compared their age, height, body surface area(BSA), central blood pressure parameters, peripheral blood pressure parameters, differences between peripheral and central PP, heart rate(HR), left ventricular mass index(LVMI) and biochemistry data, respectively.

Multianalysis of correlation indicated that central PP of hypertensive group was significantly and positively associated with age, central systolic blood pressure, APFV, A/E ratio, arterial stiffness index ($r=0.411$, 0.893, 0.382, 0.234, $P<0.01$); whereas it negatively associated with height, weight, BSA, HR, CO ($r=-0.388$, -0.319, -0.366, -0.198,-0.217, $P<0.05\sim0.01$); moreover, it was not associated with history of hypertension, central diastolic blood pressure, LVMI, fasting blood sugar, serum creatinine, serum total cholesteral. Use central PP as dependent variable, and Age, cSBP, APFV, A/E ratio, AS index, Height, Weight, BSA, HR, CO as independent variable, the regression equation was: central PP$=0.692\times$cSBP$+0.372\times$Age$+0.112\times$APFV$-14.305\times$BSA$-0.194\times$HR$-3.350\times$A/E ratio-16.808 ($P<0.001$, $R^2=87.5\%$). In normatensive group, central PP was significantly correlated with the level of central systolic blood pressure, central diastolic blood pressure, serum total cholesteral and LDL($r=0.889$, 0.373, 0.313, 0.339, $P<0.05\sim0.001$).

Age, central systolic blood pressure, HR and BSA were the main determinants of central PP in hypertensives; and central systolic blood pressure and diastolic blood pressure were both associated with central PP in normotensives.

Key Words: Pulse Pressure, Hypertensive, Normotensiv

P-277

COMPARISON OF NICOTINE EFFECTS ON HEMODYNAMICS AND ARTERIAL STIFFNESS BETWEEN SMOKERS AND NON-SMOKERS

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Reduction in arterial compliance occurs at an early stage of cardiovascular disease. Previously we have reported that nicotine increases blood pressure and decreases arterial compliance in healthy, normotensive non-smokers. The present study compared the effects of nicotine patch on the hemodynamics and arterial compliance in 10 chronic smokers and 10 nonsmokers. All study subjects were healthy normotensives and were not on any medication. Non-smoker group consisted of 4 males and 6 females, and the mean age was 42 $\pm$ 12 years. The smoking group consisted of 3 males and 7 females, and the mean age was 34 $\pm$ 12 years. Average number of cigarettes smoked per day was 13 with a mean number of smoking years of 15. A 7 mg nicotine patch (equivalent to 10 cigarettes a day) was placed on the right upper arm, and HR, SBP, DBP, large artery elasticity index (LAEI), and small artery elasticity index (SAEI) were measured noninvasively using HDI/Pulse Wave (CR-2000) instrument from radial artery waveforms. Systemic vascular resistance (SVR) was calculated from mean BP/cardiac output. Measurements were made at pre-nicotine patch baseline, and post nicotine patch at 20 minutes intervals for a period of 2 hrs. Both groups had similar age and gender distribution. There were no significant differences in the hemodynamic variables at baseline, except smokers had a significantly higher HR (74 vs 63 bpm, $p < 0.05$). The nicotine patch significantly increased BP, SVR, and decreased large and small arterial compliance in both groups. These changes were not significantly different between groups. The percent change from pre-nicotine baseline is summarized in the table. Hemodynamic changes occurred 40 minutes earlier in smokers as compared to nonsmokers.

Conclusion: age matched, healthy normotensive smokers and nonsmokers have similar cardiovascular response to the nicotine patch. Both groups increased blood pressure, systemic vascular resistance, and arterial stiffness. The quicker response in smokers suggests a different rate of nicotine metabolism between smokers and non-smokers.

Comparison of changes (%) to nicotine patch between smoker vs non-smoker

GROUP	SBP	DBP	HR	LAEI	SAEI	SVR
SMOKER	+7.5	+7	−5	−23	−27	+9.6
NON-SMOKER	+5	+4	−1	−12.7	−22	+5

Key Words: Nicotine, Arterial Compliance,

P-278

RELATIONSHIP BETWEEN AORTIC STIFFNESS AND ALBUMIN EXCRETION RATE IN UNTREATED ESSENTIAL HYPERTENSIVE PATIENTS

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Little is known about the relationships between microalbuminuria and large-artery stiffness. The aim of our study was to evaluate, in a group of non-diabetic essential hypertensive patients with normal renal function, the relationship between albumin excretion rate (AER) and carotid-femoral pulse wave velocity (PWV), as an index of aortic stiffness.

We enrolled 70 subjects with mild-to-moderate essential hypertension, aged 42 $\pm$ 8 years, never pharmacologically treated. All patients underwent routine laboratory tests, 24-h ambulatory blood pressure monitoring, measurement of carotid-femoral PWV, by means of a computerized method, and AER.

Subjects with AER above the median value (15.3 μg/min), when compared to patients with lower levels of albuminuria, showed more elevated 24-h systolic blood pressure (135 $\pm$ 8.7 vs 128 $\pm$ 7.3 mmHg; $p = 0.001$) and higher values of carotid-femoral PWV (10.2 $\pm$ 1.7 vs. 8.9 $\pm$ 1.4 m/sec; $p = 0.002$). This latter difference remained statistically significant, even after correction by ANCOVA for 24-h systolic BP, gender, age, glycaemia and HDL cholesterol ($p = 0.01$). Univariate regression analysis disclosed a tight correlation between AER e carotid-femoral PWV ($r = 0.42$; $p = 0.0003$). This association was confirmed in a multiple regression model ($\beta = 0.35$; $p = 0.009$) in which, as independent variables, besides PWV, were added: 24-h blood pressures, age, serum glucose values, smoking status, gender and body mass index.

Our results seem to confirm that microalbuminuria may represent the early renal manifestation of a widespread vascular dysfunction, and therefore it may be considered as an integrated marker of cardiovascular risk.

Key Words: Aortic Stiffness, Arterial Hypertension, Microalbuminuria

P-279

ANOMALIES OF WAVE REFLECTION PHENOMENA IN CLINICAL STUDIES

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Pulse waveform analysis is contentious, with spirited debate between investigators who use a generalised transfer function to generate the aortic waveform from the radial pressure pulse, and those who rely on non-invasive measurement of the carotid pressure waveforms, or invasive measurement of the aortic waveform with fluid-filled catheter systems. The present report seeks to clarify this issue.

In a cohort of 999 patients attending a clinical cardiovascular outpatient service, pressure waveforms were measured non-invasively in the radial artery (8917 reports), with the ascending aortic pressure waveform