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Abdominal obesity and structure and function of the heart in healthy male Koreans
The ARIRANG study

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Abstract

Although central obesity is a more powerful predictor of cardiovascular disease (CVD) than general obesity, there is limited information on structural and functional changes of the heart in central obesity. Therefore, we evaluated the association between abdominal obesity and geometric and functional changes of the heart in healthy males. A total of 1460 healthy males aged 40 to 70 years without known CVD from the Korean Genome and Epidemiology Study on Atherosclerosis Risk of Rural Areas in the Korean General Population were included. All individuals underwent conventional 2-dimensional echocardiography and tissue Doppler imaging to measure left atrial (LA) and left ventricle (LV) geometry and function. Increasing tertiles of waist circumference (WC) were associated with stepwise increases in LA volume, LV end-diastolic dimension, LV mass to height^2, deceleration time of E wave, and lower E/A ratio (all P trends <0.001). In multivariable logistic regression models, the odds ratios for LA enlargement, LV hypertrophy, LV enlargement, and diastolic dysfunction comparing the upper tertile of WC (>89 cm) to the lowest tertile (<82 cm) were 2.81 (95% confidence interval [CI] 2.24–3.54), 3.65 (95% CI 2.54–5.26), 4.23 (95% CI 2.61–6.87), and 1.75 (95% CI 1.37–2.22), respectively. LV ejection fraction and relative wall thickness were not increased with increasing WC. The association between WC and LA enlargement, LV enlargement, and diastolic dysfunction persisted after stratification by body mass index tertiles. Central obesity may be a stronger predictor than general obesity of geometric and functional changes in the LV and LA.

Abbreviations: BMI = body mass index, CVD = cardiovascular disease, DBP = diastolic blood pressure, HDL = high-density lipoprotein, hs-CRP = high-sensitivity C-reactive protein, LA = left atrial, LDL = low-density lipoprotein, LV = left ventricle, SBP = systolic blood pressure, WCs = waist circumferences.

Keywords: abdominal obesity, left ventricular function, ventricular remodeling

1. Introduction

Central obesity is a stronger risk factor for atherosclerotic cardiovascular disease (CVD), heart failure, and diabetes than general obesity.[1–5] Furthermore, waist circumference (WC), a measure of central obesity, shows a graded association with CVD even in lean subjects.[4,5] The impact of central obesity on the structure and function of the heart, however, has received far less attention, with most reports focusing on the association of WC or body mass index (BMI) with left ventricular (LV) mass.[6–8] It is also still unclear if central adiposity is a stronger determinant of cardiac structural and functional changes than general adiposity, particularly in lean individuals.

The objective of this study was thus to evaluate the association of WC and BMI with LV structural and functional changes in the heart in an apparently healthy male population in Korea. We hypothesized that WC would be more strongly associated than BMI with geometric changes in the LV and left atrium (LA), and with functional changes in LV.

2. Methods

2.1. Study population

We used data from the Korean Genome and Epidemiology Study on Atherosclerosis Risk of Rural Areas in the Korean General

Population (KoGES-ARIRANG), a population-based prospective cohort study to assess the prevalence, incidence, and risk factors for chronic degenerative disorders such as hypertension, diabetes, and CVD. KoGES-ARIRANG invited all adults residing in the rural areas of Wonju and Pyeongchang in South Korea, where demographic shifts are infrequent and the population can be followed long term, to participate. For this report, we are using data from the baseline survey, carried out from November 2005 to January 2008, which included 2127 men aged 40 to 70 years. After excluding men with a history of CVD or cancer, we performed a complete echocardiographic assessment in 1576 men. We then excluded 116 participants who were taking antihypertensive medications or had a high creatinine level (>1.4 mg/dL), resulting in a final sample size of 1460 men.

2.2. Data collection

Study participants completed a standardized medical history and lifestyle questionnaire and underwent a comprehensive health examination according to standard procedures. Body weight and height were measured while participants were wearing light indoor clothing without shoes. BMI was calculated as weight in kilograms divided by height in meters squared. WC was measured in a horizontal plane, midway between the inferior margin of the ribs and the superior border of the iliac crest using a tape measure (SECA-200, SECA, Hamburg, Germany). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured twice in the right arm using a standard mercury sphygmomanometer (Baumanometer, Copiague, NY). The lower SBP and DBP readings were used for data analyses. Smoking status was determined based on self-report.

A venous blood sample was drawn from study participants after fasting for >12 hours or overnight. Fasting glucose was determined by a glucose oxidase–based assay. Fasting insulin was determined by a double-antibody RIA assay (Biosource, Nivelles, Belgium). Serum concentrations of low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides were determined by enzymatic methods (Advia 1650, Siemens, Tarrytown, NY). High-sensitivity C-reactive protein (hs-CRP) was measured by the Denka Seiken (Tokyo, Japan) assay, which has been validated against the Dade Behring (Newark, USA) method.

2.3. Echocardiographic assessment

Echocardiography was performed in the harmonic imaging mode using a 3-MHz transducer in a commercial ultrasound system (Vivid-7; General Electric-Vingmed, Milwaukee, WI). LV internal dimensions, LV wall thickness, and LV ejection fraction (LVEF; measured using the biplane modified Simpson rule) were measured following the recommendations of the American Society of Echocardiography (ASE).[9]

LV mass was calculated following ASE recommendations as LV mass $= 0.8 \times \{1.04 \times [(PWTd + VSTd + LVIDd)^3 - (LVIDd)^3]\} + 0.6 \, g$, where PWTd and SWTd were the posterior and septal wall thickness at end diastole, respectively, and LVIDd was the M-mode LV dimension with the short axis view at end-diastole.[10] Allometric height-based adjustments were used to express the LV mass index per height$^{2.7}$, which provides a more accurate estimation of LV hypertrophy and other pathological changes in heart structure, particularly in obese subjects.[11]

Relative wall thickness (RWT), which increases with concentric remodeling and concentric hypertrophy, was calculated as RWT $= 2 \times$ PWTd/LVIDd. The LA dimension was measured by 2-dimensional-guided M-mode echocardiography using the parasternal short-axis view at the base of the heart. Three LA dimensions were used to calculate the LA volume as an ellipse using the formula LA volume $= (\pi/6) \times (SA_1 \times SA_2 \times LA)$, where SA_1 is the M-mode LA dimension, and SA_2 and LA are measurements of the short- and long-axis, respectively, in the apical 4-chamber view at ventricular end-systole. The LA volume index was calculated by dividing LA volume by body surface area.[9]

Transmitral inflow velocities were measured using pulsed-wave Doppler in the apical 4-chamber view with the sample volume placed at the mitral valve leaflet tips.[12] Measurements of the transmitral early diastolic (E wave) and atrial (A wave) velocities were used to calculate the E/A ratio and E wave deceleration time. Tissue Doppler imaging in the apical 4-chamber view was used to measure LV myocardial velocities, with the sample volume placed at the septal mitral annulus. The early (E′) and late diastolic velocities (A′) were measured, and the E/E′ ratio was calculated.[12,13]

Cutoffs for defining abnormal chamber size and function followed ASE recommendations.[9,12] LV hypertrophy was defined as a LV mass >48 g/m$^{2.7}$, LA enlargement was defined as LA volume >58 mL, and LV enlargement was defined as a LV end-diastolic dimension >5.9 cm. Diastolic dysfunction was graded on a 4-point ordinal scale: normal; mild diastolic dysfunction, defined as abnormal relaxation without increased LV end-diastolic filling pressure (E/A ratio <0.75); moderate or "pseudonormal" diastolic dysfunction, defined as abnormal relaxation with increased LV end-diastolic filling pressure (E/A ratio 0.75–1.5, deceleration time >140 ms, and 2 other Doppler indices of elevated LV end-diastolic filling pressure); or severe diastolic dysfunction, defined as advanced reduction in compliance with restrictive filling (E/A ratio >1.5, deceleration time <140 ms, and Doppler indices of elevated LV end-diastolic filling pressure).

2.4. Statistical analyses

We divided the study population into tertiles of WC (cut-points at 82 and 89 cm) and compared CV risk factors and echocardiographic parameters across tertiles of WC using the analysis of variance test. Multivariable logistic regression was used to assess the independent association of WC with LA enlargement, LV enlargement, LV hypertrophy, diastolic dysfunction adjusting for age (continuous variable), SBP (continuous variable), smoking (current/former/never), total cholesterol (continuous variable), and C-reactive protein (log-transformed continuous variable). Results were expressed as odds ratios with 95% confidence intervals (CIs). P values <0.05 were considered statistically significant. All analyses were performed using SPSS 12.0 software (SPSS Inc., Chicago, IL).

2.5. Ethics approval

The study protocol was approved by the Institutional Review Board of the Wonju Severance Christian Hospital. All participants provided written informed consent.

3. Results

The average (SD) age, WC, and BMI of study participants were 54.5 (8.1) years, 84.8 (6.2) cm, and 24.4 (2.6) kg/m^2, respectively.

Table 1

Demographic, laboratory, and echocardiographic characteristics by waist circumference tertile.

	First (<82 cm) (n = 487)	Second (82–89 cm) (n = 464)	Third (≥89 cm) (n = 509)	P
Age, y	52.6 ± 8.0	54.4 ± 7.9	55.7 ± 8.0	<0.001
SBP, mm Hg	128.9 ± 16.9	135.2 ± 19.5	136.9 ± 18.6	<0.001
SBP ≥ 140 mm Hg, n (%)	103 (21.2)	160 (34.5)	211 (41.5)	<0.001
DBP, mm Hg	79.5 ± 10.7	83.4 ± 11.87	82.6 ± 11.6	<0.001
Pulse rate, rate/min	71.1 ± 10.1	71.2 ± 10.2	72.4 ± 10.5	0.17
BMI, kg/m^2	22.0 ± 2.2	24.2 ± 2.0	27.0 ± 3.7	<0.001
Fasting glucose, mg/dL	93.8 ± 16.5	98.9 ± 22.6	100.8 ± 23.5	<0.001
Total cholesterol, mg/dL	196.8 ± 35.4	206.2 ± 39.4	209.6 ± 40.1	<0.001
HDL-cholesterol, mg/dL	48.4 ± 11.9	44.9 ± 10.3	43.4 ± 10.2	<0.001
LDL-cholesterol, mg/dL	110.9 ± 30.6	117.9 ± 21.8	120.6 ± 35.3	<0.001
Triglyceride, mg/dL	119.6 ± 75.8	167.1 ± 165.0	187.2 ± 114.9	<0.001
hs-CRP, mg/dL	1.61 ± 6.5	1.82 ± 3.9	2.47 ± 6.0	0.09
LA volume, mL	28.2 ± 9.4	32.5 ± 9.8	35.7 ± 10.7	<0.001
LA volume index, mL/m^2	17.9 ± 5.6	19.2 ± 5.7	19.9 ± 5.6	<0.001
LA enlargement, n (%)	4 (0.70)	6 (1.3)	14 (2.8)	0.045
LVEDd, cm	4.9 ± 0.5	5.1 ± 0.5	5.2 ± 0.6	<0.001
LVESd, cm	3.1 ± 0.5	3.3 ± 0.5	3.4 ± 0.6	<0.001
LV enlargement, n (%)	10 (2.1)	21 (4.6)	52 (10.2)	<0.001
LV mass, g	122.0 ± 31.8	142.8 ± 36.7	157.4 ± 39.2	<0.001
LVM/height$^{2.7}$, g/m$^{2.7}$	35.4 ± 9.6	38.4 ± 10.9	40.9 ± 10.9	<0.001
RWT	0.31 ± 0.06	0.32 ± 0.74	0.32 ± 0.08	0.13
LV hypertrophy, n (%)	30 (6.2)	68 (14.7)	124 (24.4)	<0.001
LVEF, %	65.1 ± 7.7	63.4 ± 8.6	62.8 ± 8.7	0.048
D-time, ms	217.3 ± 53.3	230.2 ± 57.3	240.2 ± 76.4	<0.001
E/A	1.12 ± 0.34	1.03 ± 0.38	0.97 ± 0.31	<0.001
E/E'	9.84 ± 3.01	10.24 ± 2.86	10.31 ± 3.26	0.06

Data are expressed as mean ± standard deviation. BMI = body mass index, DBP = diastolic blood pressure, D-time = deceleration time, HDL = high-density lipoprotein, hs-CRP = high-sensitivity C-reactive protein, LA = left atrium, LDL = low-density lipoprotein, LV = left ventricle, LVEF = left ventricle ejection fraction, LVEDd = left ventricular end-diastolic dimension, LVESd = left ventricular end-systolic dimension, LVM = left ventricular mass, RWT = relative wall thickness, SBP = systolic blood pressure.

Increasing WC was significantly associated with stepwise increases in blood pressure, fasting glucose, triglycerides and LDL-cholesterol, and with decreases in HDL-cholesterol (Table 1). With respect to cardiac structure and function measures, increasing WC was significantly associated with a progressive increase in LA volume index, LV end-diastolic and end-systolic dimensions, and LV mass index (Table 1). RWT did not increase with increasing WC. Increased WC was also associated with decreasing LVEF, but most of the difference occurred between the first and the second tertiles of WC.

Increasing WC was also associated with diastolic dysfunction, including significantly longer deceleration time of the E wave and decreasing E/A ratio ($P < 0.01$). Although E/E′ tended to increase with increasing WC, this relationship was only marginally significant ($P = 0.06$).

In multivariable logistic regression models adjusted for age, SBP, total cholesterol, hs-CRP and fasting glucose, the odd ratio for LA enlargement comparing men in the highest tertile of WC to the lowest tertile was 2.81 (95% CI 2.24–3.54) (Table 2). The corresponding odds ratios and 95% CI for LV enlargement, LV

Table 2

Odds ratios for LA enlargement, LV enlargement, LV hypertrophy, and LV diastolic dysfunction according to the tertile of waist circumference and clinical variables.

		LA enlargement	LV enlargement	LV hypertrophy	LV diastolic dysfunction
Waist circumference	First tertile (<82 cm)	1	1	1	1
	Second tertile (82–89 cm)	1.83 (1.46–2.29)	1.94 (1.15–3.30)	2.18 (1.49–3.19)	1.56 (1.22–1.98)
	Third tertile (≥89 cm)	2.81 (2.24–3.54)	4.23 (2.60–6.87)	3.65 (2.54–5.26)	1.75 (1.37–2.22)
Age, per y		1.02 (1.01–1.03)	1.01 (0.99–1.03)	1.06 (1.05–1.09)	0.99 (0.98–1.01)
Fasting glucose, per mg/dL		0.99 (0.99–1.00)	1 (0.99–1.01)	0.99 (0.99–1.00)	1 (1.00–1.01)
SBP, per mm Hg		0.99 (0.99–1.00)	0.99 (0.98–1.00)	1.01 (1.01–1.02)	1.01 (1.00–1.01)
Total cholesterol, per mg/dL		1 (1.00–1.01)	1 (0.99–1.01)	0.99 (0.99–1.00)	1 (0.99–1.00)
hs-CRP, per unit of log-transformed mg/L		0.99 (0.99–1.01)	1.01 (0.98–1.04)	0.98 (0.95–1.02)	0.99 (0.97–1.01)

Odds ratios were adjusted for all variables in the table. CI = confidence interval, hs-CRP = high-sensitivity C-reactive protein, LA = left atrium, LV = left ventricle, OR = odds ratio, SBP = systolic blood pressure.
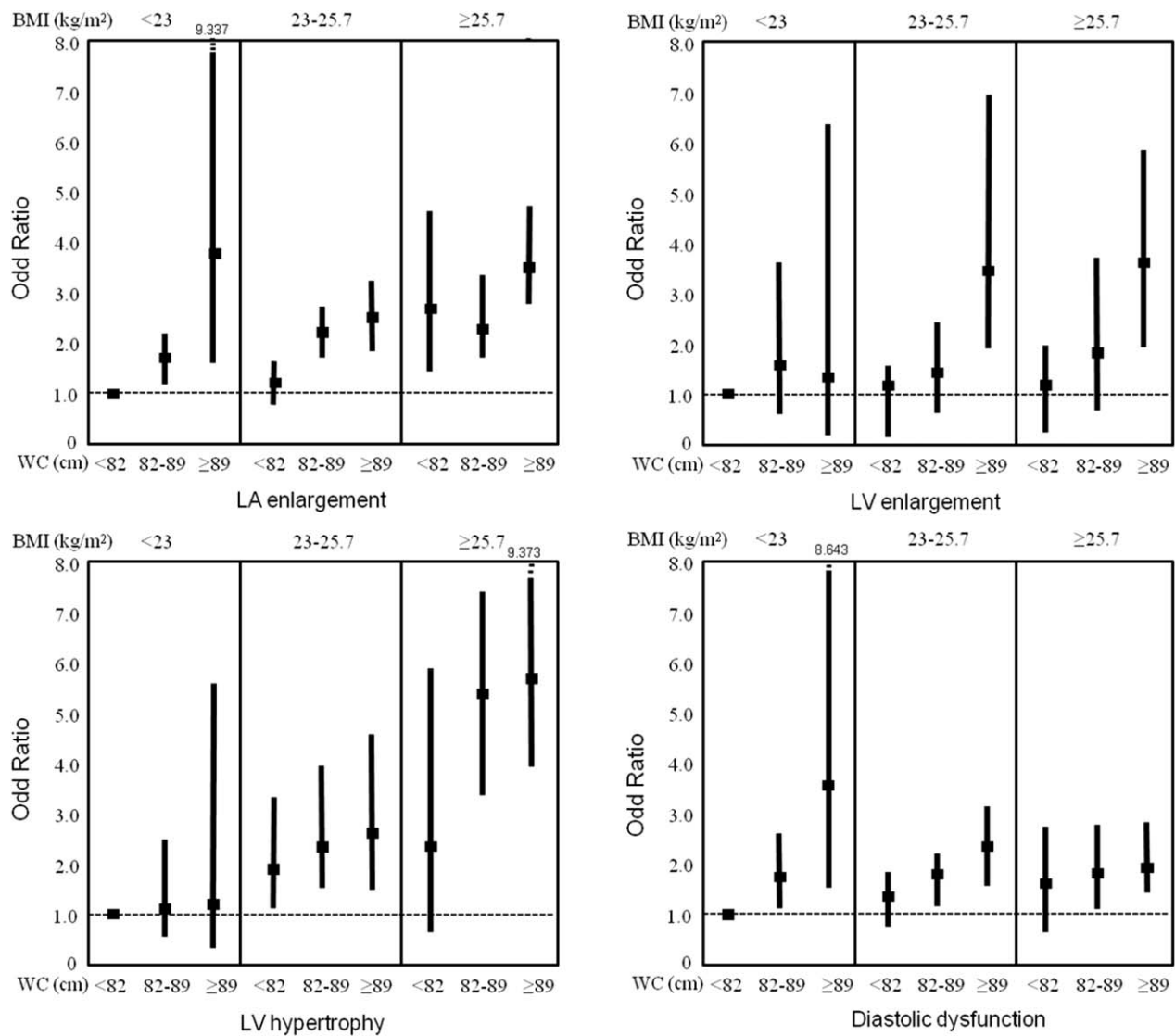
Figure 1. Adjusted odds ratios for left atrial enlargement, left ventricle (LV) enlargement, LV hypertrophy, and diastolic dysfunction according to the each BMI and WC tertiles. Adjusted for age, smoking, systolic blood pressure, fasting glucose, total cholesterol, and C-reactive protein. Bars indicate 95% confidence interval for odds ratio of each dependent variable. BMI = body mass index, LA = left atrium, LV = left ventricle, WC = waist circumference.

hypertrophy, and diastolic dysfunction were 4.23 (2.60–6.87), 3.65 (2.54–5.26), and 1.75 (1.37–2.22). Increasing WC, however, was not associated with LVEF (odd ratio 1.25, 95% CI 0.82–1.90).

The independent association between WC and increasing odds of LA enlargement, LV enlargement, and diastolic dysfunction persisted within subgroups of BMI, except LA enlargement in BMI ≥ 25.7 and LV enlargement in BMI < 23, but tend to persist (Fig. 1). Indeed, both WC and BMI were associated with LA enlargement, LV enlargement, and diastolic dysfunction, but the association was stronger with WC compared to BMI. LV hypertrophy, however, was associated with increasing BMI, but not with increasing WC (Fig. 1).

4. Discussion

In the ARIRANG study, we found that WC, a measure of central obesity, was an independent predictor of LA enlargement, eccentric LV hypertrophy, and LV diastolic dysfunction. This

association was independent from and stronger than the association of BMI with these parameters. While both WC and BMI contributed to LA and LV enlargement and to LV diastolic dysfunction, WC seemed more closely linked to these abnormalities than BMI. LV hypertrophy, however, was related primarily to BMI instead of WC.

Excess adiposity increases metabolic demands and results in increased cardiac output and chronic volume overload, leading to LV structural remodeling with LV dilatation and compensatory LV hypertrophy.[14,15] Several studies, however, have reported concentric rather than eccentric LV hypertrophy in obese subjects,[7,8,16] generating some controversy on the nature of LV remodeling in obesity.[17,18] In ARIRANG, we found that increasing WC was independently associated with eccentric LV remodeling and more closely linked to LV enlargement than BMI, a phenomenon that has been identified in previous studies.[1,19] A novel finding of our study was that BMI was more strongly associated with LV hypertrophy than WC. This finding could be explained by the fact that intrinsic lean body mass, rather than

abdominal fat mass, determines the majority of the metabolic activity and intrinsic difference of LV mass, which thus may be a key contributor of cardiac output and LV hypertrophy.[20,21]

Few studies have evaluated the association of central obesity with diastolic dysfunction. Ammar et al[1] found an odds ratio for diastolic dysfunction of 1.36 for each 1 standard deviation increase in WC. Tadic et al[22] also found that abdominal obesity is associated with LV diastolic dysfunction among the criteria of metabolic syndrome in hypertensive patients. Tsioufis et al,[19] however, found that that central obesity was associated with diastolic dysfunction only in females. In the present study, both BMI and WC were associated with diastolic dysfunction in men, and WC showed a stronger association with LV diastolic dysfunction than BMI. These data imply that central obesity-induced LV diastolic abnormalities might represent a progressive preclinical condition that contributes to obesity-induced heart failure and argue for a potential role of abdominal weight loss in reversing LV diastolic dysfunction.[23]

Several studies have demonstrated increased LA dimensions in general obesity compared with normal-weight subjects.[7,24–26] Unlike LV hypertrophy, LA dimension is not directly associated with body size. In the present study, WC was an independent predictor for high LA volume even subgroups of participants with similar BMI (Fig. 1). Increased LA dimensions with increasing WC could be a consequence of LV hypertrophy in central obesity, which impairs LV diastolic filling, facilitating the development of diastolic dysfunction, and ultimately leading to increasing LA pressure and LA enlargement. LA enlargement is a strong risk factor for several cardiovascular events, including stroke, cardiovascular death, and the risk of developing of atrial fibrillation.[27–29]

4.1. Limitations

Some strengths of our study include the use of a large, generally healthy Korean male population and the use of standardized echocardiographic measurements of LV geometry and function as well as LA structure. However, it was cross-sectional, so cause–effect inferences are difficult to establish. The highest tertile of WC in our study (>89 cm) is still considered "thin" in Western populations. While our results may not generalize to other populations with a higher prevalence of obesity, they also show that central adiposity may affect heart structure and function even in relatively lean populations. Furthermore, our results are directly applicable to many Asian populations, which are still lean by Western standards but are showing increasing prevalence of obesity.

5. Conclusion

Abdominal obesity was independently associated with eccentric LV hypertrophy, LV diastolic dysfunction, and LA enlargement in healthy male Korean adults. The associations between abdominal obesity and LA enlargement, LV enlargement and diastolic dysfunction persisted even within subgroups of BMI. These results suggest that central obesity may be a stronger predictor than general obesity of geometric and functional changes in the LV and LA.

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