Early Hypertension Is Associated With Reduced Regional Cardiac Function, Insulin Resistance, Epicardial, and Visceral Fat

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Abstract—Mild-to-moderate hypertension is often associated with insulin resistance and visceral adiposity. Whether these metabolic abnormalities have an independent impact on regional cardiac function is not known. The goal of this study was to investigate the effects of increased blood pressure, insulin resistance, and ectopic fat accumulation on the changes in peak systolic circumferential strain. Thirty-five male subjects (age: 47±1 years; body mass index: 28.4±0.6 $kg \cdot m^{-2}$; mean±SEM) included 13 with normal blood pressure (BP: 113±5/67±2 mm Hg), 13 with prehypertension (BP: 130±1/76±2 mm Hg), and 9 newly diagnosed with essential hypertension (BP: 150±2/94±2 mm Hg) who underwent cardiac magnetic resonance tissue tagging (MRI) and MRI quantitation of abdominal visceral and epicardial fat. Glucose tolerance, on oral glucose tolerance test, and insulin resistance were assessed along with the serum lipid profile. All of the subjects had normal glucose tolerance, left- and right-ventricular volumes, and ejection fraction. Across the BP groups, left ventricular mass tended to increase, and circumferential shortening was progressively reduced at basal, midheart, and apical segments (on average, from −17.0±0.5% in normal blood pressure to −15.2±0.7% in prehypertension to −13.6±0.8% in those newly diagnosed with essential hypertension; $P=0.004$). Reduced circumferential strain was significantly associated with raised BP independent of age ($r=0.41$; $P=0.01$) and with epicardial and visceral fat, serum triglycerides, and insulin resistance independent of age and BP. In conclusion, regional left ventricular function is already reduced at the early stages of hypertension despite the normal global cardiac function. Insulin resistance, dyslipidemia, and ectopic fat accumulation are associated with reduced regional systolic function. (***Hypertension*****. 2008;51:1-7.)**

Key Words: hypertension ■ visceral fat ■ epicardial fat ■ insulin resistance ■ cardiac MRI ■ circumferential strain ■ left ventricular function

The mechanisms of cardiovascular damage in hypertension are still partially unclear; in particular, it is not known what role the metabolic changes frequently associated with high blood pressure (ie, insulin resistance, dyslipidemia, glucose intolerance, and ectopic fat accumulation) may play. Mild increases in blood pressure are recognized as a risk factor for myocardial infarction and coronary artery disease.[1] For this reason, in 2003, the Joint National Committee on High Blood Pressure identified "prehypertension" as a new category of blood pressure in adults[2]: prehypertensive (pre-HT) individuals carry a higher risk (≤3-fold) of developing hypertension and cardiovascular disease in comparison with normotensive (NT) subjects.[3]

We have shown recently that ectopic fat accumulation in visceral and epicardial areas is a characteristic feature of essential hypertension[4]; the consequences, however, for cardiac morphology and function have not been investigated. We have assessed cardiac function using MRI and tagging.[5–7] MRI provides a "1-shop" method for assessing not only cardiac morphology and function but also epicardial and visceral fat mass during the same session. MRI tagging, which directly measures intramural circumferential myocardial shortening, is a highly accurate method for direct quantification of regional systolic function, allowing one to reveal small, otherwise undetectable functional abnormalities of the myocardium.[7]

We hypothesized that the metabolic abnormalities of prehypertension and early hypertension, especially insulin resistance, may be linked with altered regional left ventricular (LV) function by affecting mechanisms involved in sustaining normal cardiac function. Thus, we set forth to investigate the early changes in heart morphology and dynamics in

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pre-HT subjects and newly diagnosed, untreated hypertensive subjects and their relationship with metabolic abnormalities.

Materials and Methods

Subjects

Thirty-five male subjects were recruited among patients attending our clinic. Inclusion criteria were as follows: no previous diagnosis of hypertension; no previous treatment with antihypertensive or antidiabetic drugs or any other drug known to affect glucose and lipid metabolism; absence of diabetes (ie, a fasting plasma glucose $<$7.0 mmol/L and a 2-hour plasma glucose concentration $<$11.1 mmol/L on a 75-g oral glucose tolerance test [OGTT]) or severe obesity (ie, a body mass index $\geq$40 kg·m^{-2}); and no history of chest pain or previous cardiovascular disease. Subjects were studied within 3 months from first visit and were divided into 3 groups according to their blood pressure (BP) values: NT, pre-HT, and newly diagnosed hypertensive (new-HT) according to the Seventh Report of the Joint National Committee.[2] The study protocol was approved by the institutional review board of the University of Pisa, and each subject gave informed written consent to participate.

Study Design

All of the subjects underwent the following: (1) a 3-hour OGTT; (2) measurement of global cardiac function and regional LV function by MRI; and (3) quantitation of abdominal subcutaneous and visceral fat and epicardial fat content by MRI. The OGTT was performed in the morning (8:00 AM) after a 10- to 12-hour overnight fast. Timed blood samples (at −15, 0, 15, 30, 60, 90, 120, 150, and 180 minutes) were collected for the measurement of plasma glucose, free fatty acids (FFAs), and insulin concentrations. Plasma glucose was measured by the glucose oxidase reaction (Beckman Glucose Analyzer). Plasma insulin, leptin, and adiponectin concentrations were measured by radioimmunoassay using specific kits (Linco Research). Plasma FFAs were measured spectrophotometrically (Wako). Serum lipid profile was determined by standard laboratory methods. Plasma norepinephrine concentration was assayed by high-performance liquid chromatography (HLC 725 apparatus) using electrochemical detection (Eurogenetics).

After a 30-minute acclimation period, BP was measured 3 times to the nearest 2 mm Hg in the sitting position using a mercury sphygmomanometer and appropriately sized cuffs. The average of 3 measurements was used to calculate systolic and diastolic BPs; mean BP was calculated as the diastolic value plus one third of the pulse pressure value.

MRI and Tagging

All of the subjects underwent a single MRI examination, inclusive of the cardiac and abdominal session. The MRI examination was performed on a whole-body MRI scanner (GE Signa CV/i 1.5T scanner; slew rate: maximum 150 mT/m per second), which operates with a 40-mT/m using a 4-channel cardiac or body coil.

Cardiac MRI acquisition was performed according to a standardized protocol. Cardiac images were obtained using breath-hold segmented gradient echo Fast Imaging Employing Steady State Acquisition, electrocardiographically triggered sequences. The echo time was 1.7 ms; repetition time was 4.0 ms; slice thickness was 8 to 10 mm with no interslice gap; field of view ranged from 320 to 380 mm; data matrix size was 256×192; trigger delay was minimum, with 8 to 12 views per segment depending on heart rate; and flip angle was 45°. Thirty cine frames were obtained both in 4-chamber and 2-chamber views to measure LV dimensions. On the 4-chamber view slice, the LV was covered from the base to apex, with a stack of slices in the true short axis. The basal short-axis slice was positioned just forward of the atrioventricular ring, and all of the subsequent breath-hold slices were acquired toward the apex. The tagging pulse sequence consisted of nonselective radiofrequency pulses separated by spatial modulation of magnetization-encoding gradients to achieve a tag separation of 7 mm.[8] Three base-to-apex short-axis sections were prescribed (basal, equatorial, and apical regions). Two sets of identical short-axis views were acquired, with the second set rotated by 90°; in this way, we imaged the 3 slices within 3 breath holds (≈20 seconds each). The number of views per phase was optimized on the basis of the patient's heart rate.

Epicardial, visceral (VF) and subcutaneous depots were measured by MRI using previously published procedures.[4,9] For epicardial fat imaging, cardiac coil and ECG triggering were used for the sequences; during the acquisition time, patients were in breath hold (10 to 12 seconds). Epicardial adipose tissue scans were obtained by fast-spin echo T1-weighted sequences with oblique axial orientation for a correct study of horizontal long axes of the heart (echo time: 42 ms; echo train length: 23; bandwidth: 62.50 KHz; slice thickness: 8 mm; slice gap: 0 mm; field of view: 38 cm; matrix: 288×224; phase field of view: 0.75; number of excitations: 1; trigger delay: minimum). For abdominal fat imaging, a sagittal localizing image was used to center transverse sections on the line through the space between L4 and L5. Thirty-two transverse, T1-weighted 256×256 images (repetition time: 135 ms; echo time: 4.2 ms; flip angle: 90°) were acquired in breath hold with a slice thickness of 5 mm with no overlap.

Data Analysis

LV contours were drawn in end-diastolic and end-systolic short-axis frames using a commercial postprocessing program (Mass Analysis, Medis) on an independent Sun-sparc Station. LV end-diastolic volume (EDV) and end-systolic volume (ESV) were measured by manual planimetry and subsequent multiplication with slice thickness. Final LV volumes were calculated by simple addition of the individual slice volumes in the stack of contiguous slices for the entire left ventricle. Indexed LV volumes were obtained by dividing EDV and ESV by the body surface area. LV mass index (grams per meter squared) was calculated by multiplying the difference between epicardial and endocardial indexed EDV by the density of myocardial tissue (1.05 g/mL). Both EDV and ESV were used to calculate LV indexed stroke volume (milliliters per meter squared) and ejection fraction. Cardiac index (liters per minute per meter squared) was obtained as the product of stroke volume and heart rate. Left ventricle was divided into 17 segments according to the American Heart Association standardized myocardial segmentation.[10] The analysis of tagged cardiac images was performed using the harmonic phase technique, obtaining a fast and accurate assessment of 2D strain fields.[11] Peak systolic midwall circumferential strain was determined in 16 segments in 3 slices (6 basal, 6 midcavity, and 4 apical slices). Peak systolic circumferential strain values are conventionally negative to express circumferential shortening (absolute value), ie, the circumferential length reduction that occurs during systole. Therefore, a less negative circumferential strain indicates reduced regional function. The sensitivity of harmonic phase to small changes in myocardial strain and its potential application in clinical cardiology have been described.[12] During the analysis of the LV parameters, operators were blinded to the patient group. We also calculated end-systolic wall stress using the Grossman formula, as follows[13]:

$$WS = 0.133 \times SP \times R/[2T \times (1 + T/2R)]$$

where SP is the peak systolic ventricular BP (in mm Hg) measured during the MRI session, 0.133 is a conversion factor to express the final results in 10^3 N/m^2, R is the inner radius, and T is the wall thickness measured during the end systole.

Visceral and subcutaneous volumes were analyzed by an ad hoc developed software,[9] as validated recently.[14] A factor of 0.92 was use to convert adipose tissue volume into adipose tissue mass.[15] Epicardial fat areas were measured using a semiautomatic program for the detection of the margins of adipose tissue around the heart as region of interest from the 4 chambers images and by counting the number of pixels.[4]

Statistical Analysis

Data are given as the mean±SEM. Data with a skewed distribution (ie, plasma triglyceride [TG] and cholesterol concentrations and

Table 1. Characteristics of the Study Subjects

Subjects Characteristics	NT (n=13)	Pre-HT (n=13)	New-HT (n=9)	P*
Age, y	45±3	46±2	50±2	0.32
BMI, kg · m^{-2}	27.7±1.2	28.3±0.9	29.4±0.7	0.17
Lean mass, kg	64±2	66±3	64±2	0.67
Fat mass, kg	21±2	23±2	27±2	0.07
Fat mass, %	24±1	25±1	29±1	0.05
VF mass, kg	1.1±0.2	1.4±0.2	1.8±0.2	0.04
SC mass, kg	3.0±0.3	3.4±0.3	3.2±0.2	0.66
Total cholesterol, mmol/L	4.8 (4.1 to 5.2)	4.8 (4.0 to 5.7)	5.2 (4.9 to 5.9)	0.14
HDL cholesterol, mmol/L	1.1 (1.1 to 1.3)	1.2 (1.1 to 1.3)	0.9 (0.9 to 1.1)	0.17
LDL cholesterol, mmol/L	3.4 (3.1 to 3.8)	3.2 (2.5 to 4.1)	3.5 (3.3 to 3.7)	0.36
Triglycerides, mmol/L	0.66 (0.55 to 0.79)	0.88 (0.67 to 1.25)	1.40 (1.04 to 2.44)	0.002
HbA_{1c}, %	4.9±0.1	4.9±0.1	4.9±0.1	0.95
Fasting plasma glucose, mmol/L	5.5±0.1	5.6±0.2	5.7±0.2	0.40
Fasting plasma insulin, pmol/L	72 (64 to 143)	85 (77 to 106)	87 (60 to 120)	0.60
Fasting plasma FFA, mmol/L	0.374±0.036	0.566±0.055	0.627±0.082	0.027
Mean glucose (0 to 180), mmol/L	6.9±0.2	7.6±0.3	8.0±0.5	0.058
Mean insulin (0 to 180), pmol/L	280 (219 to 333)	449 (322 to 672)	563 (335 to 696)	0.01
Mean FFA (0 to 180), mmol/L	0.179±0.019	0.228±0.023	0.280±0.030	0.036
Insulin sensitivity, mL · min^{-1} · kg^{-1}	9.5±0.3	9.1±0.4	7.8±0.4	0.018
Plasma adiponectin, μg/mL	6.0±0.8	6.0±0.5	5.7±0.9	0.96
Plasma leptin, ng/mL	3.6±0.5	4.8±1.2	4.5±0.9	0.68
Plasma norepinephrine, pg/mL	263±23	228±18	258±27	0.50
SBP, mm Hg	113±1	130±1	150±2	<0.0001
DBP, mm Hg	67±2	76±3	94±2	<0.0001
MBP, mm Hg	80±1	94±2	112±2	<0.0001

Data are given as mean±SE or median (interquartile range). BMI indicates body mass index; SBP, systolic BP; DBP, diastolic BP; MBP, mean BP; LDL, low-density lipoprotein; HDL, high-density lipoprotein; SC, subcutaneous.

*Data are from an ANOVA or Kruskal-Wallis test.

insulin parameters) are given as median and interquartile range and were log transformed for use in statistical analysis. Mean glucose, insulin, and FFA during OGTT were calculated by dividing areas under the curves (calculated by the trapezoidal rule) by 180 minutes. Insulin sensitivity was calculated as the oral glucose insulin sensitivity index, which equals the average metabolic clearance rate of glucose during the OGTT and has been validated against the euglycemic insulin clamp technique.[16,17] Group values were compared by ANOVA or Kruskal-Wallis test for normal and nonnormal variables, respectively. General mixed-linear models were used to control for covariance; linear contrasts were then applied to estimate group differences.

Results

Clinical and Metabolic Parameters

Total adiposity (as percentage of fat mass) and VF (but not subcutaneous) increased across groups, whereas all of the other anthropometric variables did not differ (Table 1). Among metabolic parameters, serum TGs, fasting and postglucose FFAs, and postglucose insulin levels rose from NT to new-HT through pre-HT, whereas insulin sensitivity declined. Norepinephrine levels were similar among the 3 groups, and also the adipocytokines (adiponectin and leptin) did not differ significantly.

Global and Regional Cardiac Function

Indices of global cardiac function (left and right ventricle ESV and EDV and ejection fraction) were within the respective reference ranges in all of the subjects and did not differ among groups (Table 2). End systolic wall stress, calculated using the Grossman formula, was also similar. Only LV mass showed a tendency to increase across the groups. On the other hand, epicardial fat area increased across the groups by ≈50%.

LV myocardial shortening showed a marked regional variation: in the whole data set, systolic shortening at the base was consistently lower than in apical segments (−14.9±0.4 versus −16.0±0.6; P=0.03). In all 3 of the segments, circumferential shortening was reduced across the groups when compared with NT individuals (Figure 1), indicating that there was already an alteration in regional cardiac function as subjects progress from pre-HT to essential hypertension.

Correlations

In univariate analysis of the whole data set, higher BP (as the mean BP) was associated with older age, increased fat mass in the intra-abdominal and epicardial areas, higher fasting TG

Table 2. Cardiac MRI Parameters

Cardiac Parameters	NT (n=13)	Pre-HT (n=13)	New-HT (n=9)	P*
Left Ventricle				
LV-EDV$_i$, mL · m^{-2}	78±3	72±3	65±4	0.06
LV-ESV$_i$, mL · m^{-2}	29±1	25±2	21±3	0.04
LV-SV$_i$, mL · m^{-2}	48±3	47±2	44±3	0.51
LV-mass$_i$, g · m^{-2}	67±2	73±3	77±5	0.06
LV-EF, %	62±1	65±2	67±3	0.19
Basal LV circumferential strain, %	−16.3±0.4	−13.9±0.8	−14.1±0.9	0.04
Mid-LV circumferential strain, %	−16.9±0.6	−15.7±0.8	−13.7±0.8	0.03
Apical LV circumferential strain, %	−18.1±0.6	−16.2±0.8	−12.7±1.1	0.0004
Mean LV circumferential strain, %	−17.0±0.5	−15.2±0.7	−13.6±0.8	0.004
Wall stress, 10^3 N/m^2	6.6±0.5	7.3±0.6	6.4±0.7	0.49
Right ventricle				
RV-EDV$_i$, mL · m^{-2}	77±4	74±3	71±3	0.51
RV-ESV$_i$, mL · m^{-2}	30±2	27±2	26±1	0.34
RV-SV$_i$, mL · m^{-2}	47±3	46±2	45±3	0.91
RV-EF, %	61±2	63±2	63±2	0.57
Cardiac ectopic fat				
Epicardial fat, mm^{-2}	2368±227	3222±313	3818±336	0.005

Data are given as mean±SE. EF indicates ejection fraction; i, indexed; RV, right ventricular; SV, stroke volume.

concentration, higher FFA levels during the OGTT, and impaired insulin sensitivity (Table 3). Among cardiac parameters, mean BP was associated with increased LV mass and decreased circumferential shortening in all of the segments. No association was evident between norepinephrine and regional or global cardiac function.

Decreased circumferential shortening was associated with higher fat mass and ectopic fat accumulation in both visceral and epicardial areas, higher fasting TG and post-OGTT FFA levels, and insulin resistance. Importantly, in these relationships, data from all of the study subjects were distributed continuously along the regression lines (Figure 2) and were still significant after controlling for age. Both TG level and epicardial fat alone explained >35% of the variability on apical shortening. The tight association between apical shortening and ectopic fat accumulation was still significant after adjusting for both age and BP (r=0.60, P<0.0003 for epicardial fat and r=0.58, P<0.04 for visceral fat).

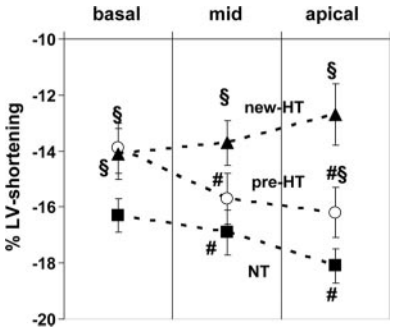

Figure 1. LV circumferential strain in basal, midheart, and apical regions in subjects with normal BP (NT, filled squares), pre-HT (open circles), and new-HT (filled triangles). # P<0.05 for each group vs basal strain; §P<0.05 vs NT.

When both epicardial and TG levels were put in a multiple regression analysis, the apical shortening variability that was explained by this model was 46%. The addition as independent variables of age, mean BP, body mass index, VF, or insulin sensitivity did not improve the variability explained, showing that these 2 variables (TG level and epicardial fat) are very important to LV shortening, even if this will require further studies to know the implications.

LV mass was associated with increased BP, but after accounting for mean BP and age, the only parameter that remained significantly correlated was insulin resistance (r=0.34; P=0.05). Insulin resistance was associated with increased BP, reduced LV shortening (Table 3), and increased visceral and epicardial fat. The latter correlations hold even after adjusting for age and mean BP (VF: partial r=−0.58, P=0.0004; epicardial: partial r=−0.35, P=0.04).

Discussion

The main finding of the present study is that patients with pre-HT and early, untreated hypertension show a graded reduction in systolic function, as documented by decreased LV shortening, despite normal ventricular volumes and pump function and similar values of wall stress. The reduction in regional systolic function was directly related to BP in a continuous fashion but also, and more interestingly, to metabolic abnormalities, such as increased epicardial and visceral fat accumulation, TG concentration, and insulin resistance.

Regional Cardiac Function and BP

We found LV shortening reduced in pre-HT and new-HT patients in all 3 of the regions (basal, middle, and apical LV segments) as compared with a control group of NT volunteers. These results are in line with a recent study that documented a reduction in circumferential shortening in a

Table 3. Correlation Matrix

Variables	MBP	LV Mass$_i$	Mean LV % Shortening	Basal LV % Shortening	Mid-LV % Shortening	Apical LV % Shortening
Age	0.36	NS	NS	NS	NS	NS
MBP	1	0.42	−0.41	NS	NS	−0.49
% fat	NS	NS	−0.46	NS	−0.50	−0.48
VF	0.39	NS	−0.49	−0.34	−0.48	−0.46
Epicardial fat	0.51	NS	−0.46	−0.32	−0.36	−0.59
Mean insulin	NS	NS	NS	NS	NS	NS
Mean FFA	0.42	NS	−0.53	NS	−0.55	−0.52
Mean glucose	0.33	NS	NS	NS	NS	NS
TGs	0.57	NS	−0.46	NS	−0.55	−0.60
Insulin sensitivity	−0.37	NS	0.43	NS	0.49	0.40
Adiponectin	NS	NS	NS	NS	NS	NS
Leptin	NS	NS	NS	NS	NS	NS

NS indicates not significant; i, indexed; MBP, mean BP.

large cohort of hypertensive patients[18] but has as a limitation the inclusion of patients who were pharmacologically treated or had diabetes; made no distinction between pre-HT, new HT, and treated hypertension; and, most importantly, did not report cardiac function. The patients studied here were only men, carefully selected with pre-HT or new HT, without diabetes (OGTT proven), and with a normal cardiac function, thus avoiding the most common confounding factors. Our results suggest that regional LV function is a progressive defect that worsens progressively as BP increases and is already present in subjects with mild increased BP also when levels are not yet considered pathological (ie, pre-HT) and in the absence of other cardiac abnormalities (ie, cardiac volumetry and global function). Thus, the impaired intramural systolic function may precede alterations in LV volumes and global function.

Regional Cardiac Function and Metabolic Abnormalities

Although it was not surprising that the observed reduction in LV circumferential shortening was directly related to BP (even after accounting for age), a novel finding was the association with metabolic abnormalities, such as insulin resistance, ectopic fat (visceral and epicardial) accumulation, and hypertriglyceridemia. Insulin resistance has been shown previously in patients with essential hypertension directly

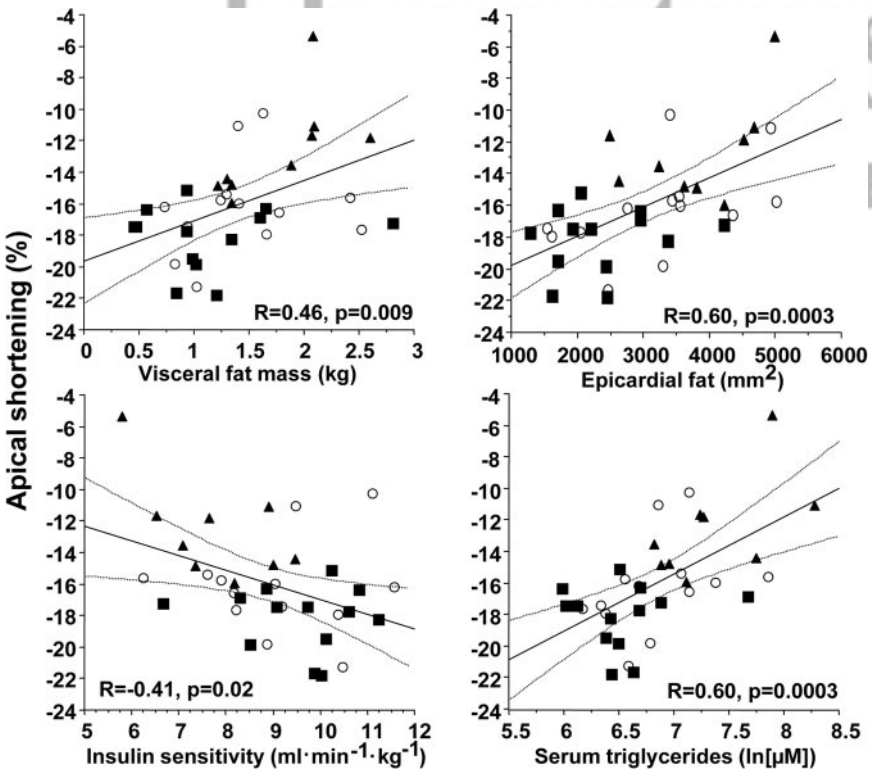

Figure 2. Relationships between apical LV circumferential shortening and metabolic parameters in subjects with normal BP (filled squares), pre-HT (open circles), or new-HT (filled triangles). The full lines are the best fit (and the dotted lines are their 95% CIs) of the age-adjusted residuals.

related to BP,[4,19,20] and many studies have linked insulin resistance with cardiovascular risk.[21] We found insulin resistance associated not only with increased mean BP but also with reduced LV shortening and increased LV mass, even if this value was within reference ranges.

Insulin resistance and increased BP have often also been associated with ectopic fat accumulation and lipid abnormalities.[4,22] Both visceral and epicardial fat were independently associated with insulin resistance and mean BP, but the novel finding is that both were associated with reduced circumferential shortening. Because the role of ectopic fat deposits on cardiac metabolism is still not well understood, we can only speculate. Both visceral and epicardial fat are highly lipolytic,[23–25] and there is growing evidence that alterations in energy substrate metabolism contribute to LV remodeling, systolic dysfunction, and cardiac hypertrophy.[26] A possible role of excess lipids in altered heart metabolism has been hypothesized recently,[26] and experimental studies in Zucker diabetic fatty rats indicate that cardiac "steatosis" can lead to impaired myocardial contractility and cardiomyopathy.[27,28] In this cohort of subjects, impaired suppression of FFA by insulin during the OGTT and higher TG levels correlated with epicardial fat, reduced regional ventricular shortening, and reduced insulin sensitivity, indicating a role for chronic substrate alterations in the reduction of regional LV function. It is known that adipose tissue is not only a source of FFA but also secretes adipokines and inflammatory cytokines (monocyte chemotactic protein-1, interleukin-6, interleukin-1β, and tumor necrosis factor-α),[29,30] and it was shown recently that epicardial adipose tissue had a high expression of chemokines and inflammatory cytokines.[31,32] In this study, epicardial fat alone explained 35% of the total variability of reduced LV shortening at the apical level, suggesting epicardial fat as a new cardiometabolic risk marker.[29,30]

Limits of the Study

Among the limits of this study there is the small size of the studied cohort. However, this cohort of subjects was composed only of male subjects (the major part of the hypertensive population) without diabetes with similar body mass index and age and without treatments that can affect cardiac function or metabolism. This high selection, together with the high resolution and reproducibility of the tagging technique, the abdominal imaging, and the careful metabolic analyses, optimized the likelihood of detecting the subtle differences that characterize the early stages of the hypertensive disease. In conclusion, regional LV function is already reduced at early stages of hypertension, especially at the apical level, and progressively decreased with increased mean BP and deterioration in metabolic profile, such as increased insulin resistance, TG concentration, and epicardial and visceral fat accumulation.

Perspectives

Regional LV function is a progressive defect that worsens progressively as BP increases and is already present in subjects with mild increased BP also when levels are not yet considered pathological (ie, pre-HT) and in the absence of other cardiac abnormalities (ie, cardiac volumetry and global function). The concomitant presence of metabolic derangements (eg, insulin resistance, increased TGs, obesity, and increased waist circumference) suggests that they could play important roles and should be evaluated also in the early stage of hypertension. In the presence of pre-HT and metabolic alterations, changes in lifestyle should be recommended to prevent a possible deterioration in LV function and development of hypertensive cardiomyopathy.

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Disclosures

None.

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