normotensive and 83 to have borderline or established hypertension. The subjects were divided into 2 groups according to the level of Doppler-derived ratio of peak early to atrial velocity (E/A): 50 with E/A > 1 (normal diastole) (ND) and 51 with E/A < 1 (impaired diastole) (ID). The 2 groups were comparable for gender, body mass index, clinical BP and heart rate (HR) while age was higher in ID (p < 0.0001). No significant difference was found in LV mass/height (LVM/HT) between the 2 groups. ID had comparable awake BP but higher nocturnal (either systolic or diastolic) BP and lower day-night BP drop (all p < 0.01) in comparison with ND. Potential determinants of E/A were analysed by a multivariate model, including age, HR, LVM/HT, awake and nocturnal BP. Age (beta coefficient = -0.49, p < 0.001), nocturnal diastolic BP (beta = -0.39, p < 0.001) and HR (beta = -0.17, p = 0.02) were independent predictors of E/A in the pooled population (multiple R = 0.68, p < 0.0001).

In conclusion, high nocturnal BP is a powerful marker of LV filling impairment and ABP monitoring provides prognostic information about subjects who progress from hypertension to diastolic dysfunction.

**966-41** Impact of Hypertension on Aortic Root Size and Prevalence of Aortic Regurgitation

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Although we previously showed similar M-mode aortic diameters in age-matched normotensive and hypertensive subjects, the entire aortic root and prevalence of aortic regurgitation have not been examined by 2-dimensional and Doppler echo. We therefore measured diameters of the aortic annulus, sinuses of Valsalva, supra-aortic and proximal ascending aorta in 110 normotensive (NL) and 110 hypertensive (HTN) subjects (matched age = 55 ± 12 years and gender = 33% women in both groups). Compared to rest and ambulatory blood pressures (BP) and measures of body size.

The groups had similar body surface area (LSA), but hypertensives had slightly higher body mass index (27 ± 4 vs 26 ± 4 kgm², p < 0.05). Rest systolic (159 ± 19 vs 124 ± 12 mmHg) and diastolic (94 ± 11 vs 74 ± 8 mmHg) and awake systolic (130 ± 9 vs 148 ± 17 mmHg) and awake diastolic (81 ± 9 vs 90 ± 12 mmHg) BP were increased in HTN. Aortic diameters at the annulus (2.4 ± 0.3 vs 2.3 ± 0.2 cm, p < 0.06) and sinuses (3.5 ± 0.4 vs 3.4 ± 0.4 cm, p < 0.08) tended to be higher in HTN whereas diameters at the supra-aortic ridge (2.9 ± 0.4 vs 2.9 ± 0.4 cm, p < 0.01) and ascending aorta (3.3 ± 0.5 vs 3.1 ± 0.3 cm, p < 0.01) were significantly increased in HTN. In both NL and HTN, aortic diameters were most strongly related to BSA. Rest systolic BP bore a significant but weak relation to diameters at the sinuses and supra-aortic ridge in NL (r = 0.28, p < 0.005 for both) which was not strengthened by comparison to ambulatory BP. Among HTN only awake ambulatory BP was related to aortic diameters (r = 0.28, p < 0.05 for sinuses, r = 0.34, p < 0.02 for supra-aortic ridge, r = 0.37, p < 0.006 for ascending aorta). Differences in aortic diameters between NL and HTN persisted in the younger half of the population but not the older half. When the entire population was divided into quartile of systolic, diastolic, pulse, and mean BPs, differences in aortic diameters were most significant when the lowest quartile was compared to the highest quartile. When the lowest diastolic was < 80 mmHg, the second highest diastolic was < 80 mmHg, and the highest diastolic was < 80 mmHg, the aortic diameters were found to be significantly increased in HTN compared to NL. These data suggest that subjects at high risk for hypertension might have an exaggerated stress-induced cardiovascular response at younger age.

**966-42** Presor Reactivity to Stress and Development of Stable Hypertension in Borderline Hypertensives

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Cardiovascular reactivity to stress may have a pathophysiological role in the development of hypertension. We studied the value of measuring the blood pressure (BP) during a standardized mental challenge (mental arithmetic) to prediction of onset of high blood pressure among 80 young patients with borderline hypertension actively followed for 10 years. Patients have been classified as normo- (NR, n=34) or hyperresponders (HR, n=46) in comparison to a control population of 20 normotensive subjects (C). After adjustment of BP drop (all p < 0.01) in comparison to expected diastolic pressure, and body mass index were found to be associated with a higher percentage of 10-year development of high blood pressure.

**966-43** Mechanical Strain-Induced Proliferation in Vascular Smooth Muscle Cells from Coronary Conductance and Resistance Arteries

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Mechanical strain has been shown to induce proliferation in neonatal vascular smooth muscle (VSM) cells. We compared the effects of strain on cultured VSM cells from epidermal and resistance coronary arteries from adult sheep. Cells were matched normotensive and hypertensive subjects, the entire aortic root and subjected to strain (60 cycles/min) using a vacuum actuated strain device. 3H-thymidine incorporation was used as a measure of DNA synthesis. Strain induced increase in DNA synthesis was 1.9 fold higher in cells obtained from resistance arteries, compared to cells obtained from the left anterior descending (LAD) coronary artery. To gain further insights into the effects of strain on coronary artery VSM cells, we studied the time course of the strain-induced increase in DNA synthesis in cultured VSM cells from the LAD. We observed an increase in 3H-thymidine incorporation in response to strain with a peak at 18 hours, identical to the time to peak effect obtained in in vitro to exogenous growth factors (PDGF and basic FGF), consistent with the continual rapid release of cellular growth factors from strained cells. Supernatant medium obtained from these cells induced a 2.01 fold increase in DNA synthesis in unstrained control coronary VSM cells. Since TGF-beta and basic FGF are both reported to be present in VSM cells, we explored the potential role of these factors in the strain induced response. Supematant from LAD cells induced an inhibitory response in mink lung epithelial cells, suggesting the presence of TGF-beta in the medium. In cultured LAD cells that had undergone multiple replications, the proliferative response was abolished by monoclonal antibodies to basic FGF. We conclude that mechanical strain is an important stimulus to proliferation in coronary VSM cells derived from both epicardial and resistance coronary arteries. In LAD cells, the increase in DNA synthesis may be mediated via release of autocrine growth factors, specifically TGF-beta and/or basic FGF.

**966-44** Conduit Coronary Artery Cross-sectional Area is Abnormal in Hypertensive Patients with Angina Pectoris and Normal Coronary Angiogram

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The mechanisms of chest pain in patients with hypertension (H) and LV hypertrophy are still debated. In the present study, we analyzed the relation between epicardial coronary artery cross-sectional area (CSA) and LV mass in 34 H patients with normal coronary angiogram; 16 pts were asymptomatic (Hs) and 18 pts presented with typical angina pectoris (Hs). Hs pts data were compared to those obtained in 17 age-matched control subjects (N). Using a computer-based quantitative coronary angiographic system, proximal diameter and derived CSA (in mm²) of the left anterior descending (LAD) and left circumflex (CX) arteries were calculated from 2 orthogonal projections over a length of 1 cm immediately beyond the bifurcation of the left main coronary artery. LV mass was calculated by M-mode echo using the methodology of the American Society of Echo corrected by the Devereux formula. Results:

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age (yrs)</th>
<th>SYS (mmHg)</th>
<th>DIA (mmHg)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NR</td>
<td>84 ± 19</td>
<td>17 ± 3</td>
<td>16 ± 8</td>
<td>20 ± 10</td>
<td>10 ± 4</td>
</tr>
<tr>
<td>Ha</td>
<td>129 ± 46*</td>
<td>22 ± 4</td>
<td>19 ± 6</td>
<td>0.20 ± 0.10</td>
<td>0.09 ± 0.49</td>
</tr>
<tr>
<td>Ca</td>
<td>139 ± 46*</td>
<td>24 ± 5</td>
<td>12 ± 6</td>
<td>0.05 ± 0.06#</td>
<td>0.53 ± 0.29#</td>
</tr>
</tbody>
</table>

CA = (LAD + CX) CSA, ILV = Indexed LV mass in g/m², P < 0.001 vs C, # P < 0.05 vs Ha, 2 h = end-diastolic [septal + posterior] wall thickness

A good linear correlation was found in Ha pts between C and 2 (r = 0.99, p = 0.0004) but not significant in in S and LV mass (r = 0.68, p = 0.04). In Ha pts, no correlation was found between C and LV wall thickness and LV mass. C was independent of sex, blood pressure and body mass index; strain and LV mass were independent of sex, blood pressure and body mass index, but no significant focal stenosis at the angiogram, epicardial coronary arteries have smaller size than in normal subjects suggesting a diffuse narrowing of the vessel. In asymptomatic hypertensive pts proximal coronary arteries