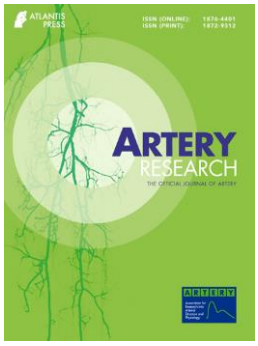

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4.2: WITHDRAWAL OF STATINS THERAPY IN PATIENTS AFTER CAROTID ENDARTERECTOMY ASSOCIATED WITH INCREASING RISK OF SIGNIFICANT RESTENOSIS

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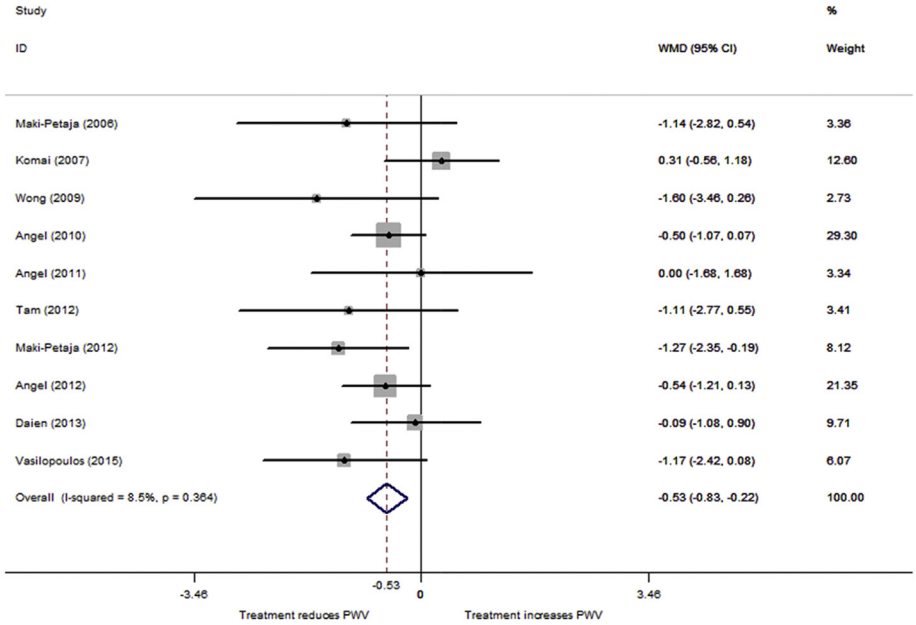

Conclusions: The balance of evidence suggests that TNF-antagonists may have a beneficial effect on arterial stiffness in RA patients. Given the predictive role of aortic stiffness for adverse cardiovascular outcomes, TNF-antagonists might confer reduction of the cardiovascular risk of these patients beyond their anti-inflammatory effect. However, larger longitudinal studies are warranted to confirm recent findings.

4.2
WITHDRAWAL OF STATINS THERAPY IN PATIENTS AFTER CAROTID ENDARTERECTOMY ASSOCIATED WITH INCREASING RISK OF SIGNIFICANT RESTENOSIS

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Background: The benefit of carotid revascularization is decreased by the occurrence of restenosis at the site of surgery, which is associated with a modestly increased risk of stroke. Preventing restenosis plays pivotal role in the overall treatment and prevention of stroke in patients with carotid artery disease.
Purpose: To evaluate influence of discontinuing of statins therapy on occurrence of restenosis in patients after carotid endarterectomy.
Methods: We studied 240 patients after carotid endarterectomy, mean age — 64.4±6.8 years. All the patients were divided into two groups: 1 group comprised 124 patients, who had taken atorvastatin in dose 10-40 mg daily and 2 group — 116 patients who discontinued statins therapy due complication 3 %, poor tolerance 9% or personal reluctance 88%. All the patients also underwent serial standardized ultrasound examination on 1, 3, 6, 12 month during first year after operation and then annually. Mean observation time was 5.6±2.1 years. Significant restenosis carotid artery (more than 70%) was established by standard Doppler velocity criteria.
Results: The significant restenosis of internal carotid artery was found in 2,4% patients with statins therapy and in 7,8% patients without statins. Statins withdrawal increased the incidence of late significant restenosis of internal carotid artery (odd ratio: 3.393 95% confidence interval: 0.895—12.857). Patients with withdrawal of statins had higher wall thickness: 4.3±0.8 against 2.9±0.9 (#1088 p<0,05).
Conclusion: Withdrawal of statins therapy in patients after carotid endarterectomy associated with increasing risk of significant restenosis carotid artery.

4.3
ELECTRONIC CIGARETTE SMOKING INCREASES AORTIC STIFFNESS IN YOUNG SMOKERS

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Purpose/Background/Objectives: Smoking increases aortic stiffness which is an important predictor of cardiovascular risk. Electronic cigarettes (EC) simulate tobacco cigarette (TC) and have been advocated as a less harmful alternative. We investigated the acute effect of EC smoking on aortic stiffness compared to the effect of TC smoking.
Methods: We studied 24 healthy smokers (mean age 30±8 years, 13 females), who were free of risk factors X from smoking. Each participant visited our unit on four separate occasions (96 in total) and smoked: a) TC over 5 minutes b) EC over 5 minutes c) EC for a period of 30 minutes. During the sham procedure, participants did not smoke anything. Carotid-femoral pulse wave velocity (PWV) was used to assess aortic stiffness.
Results: Both TC and EC smoking increased systolic and diastolic BP, and the differences in changes of BP responses between the two smoking forms were not significant. Compared to TC, EC5 min smoking resulted in a less potent PWV increase throughout the study (F=4.425, P=0.005). On the other hand, EC30min resulted in a PWV increase similar to that of TC smoking throughout the study period (F=0.268, P=0.615). EC30 min smoking resulted in a more potent effect on PWV compared to EC5 min smoking (F=3.167, P=0.030).
Conclusions: EC over 30 minutes induces an unfavorable effect on aortic stiffness similar to TC smoking. The influence of EC smoking over 5 minutes on aortic stiffness is not as prompt and is less potent compared to the effect of TC smoking.

4.4
ARTERIAL PHENOTYPE MODULATION AND REGULATION OF VASCULAR FIBROSIS IN MICE BY CONDITIONAL INACTIVATION OF INTEGRIN AV SUBUNIT IN VASCULAR SMOOTH MUSCLE CELLS

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Integrin α_v functions as a receptor for adhesion proteins and is expressed at high density in vascular smooth muscle cells (VSMC)[1,2,3,4,5] whose phenotypic modulation plays a crucial role in arterial ageing and atherosclerosis[6,7].