Altered in-stent hemodynamics may cause erroneous upgrading of moderate carotid artery restenosis when evaluated by duplex ultrasound

Maani Hakimi, MD,[a] **Peter Knez, MD,**[b] **Matthias Lippert,**[b] **Nicolas Attigah, MD,**[a] **Karen Nelson,**[b] **Tanja Laub,**[b] **Dittmar Böckler, PhD,**[a] **Thomas Schmitz-Rixen, PhD,**[b] and **Thomas Schmandra, MD,**[b] *Heidelberg and Frankfurt am Main, Germany*

Objective: **To assess the influence of stent application on in-stent hemodynamics under standardized conditions.**
Methods: **Ovine common carotid arteries before and after stent (6 × 40 mm, sinus-Carotid-RXt, combined open-closed cell design; Optimed, Ettlingen, Germany) application were used. Plastic tubes, 10 mm in length, simulating stenosis were placed in the middle of the applied stent to induce different degrees of stenosis (moderate 57.8% and severe 76.4%). Flow velocity and dynamic compliance were, respectively, measured with ultrasound and laser scan; proximal, in-stent, and distal to the stented arterial segment (1 cm proximal and distal) in a pulsatile ex vivo circulation system.**
Results: **Stent insertion caused the in-stent peak systolic velocity to increase 22% without stenosis, 31% with moderate stenosis, and 23% with severe stenosis. Stent insertion without stenosis caused no significant increase in in-stent end-diastolic velocity (EDV) but a 17% increase with moderate stenosis. In severe stenosis, EDV was increased 56% proximal to the stenosis. Compliance was reduced threefold in the middle of the stented arterial segment where flow velocity was significantly increased.**
Conclusions: **With or without stenosis, stent introduction caused the in-stent peak systolic velocity to become significantly elevated compared with a nonstented area. EDV was also increased by stent insertion in the case of moderate stenosis. The stent-induced compliance reduction may be causal for the increase in flow velocity since the stent-induced flow velocity elevation appeared in the stented area with low compliance. Because of altered hemodynamics caused by stent introduction when measured by duplex ultrasound, caution is prudent in concluding that carotid artery stenting is associated with a higher restenosis rate than carotid endarterectomy. Mistakenly upgrading moderate to severe restenosis could result in unnecessary reintervention. (J Vasc Surg 2012;56:1403-8.)**

Clinical Relevance: **Clinical experience and prior studies support the supposition that restenosis after carotid artery stenting in carotid lesions displays erroneously elevated velocity when evaluated by duplex ultrasound (DUS), thus contributing to misleading interpretation of the degree of stenosis. This study, in contrast to studies of other groups, employs exactly the same conditions to measure flow with DUS in an unstented and then stented section of the carotid artery. Since DUS is the first-choice tool for carotid artery evaluation, knowledge about inexactness of the method is essential to avoid errors in treatment or follow-up decisions.**

Carotid endarterectomy (CEA) is one of the most frequently performed vascular surgical procedures to prevent stroke associated with carotid stenosis in symptomatic and asymptomatic patients.[1,2] It is the best-evaluated surgical procedure with an evidence-based medicine level of 1 and a recommendation level of A.[3,4]

Carotid artery stenting (CAS) was initially introduced as an alternative to CEA for high-risk patients or patients with hostile neck anatomy (status after radiation or previous cervical operations such as neck dissection or injury). The clinical outcome of CAS is currently under investigation and equality of treatment, relative to CEA, has not yet been proven. Studies to date comparing the clinical outcome of CAS and CEA regarding stroke prevention, although large and randomized, have not shown a clear noninferiority of CAS to CEA.[5-8] Since CAS is performed not only by vascular surgeons but also by neuroradiologists, interventional radiologists, cardiologists, and angiologists as well, definitive study results are of crucial interest.[9]

One study has associated a significantly higher excess risk of moderate restenosis after CAS than after CEA.[10] The diagnostic method of first choice in postinterventional care to determine carotid patency is duplex ultrasound (DUS). Employing DUS has been reported to reliably detect severe in-stent restenosis (>70%, according to North American Symptomatic Carotid Endarterectomy Trial [NASCET] criteria) but to overestimate moderate (50%-70%) in-stent restenosis.[11] Differentiating between severe in-stent restenosis and moderate in-stent restenosis

From the Department of Vascular and Endovascular Surgery, University of Heidelberg, Heidelberg[a]; and the Department of Vascular and Endovascular Surgery, J. W. Goethe-University, Frankfurt am Main.[b]

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Reprint requests: Maani Hakimi, MD, Department of Vascular and Endovascular Surgery, University of Heidelberg, Im Neuenheimer Feld 110, D-69120 Heidelberg, Germany (e-mail: maani.hakimi@med.uni-heidelberg.de).

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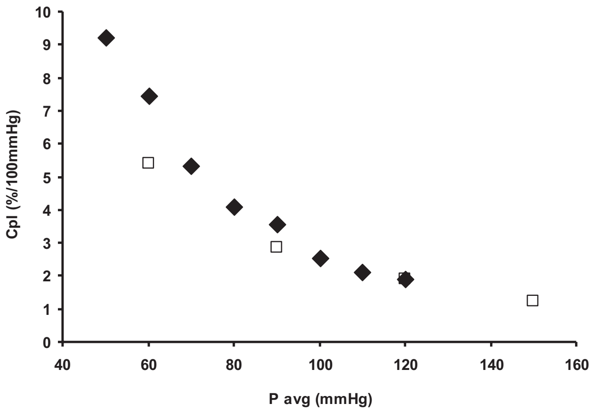

Fig 1. Compliance of human superficial femoral artery and ovine common carotid artery at different pressures. ■ = human superficial femoral artery; □ = ovine carotid artery.

plays an important role in deciding whether revision is necessary.[12-14]

In the clinical setting, it is also relevant whether an increase in flow velocity is due to an early restenosis caused by neointimal hyperplasia or is due to hemodynamic change caused by the stent itself. Whether due to restenosis or stent-induced hemodynamic change, the compliance of the stented artery is reduced. The reduction in compliance has been postulated to be responsible for an increased flow velocity measured by DUS.[15]

Many studies report on the value of DUS for evaluating the degree of in-stent restenosis. However, an exact relationship between flow velocity determined by DUS and stent placement with and without stenosis has not been established to date.[16-18] Therefore, the hemodynamics of carotid stenting with and without stenosis in a standardized ex vivo circulation model, including peak systolic velocity (PSV), end-diastolic velocity (EDV), and compliance, were investigated by means of DUS.

METHODS

Arteries. Ovine common carotid arteries were harvested at a local slaughterhouse and prepared by dissecting them from all surrounding periadventitial connective tissue. Side branches were ligated with standard suture material Vicryl 4-0 (Ethicon Inc, Somerville, NJ). The arteries were stored at 4°C in a sterile 0.9% saline solution and used in a time window between 6 and 24 hours.

The compliance behavior of human and ovine artery is similar. Fig 1 compares the compliance of a human superficial femoral artery to that of an ovine common carotid artery at physiological pressure.

Artificial circulation system. After preparation, arteries were mounted in an artificial circulation system[19,20] with nearly physiologic hemodynamic parameters: pressure 140/90 mm Hg, heart rate 110 bpm, flow volume 300 mL/min. The perfusion fluid was a particulate suspension, which mimics the viscous characteristics of blood[19,20] and also serves as a target for ultrasound reflection. The flow was adapted to the morphologic situation of the carotid bifurcation by using a collateral circulation with adjustable resistance to imitate external carotid blood flow.

Table. Configuration and number of measurements of ovine common carotid artery

Configuration	*No.*
Native without stent	12
Native with stent	12
Moderate stenosis without stent	6
Moderate stenosis with stent	6
Severe stenosis without stent	6
Severe stenosis with stent	6

Compliance measurement. The longitudinal compliance profile of the arteries was measured under pulsatile conditions by means of a laser scan micrometer (Keyence LS-5001; Keyence Corporation, Osaka, Japan) and a Statham transducer (Statham, Cleveland, Ohio) along 14 cm with the stent, when present, in the middle section. During measurement, the artery was moistened with 0.9% saline. Each measured point was scanned in three rotational planes to determine the circumferential compliance. At each point, outer diameter was determined by laser, and mean systemic pressure and pressure amplitude were recorded intraluminally with a Statham transducer. The distance between the measuring points was 2 mm up to 20 mm away from the middle point of the stent area, 1 mm up to 6 mm away from the middle point, and 0.5 mm on both sides of the middle point. The circumferential compliance was calculated by using the following equation:

$$C_{circ}(d) = \frac{\Delta d}{\Delta P \cdot d}\left[\frac{\%}{100mmHg}\right]$$

where C_{circ} is circumferential compliance, Δd is the difference between maximum and minimum diameter, ΔP is the difference between the maximum and minimum pressure, and d is diastolic diameter.

Stents and stenosis. In bovine arteries, vascular stents (6 × 40 mm, sinus-Carotid-RXt; Optimed, Ettlingen, Germany) with a combined open- and closed-cell design (closed-open-closed) were applied to the central portion of the arteries via a 7F sheath (Percutaneous-introducer-set; Angiomed GmbH and Co, Medizintechnik KG Karlsruhe, Germany) from the proximal end of the circulation system. This stent exerts a radial force of approximately 1.2 N to 1.3 N at the ends with the closed cells. Plastic tubes simulating the stenosis were placed in the middle of the applied stent. Six arterial configurations with three degrees of stenosis (0%, 57.8% ± 1.6%, and 76.4% ± 1.1%) were examined. Each degree of stenosis was examined with and without stent. All together, 48 configurations were measured (Table). Measurement accuracy was assessed by measuring one configuration twice. The double measurement produced identical results. Stenosis was induced in the middle section of the mounted artery by inserting a plastic tube with a

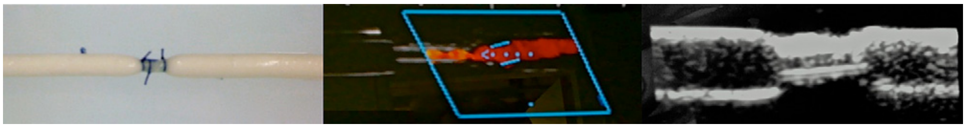

Fig 2. **A,** Ovine artery with snares to fix stenosis tube. **B,** Flow velocity measurement by duplex ultrasound (DUS), proximal to stenosis tube. **C,** B-mode ultrasound image of stenosis tube.

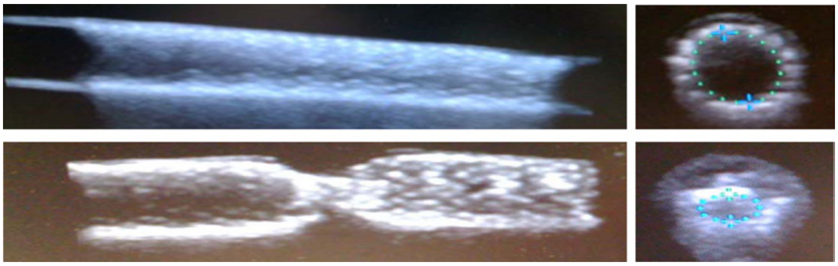

Fig 3. Ultrasound measurement of vessel diameter with perfused lumen; **A,** without stenosis; **B,** with moderate stenosis.

defined length (10 mm) and inner diameter of 2.43 mm to produce the "moderate" stenosis of 57.8%. Insertion of a tube with an inner diameter of 1.53 mm produced the "severe" stenosis of 76.4% (NASCET criteria). The stenosis tubes were fixed by two snares (Fig 2, *A*).

Duplex ultrasound scanning. After compliance measurement, the artery was submerged in a basin filled with 0.9% saline where peak flow velocity and EDV were measured by DUS scanning with a 7.5-MHz probe (Philips SD 800; Philips Medical, Shelton, Conn) at the following sites: 10 mm proximal to the stenosis, in the middle of the stenosis, and 10 mm distal to the stenosis (Fig 2, *B*).

In configurations with stents in place, all measurement points were located inside the stented area. The ultrasound probe was positioned at an angle <60° to the artery. The diameter of vessels and stenosis were measured by B-mode ultrasound at a static pressure of 90 mm Hg (Fig 2, *C*).

Measurement of stenosis. The stenosis was measured by two different methods. First, a circumferential ultrasound estimation of the perfused diameter with and without a narrowing tube (Fig 3) was used to calculate the degree of stenosis by using NASCET criteria ($n = 3$ for each degree of stenosis).

Second, a negative cast of gypsum of the perfused lumen of the whole arterial segment was made ($n = 1$ for each degree of stenosis). The diameter before and in stenosis was used to calculate the degree of stenosis by the NASCET method.

Statistical analysis. Standard parameters of descriptive statistics were applied, employing SPSS 15.0 (Statistical Package for the Social Sciences; SPSS Inc, Chicago, Ill). Box plots were generated by Graph Pad Prism 4 (GraphPad Software, Inc, La Jolla, Calif). Initial values for compliance were means of measurements in three rotational planes at a particular point. Values for PSV and EDV are expressed as means ± standard deviation and were shown to be normally distributed (by using Shapiro-Wilk test). Significance was tested by using Student's *t*-test for paired values and *P* values of <.05 were considered significantly different.

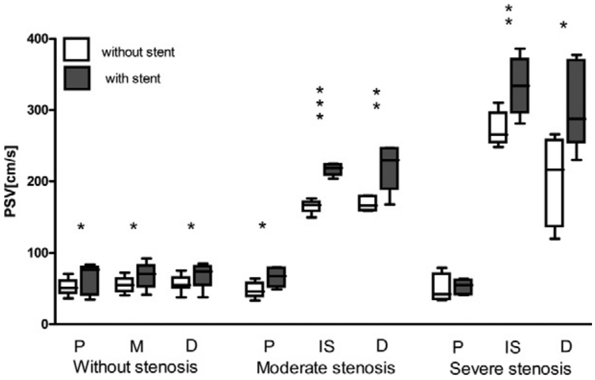

Fig 4. Influence of stenosis on peak systolic velocity (*PSV*) in ovine carotid artery with and without stent. *D*, Distal; *IS*, in stenosis; *M*, middle; *P*, proximal. $n = 12$ for without stenosis and $n = 6$ for moderate and $n = 6$ for severe stenosis. *Line* in box plot shows median. $^{*}P < .05$; $^{**}P < .01$; $^{***}P < .001$.

RESULTS

PSV. Stent insertion into an ovine carotid artery without stenosis (Fig 4) caused a significant increase from approximately 55 to 67 cm/s (22%). With moderate (57.8%) stenosis, stent insertion caused a significant increase in the PSV from 166 to 217 cm/s (31%) in stenosis.

With severe stenosis (76.4%), stent insertion caused a significant increase from 272 to 334 cm/s (23%) in the stenosis. Distal to the stenosis, the stent-induced increases in PSV were 32% with a moderate stenosis and 49% with a severe stenosis.

EDV. Stent insertion into an ovine carotid artery without stenosis (Fig 5) did not cause as many significant

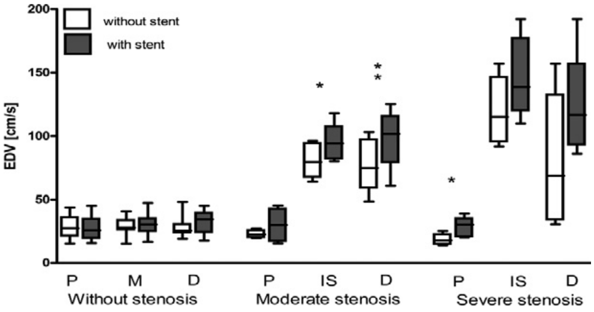

Fig 5. Influence of stenosis on end-diastolic velocity (*EDV*) in ovine carotid artery with and without stent. *D*, Distal; *IS*, in stenosis; *M*, middle; *P*, proximal. n = 12 for without stenosis and n = 6 for moderate and n = 6 for severe stenosis. *Line* in box plot shows median. $^{*}P < .05$; $^{**}P < .01$.

increases in EDV as was noticed in PSV. However, significant increases in EDV caused by stent insertion were noted in stenosis and distal to the stenosis with a moderate stenosis, 17% and 29%, respectively. A significant stent-caused increase (56%) was also noted proximal to the severe stenosis.

Dynamic compliance. Stent insertion into an ovine carotid artery without stenosis caused a threefold compliance drop compared with that measured in the unstented artery (Fig 6).

DISCUSSION

CAS has emerged as an alternative to CEA in treating carotid artery stenosis. Although the equality of CAS to CEA in preventing stroke is arguable, CAS is frequently performed and evidence is accumulating that CAS shows a higher incidence of restenosis. Several multicenter studies are now reporting an increased number of reinterventions/reoperations after CAS than after CEA.[7,10]

DUS has proven adequate in detecting and quantifying de novo stenosis of the carotid artery[21]; therefore, it should also be applicable in detecting and grading restenosis.[11] However, the impact of arterial stenting on DUS signal delivery and detection has not yet been sufficiently evaluated, and increased flow velocity and overestimation of moderate stenosis have been reported.[22,23] In these investigations, computed tomography angiography (CTA) or magnetic resonance angiography (MRA) measurements (or the endarterectomized specimen) were compared with DUS measurements. In the present investigation, DUS measurements were evaluated only with DUS, but under strictly standardized conditions.

Severe stenosis is generally defined as ≥70% constriction,[24-26] though the cutoff point has been raised to ≥80% by other groups.[22,23] A PSV value ranging from 300 to 450 cm/s has been associated with 70% to 80% stenosis. Our investigation showed a PSV of 334 cm/s for a 73.8% stenosis, which is in accordance with described clinical findings. Zhou[24] has proposed an EDV cutoff for severe stenosis of 90 cm/s, whereas Setacci[18] describes a cutoff of 140 cm/s.

A restenosis rate of 70% or higher, according to NASCET criteria both after CAS and CEA, is of clinical relevance because of the increase in stroke risk of up to 5% per year.[27-29] Our findings suggest an erroneous upgrading of moderate restenosis. Such an upgrading will, at least, lead to further diagnostics and an increase in perhaps unnecessary cost.

The results presented here show that introducing a stent into ovine carotid artery causes the PSV and EDV to increase, compared with unstented vessel areas. The stent-induced increase, which was more than 30% in the case of PSV, where moderate or severe stenosis was simulated, is particularly conspicuous. A 30% increase in PSV can easily cause a moderate stenosis to mistakenly be upgraded to a severe stenosis. This has considerable clinical impact in the form of unnecessary invasive and potentially risky treatment. Additionally, since PSV measured by DUS is increased by stent placement, attaching higher risk to restenosis after CAS than after CEA, as has been established in several studies, might not be legitimate.

In clinical practice, the PSV ratio, internal carotid artery to common carotid artery (ICA/CCA), is commonly used to reduce the influence of heart rate, a stenotic aortic valve, or hypertension on flow velocity values. With an increasing degree of stenosis, the ICA/CCA ratio may concurrently increase up to fourfold.[30,31] In our study, all interfering parameters were standardized and, thus, equal in all measurements. The ratio did not change with an increasing degree of stenosis in both the stented and unstented experimental groups. This finding introduces doubt in the value of employing the ICA/CCA ratio, since distal resistance or secondary stenosis of ICA and CCA are often unknown and the ratio is subject to many variables.

The reason for increased PSV and EDV in a stented area, detectable for almost all degrees of stenosis, is not clear. A possible explanation might be pseudoacceleration of the detected velocity caused by the stent material interfering with the ultrasound signal. Another hypothesis has been proposed by Nederkoorn et al, who postulate elastic mismatch between stented and native areas of the artery.[16] Impedance changes at the beginning and end of the stent are thought to alter hemodynamic flow patterns and lead particularly to elevated PSV levels. An approximately threefold compliance drop in the stented area has been shown in the present investigation, supporting the hypothesis that compliance may be causal to the increased PSV in stented areas.

Stent type might also influence measurement of PSV and EDV by DUS. Hussain et al and Pierce et al have shown that stents with a closed cell design lead to a higher postinterventional increase of PSV than open-cell stents.[13,14] Therefore, it might be useful to test stent hemodynamics in a standardized ex vivo circulation model. A correction factor accounting for the stent-induced elevation in PSV/EDV could thereby be determined for each stent type.

This study employed standardized conditions, which cannot be reproduced in the clinical setting. Optimal posi-

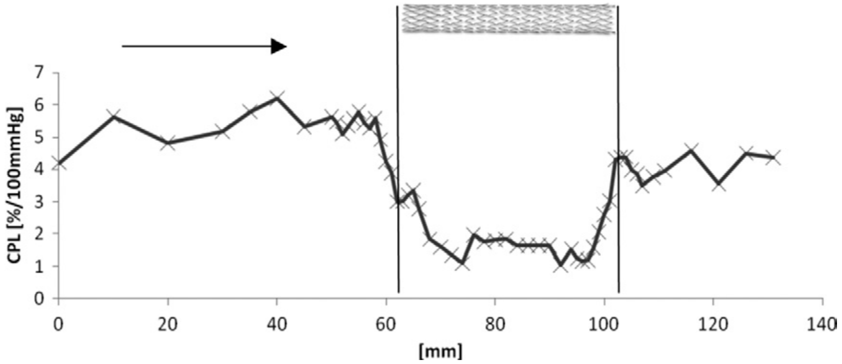

Fig 6. Mean circumferential compliance without and with stent. Stent segment from 60 mm to 100 mm, *arrow* shows flow direction, n = 4.

tioning with defined distances and angle of the ultrasound probe at each measuring point and defined circulatory parameters were part of the laboratory setting.[32] To facilitate comparison, only one stenosis length (10 mm) was investigated in this study. It should, however, be kept in mind that the stenosis length also influences flow velocity. Another factor influencing flow is the extent of calcification, which was not investigated in our model. The measurements were performed with the purpose of reducing variables such as stent length and design, circulation parameters, or stenosis morphology. Such a setting ensures reliable conditions for generating highly reproducible data to clearly compare the influence of stents on DUS parameters.

However, transferring these findings to a clinical setting is limited. Since DUS is a noninvasive, cost-effective, and well-validated method for monitoring unstented carotid arteries, it is important to establish reliable criteria for DUS monitoring of stented arteries, as well. No investigation has shown that DUS underestimates the degree of stenosis.

Although overestimation of postinterventional restenosis is a potential risk in a stented carotid artery, DUS remains a highly sensitive tool for the detection of restenosis. At present, reintervention based only on DUS does not meet international standards, which recommend additional imaging (CTA, MRA, digital subtraction angiography) before reintervention. To increase the applicability of DUS after CAS, understanding measurement disturbances, which have previously been reported, is important. This study was designed to investigate these disturbances and may be viewed as a preliminary step in establishing a correction factor for DUS use in accurately estimating stent restenosis. The object is to avoid mistakenly upgrading moderate to severe restenosis that could result in unnecessary reintervention.

Establishing DUS as a reliable follow-up method after CAS would result in reduced costs and reduced exposure of patients to radiation and contrast agents by curtailing the need for other diagnostic measures. The risk of overestimating the degree of restenosis could be reduced by introducing a correction factor for stented carotid arteries. Future investigations should be aimed at establishing correction factors under standardized laboratory conditions, taking into account blood pressure, stent length and stent design, plaque shape, and degree of calcification as well as outflow resistance. The applicability of the correction factor could then be clinically tested by comparing the degree of restenosis established with DUS, employing the appropriate correction factor, with presently accepted CTA, MRA, and/or digital subtraction angiography imaging.

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AUTHOR CONTRIBUTIONS

Conception and design: MH, PK, NA, TL
Analysis and interpretation: MH, PK, KN, TS
Data collection: MH, KN, ML, PK, TL
Writing the article: MH, KN, ML, TS
Critical revision of the article: MH, KN, PK, ML, TS, TSR, DB
Final approval of the article: MH, KN, TSR, DB
Statistical analysis: MH, TS, PK, ML, TL
Obtained funding: MH
Overall responsibility: MH

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