B001
HIGH SODIUM DIET INDUCES STRUCTURAL VASCULAR CHANGES IN RATS WITHOUT RAISING BLOOD PRESSURE
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Long-term high sodium diet of individuals and experimental animals has been associated with increased large artery stiffness and vascular hyperreactivity. To investigate the structural vascular basis of these findings, adult male Sprague-Dawley rats were fed 2% NaCl diet for 3 and 6 months (N=15 and 14); rats fed 0.7% NaCl diet were controls (N=23). Then, the mesenteric circulation of rats was perfusion-fixed at maximal dilatation and 50–60 mmHg pressure. Morphometric measurements of 2nd order arterioles (250–350 μm OD) and of arterioles (<100 μm OD) were performed by light microscopy. Electron microscopy was used for volume density measurements of medial components of arteries. Monthly tail SBPs and directly measured MAPs at the end of the study in awake salt-fed and control rats were the same. Compared to controls, there was increased lumen diameter (+7%, p<0.01, 2-factor ANOVA), unchanged medial wall area and reduced wall-to-lumen ratio (W/L) (−7%, p<0.01) of 2nd order arteries (eutrophic outward remodeling) in sodium-fed rats, with relative increase in collagen content (14 vs. 10% at 3 months, p<0.05). In contrast, the W/L of arterioles in salt-fed rats was increased (+8%, p<0.01). The findings suggest increased flow-dependent dilatation with increased transmural stress and sclerosis of mesenteric arteries and autoregulatory constriction of mesenteric arterioles in salt-fed rats, and may explain increased large artery stiffness and vascular hyperreactivity during chronic salt feeding.

Key Words: Vascular remodeling; mesenteric arteries; wall-to-lumen ratio; morphometry

B002
HYPERTENSIVE RETINOPATHY: THE IMPORTANCE OF OPHTHALMOSCOPY IN THE ASSESSMENT OF TARGET-ORGAN DAMAGE (TOD)
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The presence of target-organ damage (TOD) should be considered in treatment decision making. The detection of renal or cardiac damage, and documentation of cardiovascular risk factors, is done more frequently than the noting of retinopathic abnormalities. A possible reason for the omission of retinopathy in TOD assessment is that previous retinopathy classifications have been confusing. I propose that funduscopists employ a simplified two grade classification. Grade I (arteriolar narrowing and focal constriction) denotes TOD and should be considered a factor in the therapeutic decision making process. The finding of Grade II (hemorrhages and exudates with or without disc edema) confers even greater risk and calls for even more immediate and intensive antihypertensive intervention. The lack of funduscopic abnormalities in patients with an elevated blood pressure may be explained by the “white-coat” hypertensive response, recent onset hypertension, or poor ophthalmoscopic technique. Although an assessment of all target organs is recommended for the complete evaluation and treatment of hypertensive patients, fundoscopy is a readily available technique for the immediate identification of TOD.

Key Words: Target-organ damage (TOD); retinopathy; fundoscopy

B003
DIFFERENCES IN LARGE AND SMALL ARTERY RESPONSE TO ACUTE INHIBITION OF NITRIC OXIDE SYNTHASE IN HUMAN SUBJECTS
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Vascular tone is influenced by endothelial function mediated largely through nitric oxide (NO) release. To evaluate the influence of basal NO release on arterial tone L-NAME (1.5 to 5.0 mg/kg) was infused in 7 young normal volunteers and observations made over 6 hours. Arterial tone was evaluated by: 1) pulse wave analysis utilizing a modified Windkessel model allowing separate calculation of large artery capacitive compliance (C1), small artery reflective compliance (C2), and systemic vascular resistance (SVR); 2) brachial artery compliance (BC) by echo-tracking ultrasound; 3) carotid to femoral pulse wave velocity (PWV); and 4) brachial artery flow-mediated vasodilation (FMV).

Systolic, diastolic, and mean blood pressure (BP) rose modestly after L-NAME (114 to 125, P<.05; 66 to 76, P<.01; 81 to 92 mmHg, P<.01). Heart rate (HR) tended to fall (63 to 57 bpm, P=NS). C1 was unchanged 16.4 to 17.0 ml/mmHg × 10, and PWV 8.32 to 8.65 m/s, BC (5.1 × 10−3 to 6.0 × 10−3 mm²/mmHg, P=NS) and FMV 5.52 to 5.15% also did not change. However, SVR rose 1208 to 1490 dynes · sec · cm⁻5, P<.01 and C2 fell 10.1 to 6.5 ml/mmHg × 100 P<.001.

Thus NO synthase inhibition exhibits its effect predominantly on the small arteries (reflecting sites and arterioles) that are thin-walled and not on the conduit arteries in which NO release by the endothelium may not penetrate the wall.

Key Words: Nitric oxide; arterial compliance; systemic resistance

B004
WHITE-COAT HYPERTENSION AND TARGET-ORGAN DAMAGE
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The relationship between arterial hypertension cardiovascular and cerebrovascular events are well-known as well as to target-organ damage. It is not yet definitively clear whether white-coat hypertension (WC-HT) constitutes a risk factor.

We have studied the relationship between WC-HT and target-organ damage in 60 subjects (32 M, 28 F; mean age