the development of LVH in hypertensive patients, either directly or through the effects of BP.

10:45

710-2 Association of DD Genotype of Angiotensin Converting Enzyme With Hypertension

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Angiotensin converting enzyme (ACE) gene polymorphism has been associated with increased levels of the enzyme as well as with acute myocardial infarction and other cardiovascular syndromes. However, the data on the association of DD genotype with hypertension are contradictory. This could be due to the multifactorial etiology of hypertension, sampling variation and incomplete adjustment for confounders.

To evaluate the relationship of ACE polymorphism to hypertension, we studied 209 patients with essential hypertension requiring drug therapy and 96 age matched normotensive controls.

Among the 209 patients with hypertension 78 (37.3%) had DD genotype, 102 (48.8%) ID and 29 (13.9%) II genotype. Among the 96 normotensive controls, the corresponding frequencies in the control group were 28 (29.2%) DD, 46 (47.9%) ID and 22 (22.9%) II ($p = 0.035$ for DD vs. II). The allele frequency of D was 0.62 in the hypertension group and 0.53 in the control group. The unadjusted odds ratio for the association of DD with hypertension was 1.45 (95% C.I. 0.82–2.54).

Patients with hypertension were of similar age (65 ± 7 vs. 68 ± 4 year old) but had higher BMI (28.7 vs. 26.8, $p < 0.001$) than the normotensive controls.

Multiple logistic regression adjusting for sex, age and body mass index identified DD genotype (vs. II) as independently associated with hypertension (logist odds ratio 1.57, 95% confidence interval 1.06, 2.36, $p = 0.02$).

In conclusion, we observed a positive association of ACE DD genotype with hypertension. This association was independent of age, sex and body mass index in this population.

11:00

710-3 Restoration of Flow-Dependent Coronary Dilation by Converting-Enzyme Inhibition (Perindoprilat) in Hypertensive Patients Enhances Maximal Coronary Blood Flow Produced by Intracoronary Papaverine

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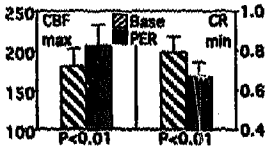
Maximal coronary blood flow (CBFmax) depends both on the maximal area of the coronary microcirculation and on the epicardial coronary artery (CA) dimensions. In hypertensive patients (HTP) flow-dependent CA dilation is abolished. To assess the influence of CA dimensions on CBFmax and minimal coronary resistance (CRmin), proximal and distal left anterior descending (pLAD and dLAD) CA diameters were determined by quantitative angiography, and flow velocity (FV) was measured in dLAD using intracoronary Doppler. Measures of CBFmax ($\pi D^2/4 \times FV$ [mL/min]) and of CRmin (mean aortic pressure/CBF [mmHg/mL/min]) following 10 mg papaverine (PAP) injection into dLAD were made before and after 1 mg i.v. perindoprilat (PER) in 10 untreated HTP with angiographically normal CA and no other risk factors. Heart rate and aortic pressures were not modified by PER.



Results (mean ± SEM) show that after PER, CBFmax was increased and CRmin was reduced. Diameter of pLAD at peak flow was increased from 3.75 ± 0.19 before PER to 4.21 ± 0.22 mm after PER ($P < 0.001$) when dLAD diameter exposed to PAP did not vary significantly (3.02 ± 0.13 and 3.08 ± 0.15 mm).

Conclusion: This study demonstrates that the increase in epicardial CA diameters participates significantly to the CR. Thus, the restoration of flow-dependent CA dilation by PER in HTP may improve the ability of coronary circulation to deliver its maximal myocardial blood flow.

11:15

710-4 Carotid Anatomy and Stiffness Is Not Affected by Hypercholesterolemia in Uncomplicated Essential Hypertension

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To assess the combined effects of hypertension and hypercholesterolemia (HC) on carotid anatomy and stiffness, we studied 79 uncomplicated essential hypertensives (EH; 56% males; age = 53 ± 12 years) with cholesterol plasma levels < 250 mg/dl (206 ± 27 mg/dl), 34 with cholesterol ≥ 250 (EH + HC; 56% males; age = 59 ± 14 years; cholesterol = 284 ± 31 mg/dl) and 32 normocholesterolemic normotensives (N; 71% males; age = 50 ± 17 years; cholesterol = 202 ± 29 mg/dl). Carotid ultrasonography was performed to measure intima-media thickness (I-MT, mm), diameter (CAD, mm), relative wall thickness (RWT) and intima-media cross sectional area index (CAI, mm^2/m^2) of the common carotid artery, and the presence of plaques. Carotid pressure waveforms were noninvasively recorded by applanation tonometry to obtain carotid Young's elastic modulus (E, $dynes/cm^2 \times mm \times 10^{-6}$) and augmentation index (AI, %), an index that expresses the relative contribution of reflected pressure waves to the carotid systolic peak pressure.

Both systolic and diastolic blood pressures (BP) were similar among EH (160 ± 19/95 ± 12 mmHg) and EH + HC (163 ± 20/92 ± 11), and significantly higher than in N group (124 ± 13/74 ± 8; both $p < 0.0001$).

	I-MT	CAD	RWT	CAI	E	Plaq %	AI (%)
N	0.69±0.16	5.7±0.6	0.24±0.05	7.5±1.9	0.87±0.35	16	9±12
EH	0.82±0.20§	5.9±0.8	0.28±0.07*	9.4±2.8§	0.96±0.49	23	17±12§
EH + HC	0.88±0.22§	6.0±0.8	0.30±0.08§	10.4±3.5§	0.86±0.49	29	18±13§

*$p < 0.05$ and §$p < 0.01$ vs N (adjusted for age, gender, BMI, cigarette smoking)

In the whole study population a multiple regression analysis showed that age, body mass index and systolic BP were independent determinants of carotid intima-media thickness ($R = 0.51$; $p < 0.0001$), while cholesterol levels, gender and smoking habit did not enter the model.

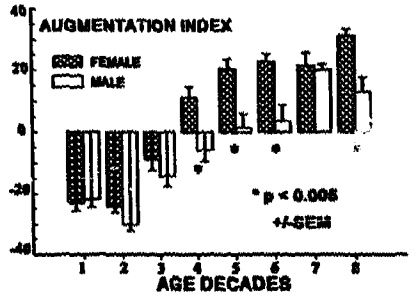
As expected, hypertension increases carotid intima-media thickness and AI. Surprisingly, hypercholesterolemia does not further augment these changes and, as indirectly suggested by AI, does not further increase aortic stiffness and pulse wave velocity. These data suggest that in uncomplicated hypertensive subjects hypercholesterolemia plays only a minor role in inducing anatomic and functional arterial modifications.

11:30

710-5 Gender Related Differences in Central Pressure Pulse Waveform

Christopher S. Hayward, Michael F. O'Rourke, Raymond P. Kelly. *Cardiology Dept St Vincent's Hospital Sydney Australia*

Despite having fewer ischemic cardiac events, women are more likely to develop cardiac failure post AMI and have an age related increase in LV mass (independent of body size) not seen in men. This study investigated the effect of age and gender on central pressure pulse contour using noninvasive carotid artery applanation tonometry. Augmentation index (AI, expressed as the difference in first (SP1) and second systolic peaks (SP2) divided by pulse pressure), an index of pulsatile afterload, was used as study endpoint. Three hundred and fifty eight healthy subjects were studied (183 female), aged 2–84 years. Subjects were divided into age decades for analysis. Females had higher AI after the first decade. This was significant from age 31 onwards $p < 0.005$ (except decade 7), see figure.



In both gender groups there was a strong age dependence ($r = 0.77$, $p < 0.001$ for females, $r = 0.66$, $p < 0.001$ for males). Males were significantly taller from age group 21–30 yrs onward. Despite the predominance of the SP2 in women, men still had higher mean blood pressures until the 6th

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