visual loss (1%) and 1 patient (0.5%) had hyperperfusion syndrome. The 2 (2%) non-stroke related deaths were from cardio-pulmonary events; the sole (0.5%) neurological death was due to intracranial hemorrhage.

### 812-5

**Angiographic and Pathologic Studies of Late Vascular Responses to Successful Carotid Artery Stenting**

**Keita Oshikiri**, Hiroyoshi Yokoi, Katsumi Inoue, Takashi Kimura, Hideyuki Notsake, Masakuni Nishiyama, Kagawa Memorial Hospital, Kitakyushu, Japan.

**Background:** There was little angiographic follow-up data and pathologic studies for patients receiving stents at extracranial carotid arteries. The purpose of this study was to assess the long-term efficacy of carotid artery stenting (CAS) angiographically and histopathologically.

**Methods:** Out of 29 patients with 36 lesions undergoing 6 months follow-up angiography, 1-2 years follow-up angiography was available in 14 patients with 16 lesions. Quantitative angiographic analysis was performed. Minimal luminal diameter (MLD), interpolated reference diameter, and % diameter stenosis were measured. Stent sites were divided into 10 segments. At each segments, minimal diameter and mean diameter were measured. Histopathologic examination was performed in patients undergoing antemortem CAS. Temporal changes in histologic pattern between 8 months and 3 years were examined.

**Results:** At stenotic segments greater than 50% before CAS, neointimal hyperplasia became greater than other segments at 6 months. Follow-up angiography of 19 lesions at 6 months and 1-2 years revealed a decrease in MLD from 4.41±0.55 mm immediately after stent implantation to 3.30±0.39 mm at 6 months, but no further decrease in diameter after 6 months. Late improvement in luminal diameter was observed after 6 months. (3.45±0.39 mm at 6 months and 3.78±0.3 mm at 1-2 years: P=0.015). Micrographs of the stenotic segment of the 6 months after CAS revealed significant proliferation of smooth muscle cells (SMCs) and abundant deposition of extracellular matrix substance chiefly composed of proteoglycans on the intimal side. In contrast, after 3 years, neointima was primarily composed of dense fibrous collagenous tissue and endothelial SMCs. Lipid core in the atheroma was covered with stable neocapillaries.

**Conclusion:** Angiographic outcomes up to 2 years after CAS were favorable. Late improvement of neointimal hyperplasia appears to occur after 6 months. Temporal changes in histlogic pattern point toward a pathologic background for the long-term efficacy of CAS.

### POSTER SESSION

**1102 Percutaneous Coronary Intervention and Inflammation**

Monday, March 18, 2002, Noon-2:00 p.m.

Georgia World Congress Center, Hall G

Presentation Hour: Noon-1:00 p.m.

#### 1102-1

**Previous Cytomegalovirus Infection and the Risk of Restenosis After a Strategy of Provisional Stenting**

**Christian Mueller**, John M. Hoggard, Heinz J. Buettner, Stephan Marsch, Helmut Roskamm, Herz-Zentrum, Bad Krozingen, Germany, University Hospital, Basel, Switzerland.

**Background:** Previous studies have shown that prior infection with cytomegalovirus (CMV) is a predictor of restenosis after stent placement.

**Methods:** We prospectively studied 78 consecutive patients scheduled for 6 months follow-up coronary angiography as part of the SPS study. Anti-CMV IgG and IgM antibodies were measured to determine whether previous exposure to CMV increased the risk of restenosis in a strategy of provisional stenting. In 79 patients (99%), the coronary angiograms before, directly after and 6 months after the intervention could be analyzed quantitatively.

**Results:** Anti-CMV IgG positive and anti-CMV IgG negative patients had similar minimal luminal diameter (MLD) in the target vessel before and directly after the intervention. After six months, however, the MLD was significantly smaller in CMV-positive as compared to CMV-negative patients (1.57±0.82 mm versus 2.00±0.83 mm, p=0.03). Net lumen gain at 6 months was significantly lower in CMV-positive patients (0.80±0.70 mm versus 1.30±0.87 mm, p<0.04), and the rate of clinically significant restenosis was significantly higher (31% versus 7%, p<0.02). In a multivariate logistic regression model, CMV-seropositivity was an independent predictor of restenosis (odds ratio 5.7 (95%CI 1.2-30.3, p=0.04)).

**Conclusion:** CMV-seropositivity is an independent predictor of restenosis following coronary intervention.

#### 1102-2

**Role of Chlamydia Pneumoniae for Restenosis After Percutaneous Coronary Intervention: The SWICA Trial**

**Willibald Mayer, Marco Corli, Thomas Onrusch, Bernd Meier, Otto M. Hess**, Cardiology, University Hospital Zurich, Zurich, Switzerland, Cardiovascular Center Bern, Bern, Switzerland.

**Background:** Recently, a reduction of restenosis rate after stenting has been reported in patients with high Chlamydia pneumoniae titer treated with the macrolide antibiotic roxithromycin. The purpose of the present study (SWICA Trial) was to evaluate the effect of clarithromycin for prevention of coronary restenosis in patients with routine percutaneous coronary intervention (PTCA with and without stenting) and nonconsequent assessment of antibody titers.

**Methods and Results:** In a randomized, double-blind, placebo-controlled single center pilot study 86 patients with coronary artery disease undergoing PTCA were randomized to receive either standard therapy (placebo group, n=42) or treatment with 2x250 mg clarithromycin for six weeks (treatment group, n=44). Primary endpoint was angiographic restenosis and secondary endpoint major adverse cardiac events (MACE). The patients underwent follow-up angiography after 3 months. Incidence of restenosis was 5% in the placebo group and 1% in the treatment group. Antibody titers were taken at randomization and at the end of the follow-up period. Age, gender, body mass index, history of myocardial infarction as well as cardiovascular risk factors were evenly distributed in the 12 groups. Diameter stenosis was similar at follow-up (33% vs. 34%, ns) in both groups. Restenosis rate (>50% diameter stenosis) was 23% in the treatment and 21% (ns) in the placebo group, respectively. MACE occurred in 18% and 29% (ns), respectively. Antibody titers for Chlamydia pneumoniae, Cytomegalovirus and Helicobacter pylori were similar in the two groups at randomization and at follow-up without any correlation to restenosis rate. Only Helicobacter pylori antibody titers decreased significantly after antibiotic therapy.

**Conclusions:** Restenosis rate after PTCA is not influenced by clarithromycin treatment. Seropositivity for Chlamydia pneumoniae, Cytomegalovirus and Helicobacter pylori was not associated with an increased risk of restenosis after percutaneous coronary interventions. Thus, a major pathogenic role of Chlamydia pneumoniae for restenosis after percutaneous coronary intervention appears unlikely.