 90 degrees. The examination included the common carotid artery proximal to the stent, the entire stent, and the ICA distal to it. Morphologic measurements were taken in the B-mode from three planes in the longitudinal view: anterior, lateral, and posterior. The calipers for measurement were placed manually. The Doppler curve was obtained with an angle of insonation of 60 degrees or less while using angle correction.

The following morphologic parameters were measured at follow-up: (1) Stent diameters were measured at the proximal and cephalad ends of the stent and in the middle stent area, at the site of the plaque causing the pre-interventional stenosis identified directly in the B-mode (Fig 1) or by its distance from the bifurcation. (2) The maximal thickness of the neointima or the layer between the stent and the perfused lumen was measured as reported recently. All data concerning the thickness of the neointima refer to the proximal stent. The individual maximal thickness of the neointima in the proximal stent area was the basis for further calculation, for the following reasons. The duplex settings were adjusted to optimal visualization of the intima media thickness of the native common carotid artery proximal to the stent. The best visualization of the neointima was achieved in the proximal stent, whereas in the middle and distal stent area its visualization was occa-
sionally disturbed by shadowing or difficulties due to patient anatomy.

Stent diameters were measured from inner surface to inner surface, with care taken not to exert pressure on the artery while scanning. These measurements were performed in the B-mode (Fig 1).

Assessment of carotid plaque type before stenting was based on the pre-interventional duplex ultrasound scanning B-mode image, assisted by color Doppler scanning in cases of echolucent plaques. Plaques were assigned to three types in accordance with the Asymptomatic Carotid Stenosis and Risk of Stroke study: soft plaque, mostly echolucent; fibrous plaque, mostly echogenic and hard; and largely calcified plaque, with extensive shadowing and very hard.

The plaque was analyzed visually by two experienced observers. Agreement between the two observers was high (weighted $k = 0.84$) and statistically significant ($P < .0001$). In case of disagreement, the plaques were assigned by consensus.

The acceptable reproducibility of the measurements of the maximal thickness of the layer between the stent and the perfused lumen (in millimeters) has been reported. The reproducibility of the stent diameter measurements was confirmed by two sonographers, who repeatedly measured stent diameters in 8 patients, in three different stent positions, three times a day for 3 consecutive days. Analysis of variance showed that the variance attributable to patients (with a variance component of 0.5) and to differences between the three positions investigated (1.85), as well as the interaction between these terms (0.56), is considerably larger than the variance attributable to the two sonographers (0.0002), the day of the measurement (0), or the measurement repetitions (0.0002).

To grade stent recurrent stenoses, the same hemodynamic parameters as those proposed by Nicolaides et al for the native carotid artery were applied, that is, peak systolic velocity and end-diastolic velocity within the narrowest site of the stent. However, the basis for grading stent recurrent stenoses was the flow ratio, calculated with the formula In-stent internal carotid artery peak systolic velocity/common carotid artery peak systolic velocity. A flow ratio greater than 4 referred to greater than 70% recurrent stenosis, and a flow ratio greater than 2.6 referred to greater than 50% recurrent stenosis.

Statistical analysis. Results are given as mean ± SD. All tests were two-sided, and $P < .05$ was considered significant. Analysis of variance models for repeated measures were used to evaluate the trend over time. Spatial covariance structures were used for changes relative to day 1, because the measurements were unequally spaced in time. The baseline measurements were also included as covariates. Because of the different behavior of some variables in the early and mid-term phases after stenting, intervention analysis was performed with a dummy variable. The Pearson coefficient was used to determine correlations be-

Fig 1. B-mode image of a carotid stent at 12 months shows the sites where proximal, middle, and distal stent diameter were measured. Small arrows mark the ingrowing "neointima" within the stent, a uniformly echogenic line on the luminal side of the stent and a less echogenic area underneath, similar to the intima-media layer of the native common carotid artery (CCA) proximal to the stent. Pre-interventional plaque, compressed by the stent, is marked by large arrows.
Dynamics of stent diameters, neointima, and hemodynamics

<table>
<thead>
<tr>
<th>Follow-up</th>
<th>Day 1 (N = 121)</th>
<th>3 Mo (N = 118)</th>
<th>6 Mo (N = 118)</th>
<th>12 Mo (N = 111)</th>
<th>24 Mo (N = 109)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal stent diameter (mm)</td>
<td>5.80 ± 0.98</td>
<td>6.16 ± 0.98</td>
<td>6.37 ± 0.96</td>
<td>6.57 ± 0.94</td>
<td>6.77 ± 0.98</td>
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<tr>
<td>Middle stent diameter (mm)</td>
<td>3.51 ± 0.76</td>
<td>4.07 ± 0.95</td>
<td>4.28 ± 0.90</td>
<td>4.60 ± 0.85</td>
<td>4.92 ± 0.89</td>
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<tr>
<td>Distal stent diameter (mm)</td>
<td>3.67 ± 0.55</td>
<td>4.04 ± 0.60</td>
<td>4.24 ± 0.61</td>
<td>4.40 ± 0.62</td>
<td>4.68 ± 0.61</td>
</tr>
<tr>
<td>Neointima (mm)</td>
<td>*</td>
<td>0.61 ± 0.28</td>
<td>0.97 ± 0.39</td>
<td>1.06 ± 0.36</td>
<td>1.12 ± 0.38</td>
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<tr>
<td>Carotid flow ratio</td>
<td>1.16 ± 0.37</td>
<td>1.23 ± 0.46</td>
<td>1.67 ± 1.37</td>
<td>1.57 ± 0.70</td>
<td>1.49 ± 0.70</td>
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<tr>
<td>Peak systolic velocity, ICA (m/s)</td>
<td>0.75 ± 0.27</td>
<td>0.88 ± 0.34</td>
<td>0.99 ± 0.48</td>
<td>1.01 ± 0.37</td>
<td>1.00 ± 0.39</td>
</tr>
<tr>
<td>End-diastolic velocity, ICA (m/s)</td>
<td>0.23 ± 0.12</td>
<td>0.28 ± 0.12</td>
<td>0.31 ± 0.20</td>
<td>0.32 ± 0.12</td>
<td>0.31 ± 0.11</td>
</tr>
<tr>
<td>Peak systolic velocity, CCA (m/s)</td>
<td>0.71 ± 0.61</td>
<td>0.70 ± 0.42</td>
<td>0.66 ± 0.25</td>
<td>0.68 ± 0.24</td>
<td>0.72 ± 0.21</td>
</tr>
</tbody>
</table>

Values represent mean ± SD.
N, Number of carotid stents evaluated at follow-up; ICA, internal carotid artery; CCA, common carotid artery.
*At day 1 the neointima is not visualized in duplex ultrasound scans.

between flow and morphologic parameters. Type of plaque and its interaction with the time trend was also considered. Calculations were made with the SAS statistical analysis system, version 8.02 (SAS, Chicago, Ill).

RESULTS

Demographic data for the 112 patients (77 men) included mean (±SD) age, 69 ± 10 years, and mean body mass index, 26 ± 4. Risk factors included arterial hypertension in 93 patients (83%), diabetes mellitus in 43 patients (38.4%), cigarette smoking in 64 patients (57%), and hyperlipidemia in 94 patients (84%). Thirty-nine of 112 patients had symptoms, and 6 of 112 patients had recurrent stenoses after carotid surgery.

In 109 of 121 consecutively stented carotid arteries (112 patients) the scheduled controls with color-coded duplex ultrasound scanning could be completed up to 24 months. Twelve patients were lost to follow-up: 9 patients died, of causes unrelated to the intervention; 3 patients dropped out, 2 because of recurrent stenosis followed by a second intervention, and 1 because of disabling stroke ipsilateral to the stent after thoracic surgery, due to cessation of antiplatelet therapy 2 months after carotid stenting. In another 8 patients morphologic measurements could not be completed because of technical problems, such as extensive shadowing or very distal bifurcation; thus exact measurements of the distal or middle stent diameter could not be obtained.

Peri-interventional angiographic results. The pretreatment degree of carotid stenosis on angiograms was 78% ± 11%, and the residual stenosis immediately after stenting was 5% ± 10%.

Dynamics of stent diameter during follow-up. At the three stent sites (proximal, middle, distal) a significant increase (P < .0001) in mean stent diameter from day 1 to 3, 6, 12, and 24 months was observed (Table). The most pronounced stent expansion occurred in all three sites within the first 3 months, whereas the diameters continued to expand slowly thereafter. The most interesting development in stent diameter was observed in the middle stent area adjacent to the original plaque. The diameter was narrowest at day 1, signaling some residual stenosis due to the neighboring plaque. However, later it revealed the most pronounced increase in stent diameter of all three sites. At 3 months it exceeded the diameter of the distal stent (Fig 2, A; Table). Expressed in percentages, the expansion of the middle stent diameter was 40 ± 23% (range, 0%-116%; baseline, 3.51 ± 0.76 mm), compared with 18% ± 13% (range, 0%-83%; baseline, 5.80 ± 0.98 mm) for the proximal stent diameter and 30% ± 18% (range, 0%-91%; baseline, 3.67 ± 0.55 mm) for the distal stent diameter. At all three sites the baseline stent diameter was inversely correlated with the quantity of expansion up to 24 months (P < .0001).

Influence of plaque composition on dynamics of diameter in the middle stent area. The middle stent area adjacent to the original plaque was the most sensitive. Therefore it appeared worthwhile to investigate the influence of the composition of the pre-interventional plaque as assessed with sonography, based on the dynamics of the neighboring plaque. However, later it revealed the most pronounced increase in stent diameter of all three sites. At 3 months it exceeded the diameter of the distal stent (Fig 2, A; Table). Expressed in percentages, the expansion of the middle stent diameter was 40 ± 23% (range, 0%-116%; baseline, 3.51 ± 0.76 mm), compared with 18% ± 13% (range, 0%-83%; baseline, 5.80 ± 0.98 mm) for the proximal stent diameter and 30% ± 18% (range, 0%-91%; baseline, 3.67 ± 0.55 mm) for the distal stent diameter. At all three sites the baseline stent diameter was inversely correlated with the quantity of expansion up to 24 months (P < .0001).

Other potential influences on the dynamics of stent expansion. In the six recurrent stenoses after carotid surgery the dynamics in middle stent diameter were significantly different from the group with primary stenosis. Its expansion was weaker (P = .038) during the first 6 months, even at 12 months, and more pronounced in the second year (P = .030). However, no significant difference was observed between symptomatic and asymptomatic stenosis (P > .05).

The development of the ingrowing neointima during follow-up was recently reported. Mean values of the maximal neointimal thickness at the proximal stent area at follow-up visits are included in the Table. There is a significant increase in the maximal thickness of neointima over time (P < .0001); a substantial increase was noted up to the
6-month follow-up visit, and it continued to increase slowly up to the 12-month follow-up visit, and was evidently stable during the second year (Fig 3).

**Dynamics of the carotid flow ratio during follow-up.** The mean flow ratio increased significantly ($P < .001$) up to the 6-month follow-up (from $1.16 \pm 0.37$ to $1.67 \pm 1.37$, or 56% from baseline), remained rather stable thereafter up to 12 months, and decreased slightly ($P > .05$) in the second year (Fig 3; Table). Two patients dropped out because of greater than 70% recurrent stenosis, which was followed by a second intervention. One of these patients (flow ratio, 13.6) was excluded after the 6-month follow-up visit, and the other (flow ratio, 10.2) after the 12-month follow-up visit; these patients were not included in the analysis.

The dynamics of the peak systolic velocity and the end-diastolic velocity during follow-up developed similarly (Table). Both of these flow parameters increased significantly up to 6 months of follow-up ($P < .001$), and stabilized thereafter.

**Influence of neointimal proliferation within the stent and stent diameter on the dynamics of the flow ratio.** A significant positive correlation between the change in flow ratio versus change in maximal thickness of the neointima was found ($\rho = .14; P = .003$). No significant correlation was found between stent diameter (irrrespective of neointimal ingrowth) and flow ratio, however ($P > .05$).

**DISCUSSION**

To our knowledge, the present study is the first to examine two counteracting mechanisms in carotid artery stenting, namely, positive arterial remodeling or stent expansion and negative arterial remodeling or lumen narrowing by neointimal proliferation, while offering an explanation for the resulting changes in stent hemodynamics, mirrored by the flow ratio, over time.

Carotid Wallstents expanded steadily from day 1 after deployment over the entire follow-up of 2 years ($P < .001$). As a whole, stent expansion was most pronounced during the first 3 months after deployment, and most distinct in the middle stent region, adjacent to the original plaque. Furthermore, expansion of the middle stent region depended on the type of plaque, which was classified visually at ultrasound scanning performed before stenting. The expansion was most pronounced in “soft” plaques, followed by “fibrous” plaques and, finally, “calcified” plaques ($P < .0001$).

In the present study the stent size was usually chosen according to the common carotid artery segment, which resulted in a markedly oversized self-expanding stent in the ICA segment. The effect of stent oversizing was studied by Piamsomboon et al., who analyzed six different stent-artery ratios in oversized carotid stenting (1.4-2). These authors found that the late lumen loss indexes were significantly lower in the groups with a high stent-artery ratio than in those with a low stent-artery ratio ($P < .01$). Stent oversizing might have favored the continuous increase in stent diameter over the entire 2-year follow-up by exerting steady radial pressure against the arterial wall in this study.

The difference in the dynamics of middle stent expansion between recurrent stenoses after carotid surgery and primary carotid stenoses might be an interesting additional aspect, that is, weaker dilation up to 6 months ($P = .38$), but more pronounced dilation in the second year ($P = .03$) in the surgical recurrent stenosis group. However, the small number of surgical recurrent stenoses in this cohort limits the interpretation of this finding.
Negative vascular remodeling secondary to neointimal proliferation (from zero at day 1 to a mean of 1.0 mm at 6 months and 1.1 mm at 12 months; \( P < .0001 \))\(^{14}\) counteracted the positive vascular remodeling process (Fig 2, A; Fig 3). Thus the flow ratio increased on average from 1.1 to 1.7 at 6 months and 1.7 at 12 months of follow-up (\( P < .0001 \)). During the second year no further change in neointimal thickness was observed (\( P > .05 \)). After a further steady increase in stent diameter during this period, the flow ratio was slightly reduced at the 24-month follow-up; however, the reduction was not statistically significant (\( P > .05 \)).

With regard to the time course of negative and positive arterial remodeling and their complex interaction, neointimal proliferation predominates during the first year, whereas stent expansion might outweigh in the second year. It should be mentioned that recurrent stenoses in self-expanding carotid stents are reported nearly exclusively during the first year,\(^{21}\) a phase with some predominance of neointimal proliferation; in these rare cases the imbalance of negative and positive remodeling might be due to a specific prevalence of neointimal proliferation, or to reduced stent expansion in cases of calcified plaques, or both. The balance of positive and negative remodeling has been meticulously investigated in coronary stents with intravascular ultrasound, mainly on the basis of mid-term results.\(^{22}\) In coronary stents, neointimal proliferation is a major factor in stent dilatation, leading to the well-known relatively high percentage of coronary stent recurrent stenoses.\(^{22}\) The reasons for this difference, that is, the moderate development of neointima and the low rate of recurrent stenosis in carotid stenting, might be complex; the wide lumen and the elastic type of the carotid artery versus the rather narrow coronary artery of the non-elastic type might have a role. Similar differences in the rate of stent recurrent stenosis exist in peripheral arteries, that is, a low rate in the wide iliac artery of elastic type versus a higher rate in the femoropopliteal arteries.\(^{23,24}\) In addition to biologic differences between coronary arteries and the carotid artery with respect to neointimal proliferation within stents, differences in the methodologic approach also must be considered, such as high-frequency (\( \approx 30 \) MHz) intravascular ultrasound in coronary arteries\(^{25}\) and 10-MHz linear transducers with the transcutaneous approach in carotid arteries. The advantage of the transcutaneous approach is obvious: noninvasive repetitive measurements over several years, with no ethical difficulties.

A limitation of this study is that it was a prospective non-controlled single-center study, which exclusively addressed self-expanding Wallstents, applying the platelet-active therapy as described. However, the results provide basic data on the development of morphologic and hemodynamic duplex parameters over 2 years.

In conclusion, in the early phase of the stent healing process, negative arterial remodeling due to neointimal proliferation\(^{14}\) and increase in flow parameters is predominant. This phase calls for enforced platelet-active therapy and careful observation of patients. Positive arterial remodeling or a steady increase in stent diameter due to the mechanical forces of the self-expanding oversized Wallstents dominate in the second year, and might additionally contribute to the good mid-term outcome after carotid stenting. However, the subject requires further long-term observation. Poor stent expansion in heavily calcified plaques could serve as a rationale for primary surgical management in these patients. Our data on remodeling in carotid stents provide a better understanding of hemodynamics. The maximal neointimal thickness in the proximal stent might be a good predictor of stent recurrent stenoses, but in routine clinical practice hemodynamic criteria will not be replaced.

REFERENCES


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