was documented. Aortic neck stability (AND <15%) was documented in 101 (95.3%) of patients at 2 years.

Conclusions: Helical endostaple fixation is associated with a significant reduction in AND compared to historical aortic endograft AND data. Metal helical endostaples may prevent AND as an etiology of late-term endograft failure.

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RR19.

Remodeling of Aortic Aneurysm and Aortic Neck on Mid- and Long-term Follow-up after Endovascular Repair with Suprarenal Fixation

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Objectives: To evaluate remodeling of the aortic neck, iliac arteries and aneurysm sac during follow-up after endovascular aortic aneurysm repair (EVAR) with the Zenith®AAA device.

Methods: Retrospective study of anatomic data related to characteristics of the aortic neck, iliac arteries and aneurysm sac collected during a pivotal clinical study of the Zenith device. 158 patients were followed for 5 years.

Results: Changes in aortic neck diameter below the renal arteries, aortic aneurysm sac diameter and iliac arteries diameter at distal attachment site for up to 5 years are shown in the Table. Multivariate analysis of all anatomic aortic aneurysm characteristics for re-intervention identified only the angle between the immediate suprarenal neck and immediate infrarenal neck as an independent risk factor for re-intervention with an Odds ration of 1.031 (CI 95%: 1.001-1.062). The mean value of the angle was 10.1±12°.

Conclusions: Expansion of the aortic neck post EVAR occurs mainly between 24-36 months while aortic aneurysm sac regression occurs more obviously at 1-12 months. Iliac arteries at the landing zone expand more rapidly in the first postoperative year. Long-term surveillance of EVAR-patients is essential to avoid late complications after aortic remodeling.

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RR20.

Inflammatory Response following Endovascular Treatment of Abdominal Aortic Aneurysm: Impact of Endograft Type

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Objectives: The aim of this prospective study was to evaluate the impact of endograft type on the inflammatory response after elective endovascular repair (EVAR) of abdominal aortic aneurysms (AAAs).

Methods: From January 2011 to November 2011, 100 consecutive patients underwent elective AAA endovascular repair. Fever, white blood cells, platelets, serum concentrations of cytokines (interleukin 6, 8 and 10, TNFa), were measured preoperatively, 24 and 48 hours postoperatively. Thirteen patients were excluded from the analysis (4 due to cancer, 3 with autoimmune disease, 2 because of recent infection, 2 patients receiving chronic antinflammatory medication and 2 because of surgery two months prior to enrollment). Patients were divided into 4 groups according to the endograft used: group A, n = 28 (Anaconda, Sulzer Vascutek), group B, n = 26 (Zenith, Cook Inc.),

Table. Analysis of aneurysm remodeling over time (mean ± SD)

	Pre	*Post*	*30 days*	*6 months*	*12 months*	*24 months*	*3 years*	*4 years*	*5 years*
Neck diameter (mm)	23.6 (±0.3)	27.8 (±0.3)	28.3 (±0.3)	28.8 (±0.3)	28.9 (±0.3)	29.1 (±0.3)	30.3 (±0.3)	30.7 (±0.3)	31.0 (±0.3)
Change to previous measurement		*4.3 (±0.2)	0.5 (±0.2)	0.5 (±0.2)	0.1 (±0.2)	0.3 (±0.2)	*1.1 (±0.3)	0.4 (±0.3)	0.3 (±0.3)
Aneurysm sac diameter (mm)	54.2 (±0.7)	57.2 (±0.7)	57.3 (±0.7)	52.4 (±0.7)	49.6 (±0.7)	47.0 (±0.7)	48.0 (±0.8)	47.1 (±0.7)	46.8 (±0.8)
Change to previous measurement		*3.0 (±0.6)	0.1 (±0.6)	*−4.9 (±0.6)	* −2.8 (±0.6)	*−2.6 (±0.6)	1.0 (±0.7)	−0.9 (±0.8)	-0.2 (±0.7)
Iliac-right (mm)	14.4 (±0.3)	16.3 (±0.3)	17.0 (±0.3)	17.8 (±0.3)	17.9 (±0.3)	18.0 (±0.3)	18.3 (±0.3)	18.4 (±0.3)	18.7 (±0.3)
Change to previous measurement		*1.9 (±0.2)	0.7 (±0.2)	0.8 (±0.2)	0.05 (±0.2)	0.1 (±0.2)	0.3 (±0.3)	0.1 (±0.3)	0.3 (±0.3)
Iliac-left (mm)	14.5 (±0.2)	15.8 (±0.2)	16.7 (±0.2)	17.6 (±0.2)	17.8 (±0.2)	18.0 (±0.2)	18.5 (±0.3)	18.6 (±0.2)	18.9 (±0.3)
Change to previous measurement		*1.4 (±0.2)	*0.8 (±0.2)	0.9 (±0.2)	0.1 (±0.2)	0.2 (±0.2)	0.6 (±0.2)	0.04 (±0.2)	0.4 (±0.2)

Changes 30-day- 12-month: neck diameter 0.6 (±0.2), Aneurysm sac diameter: *−7.7(±0.6), iliac right: *0.9(±0.2), iliac left: *1.1(±0.2) *Indicates $P<.0001$

group C, n = 23 (Excluder, W.L.Gore) and group D, n=10 Endurant (Medtronic, Inc.).

Results: Epidemiological characteristics, atherosclerotic risk factors, type of anesthesia, mean blood loss during surgery and baseline serum levels of cytokines did not differ among the patients of the four groups. Mean temperature was more pronounced postoperatively in group A. Serum levels of IL-6, and IL-10 were significantly higher 24 and 48 hours postoperatively compared to preoperative levels in all groups. Patients in Group C presented the smallest increase in levels of serum IL-6, and IL-10, 24 and 48 hours postoperatively. Mean difference in cytokines levels after aneurysm exclusion was higher for group A versus C ($P < .01$) compared to group A versus B ($P < .05$). Increased inflammatory response was associated with prolonged hospital stay.

Conclusions: Endograft type appears to influence the inflammatory response following EVAR. The impact of postimplantation inflammatory response in clinical outcomes requires further investigation.

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RR21.

Inflammatory Mediators and Cerebral Embolism in Carotid Stenting: New Markers of Risk

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Objectives: Cerebral embolism is a feared complication of carotid artery stenting (CAS) and might be associated with specific morphological patterns, however serological predictors of risk have been scarcely investigated.

Methods: Consecutive patients with carotid artery stenosis undergoing filter-protected CAS were preoperatively evaluated to identify unstable plaque at duplex ultrasound, complicated aortic plaque at trans-esophageal echocardiography and inflammatory status with high sensitivity C-reactive proteins (hs-CRP) and serum amyloid-A protein (SSA) serum levels. Aortic arch type, carotid tortuosity, and complexity of the procedure were considered. Cerebral embolism was evaluated by comparing number, volume and side of preoperative and postoperative cerebral lesions at diffusion weight resonance magnetic imaging (DW-RMI) and through light and scanning electron microscopy analysis of cerebral protection filters obtained from CAS.

Results: Twenty consecutive patients were submitted to CAS with no complications. At least 1 asymptomatic cerebral lesion on DW-MRI was present in 18 (90%) patients. Female gender was associated with a higher number of cerebral lesions (18.2±10.9 vs. 8.3±8.8 $P = .03$). Plaque morphology, supraaortic vessels anatomy and procedure complexity did not correlate with number or volume of new cerebral lesions at DW-RMI. The presence of complicated aortic plaque was associated with higher volume of contralateral cerebral lesions (2350±2593 vs. 636±632 mm3 $P = .02$). Hs-CRP >5mg/l and SAA >10 mg/l were significantly associated with a higher number of cerebral lesions (16.2 ± 10.7 vs. 4.3±3.4 $P = .02$, and 14.8 ± 10.3 vs. 2.8 ± 3.4 $P = .006$, respectively). Hs-CRP>5 and SAA>10 mg/l also correlated with greater surface involvement by embolic materials in the protection filters at microscopic analysis (37.0 ± 5.7 vs. 26.9 ± 2.5 $P = .004$).

Conclusions: Inflammatory status is associated with higher embolic risk during CAS independent from morphological and technical aspects of the procedure.

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RR22.

Magnetic Resonance Imaging for Identifying Vulnerable Carotid Plaques

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Objectives: Carotid Magnetic Resonance Imaging (MRI) may be a usefull tool to characterize carotid plaque vulnerability, but large studies are still lacking. The purpose of this study is to assess carotid MRI features of vulnerable plaque in a large study and assess changes in carotid plaque morphology with time since neurologic event.

Methods: We included 161 patients with carotid plaque. All underwent a carotid MRI using 3T High Resolution MR sequences. Stenosis degree, plaque thickness, plaque type (lipidic, fibrotic, calcified), Intra-plaque hemorrhage (IPH), fibrous cap rutpure (FCR) and gadolinium enhancement (GE) were assessed. Plaque type was classified on the basis of the predominant component of the plaque. IPH, FCR and GE were classified as absent or present.

Results: 7 patients were excluded because of poor image quality. In the 154 remaining patients, 52 were symptomatic (41 strokes, 7 transient ischemic attacks and 4 amaurosis in the last 6 month) and 102 asymptomatic. IPH (39 vs 16%; $P = .002$), FCR (30 vs 9%; $P < .0001$), GE (77 vs 55%; $P = .014$) were significantly higher in symptomatic versus asymptomatic plaque. No difference were observed for stenosis degree or plaque thickness. Plaques with exten-