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$P = 0.035$). By contrast, cAlx, as well as none of central and peripheral BP-derived parameters were significantly associated with cerebral lesion growth in univariate analysis. In multivariable regression logistic model, CF-PWV predicted cerebral lesion growth with an odds ratio of 1.43 [1.00–2.04], independently of age, and peripheral pulse pressure.

Conclusions: Increased aortic stiffness is independently associated with cerebral lesion growth in patients with acute ischemic stroke. Its deleterious effect is more important than that of BP.

4.1

EFFECT OF ALISKIREN ON VASCULAR REMODELING IN SMALL RETINAL CIRCULATION

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Background: In hypertension changes in small arterial structure are characterized by an increased wall-to-lumen ratio (WLR). These adaptive processes are modulated by the renin angiotensin system. It is unclear whether direct renin inhibitors exert protective effects on small arteries in hypertensive patients.

Methods: In this double-blind, randomized, placebo-controlled study (www.clinicaltrials.gov: NCT01318395) 114 patients with primary hypertension were after 4 weeks of standardized open-label treatment with valsartan 320 mg (run-in phase) randomized to additional therapy with either placebo or aliskiren 300 mg for 8 weeks. Parameter of arteriolar remodeling was WLR of retinal arterioles (80–140 μ m) assessed non-invasively and in vivo by scanning laser Doppler flowmetry (Heidelberg Engineering, Germany). In addition, pulse wave analysis (SphygmoCor™, AtCor Medical, Australia) and pulse pressure (PP) amplification were determined.

Results: In the whole study population no clear effect of additional therapy with aliskiren on vascular parameters was documented. When analyses were restricted to patients with vascular remodeling, defined by median of WLR > 0.3326 ($n = 57$), WLR was reduced after 8 weeks by the treatment with aliskiren compared to placebo (-0.044 ± 0.07 versus 0.0043 ± 0.07 , $p = 0.015$). Consistently, after 8 weeks of on-top treatment with aliskiren there was an improvement of PP amplification compared to placebo (0.025 ± 0.07 versus -0.034 ± 0.08 , $p = 0.013$), indicative of less stiff arteries in the peripheral circulation.

Conclusion: Thus, our data indicate that treatment with aliskiren, given on top of valsartan therapy, improves altered vascular remodeling in hypertensive patients.

4.2

THE INHOMOGENEITY OF DIASTOLIC-SYSTOLIC RISE TIME OF THE DISTENSION WAVEFORM DISTRIBUTION IN THE COMMON CAROTID ARTERY IS ASSOCIATED WITH LIPID PRESENCE OF DISTAL PLAQUES

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Objectives: Diastolic-systolic rise time characteristics of the distension waveform distribution, i.e., mean and inhomogeneity, might be modified by wave reflections from distal plaques. The aim of this study is to investigate the association between rise time characteristics for the common carotid artery (CCA) and composition of distal plaques.

Methods: Longitudinal B-mode ultrasound (US) registrations of the CCA of 129 patients (age 69 ± 9) were performed with a Philips IU22 scanner. All patients had a plaque in the ipsilateral bifurcation and recently experienced a cerebrovascular accident. Distension waveforms were extracted by edge tracking and the diastolic-systolic rise time and its inhomogeneity, defined as standard deviation of systolic-diastolic rise time distribution for an artery segment, were derived. Plaque composition was extracted from 3T-MRI measurements ($N = 125$).

Results: 118 subjects had both an adequate MRI and US registration. 58% of the plaques had a lipid-rich necrotic core (LRNC) of which 68% had a thin fibrous cap (FC). Lipids were demonstrated in the proximal part in 51 plaques (43%). Mean CCA diastolic-systolic rise time (162 ± 26 ms) did not vary with plaque composition (Student t-test, p -value > 0.2). The inhomogeneity, however, was significantly lower for vulnerable distal plaques (mean difference LRNC: 8ms, FC: 11ms, Student t-test, p -value < 0.02) and, more specifically, when lipids were present in the proximal part of the plaque (mean difference 12ms, Student t-test, p -value < 0.001).

Conclusion: Diastolic-systolic rise time inhomogeneity of CCA distension is associated with the lipid presence of distal plaques.

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4.3

THE EFFECT OF GLYCAEMIC STATE TRANSITION ON ACCELERATED AORTIC STIFFENING: A LONGITUDINAL STUDY IN THE WHITEHALL II COHORT

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In 4,759 participants from the Whitehall II study, we examined the impact of glycaemic history on aortic stiffening. Assessment of aortic stiffness by carotid-femoral pulse wave velocity (PWV) was performed twice with a 4 year interval (2007–2009 and 2012–2013). At the first aortic stiffness assessment and 5 years earlier (2002–2004 and 2007–2009 respectively), participants were categorised into 3 groups based on measurements of fasting plasma glucose (FPG), 2-hour plasma glucose (2hPG), and HbA_{1c}: normoglycaemia, dysglycaemia and type 2 diabetes. The impact of 5-year glycaemic state transition on PWV and PWV changes was analysed by mixed effect models adjusting for relevant confounders. In participants who had normoglycaemia on FPG, 2hPG and HbA_{1c} on both examinations, PWV was 8.3 m/s at baseline and increased by 0.4 m/s during 4 years. Those who progressed to dysglycaemia had a 0.3 m/s (95% CI: 0.1;0.5) steeper increase in PWV compared with stable normoglycaemia, whereas those who progressed to diabetes did not have a statistically significantly steeper increase in PWV (0.1 m/s (95% CI: -0.4; 0.6)). Participants with diabetes at both examinations had a markedly larger increase in PWV of 0.6 m/s (95% CI: 0.3;0.9) compared to participants with stable normoglycaemia. For other glycaemic state combinations there was a tendency towards a steeper increase in PWV compared with stable normoglycaemia, however not statistically significant. These results indicate that people with diabetes or deteriorating dysglycaemia, experience accelerated aortic stiffening; suggesting that prevention of dysglycaemic progression and diabetes may have a beneficial effect on the progression of aortic stiffness.

4.4

FORWARD AND BACKWARD WAVES AT THE AORTIC ROOT: STEADY-STATE AND WAVE RE-REFLECTION CONSIDERATIONS

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Background: The assumption of steady-state oscillation is often overlooked when arterial pressure and flow waveforms are decomposed into backward (P_b) and forward waves (P_f). This has led to various misinterpretations including a significant reflection-free time during early-systole and attribution of the P_f to solely a product of left ventricular contraction and proximal aortic properties.