

Left Atrial Enlargement is Related to Diastolic Dysfunction in Obese Subjects



Norman N Aiad ^{1, 2} Satyam Sarma MD. ^{1, 2, 3} Michinari Hieda MD. ^{1, 2} Benjamin D. Levine, MD. ^{1, 2, 3}

¹ Institute for Exercise and Environmental Medicine, Texas Health Presbyterian Hospital Dallas, TX

² The University of Texas Southwestern Medical School

³ Department of Internal Medicine, University of Texas Southwestern Medical Center at Dallas

ABSTRACT

Purpose: Obesity is linked to diastolic dysfunction and left atrial enlargement (LAE). The mechanisms responsible for LAE in obesity are unknown. We hypothesized that diastolic dysfunction, which is common in obesity, is the mechanism through which LAE occurs.

Methods: 27 middle-aged, obese patients (8M: 19F, 49±7years) with an average body mass index (BMI) of 38 ±5 kg/m² were compared to age and sex matched non-obese healthy controls. Diastolic function (IVRT; isovolumic relaxation time, Vp: propagation velocity) was assessed using echocardiography. Left atrium (LA) size was measured using 3D echocardiogram, and normalized to height to account for the effects of body size.

Results: Obese individuals had a significantly enlarged atrial volume when compared to healthy individuals (35.9 ± 8 vs. 19.4 ± 10.5 mL/m; p<0.01). Obese patients had a significantly shorter IVRT when compared to non-obese patients (68 ± 11 vs 100 ± 16 msec; p<0.01). Obese patients also had a significantly faster Vp when compared to non-obese patients (75 ± 19 vs 56 ± 13 cm/sec; p<0.01). In obese subjects, there was a significant negative-relationship between LA volume index and IVRT (R²=0.27; p<0.05) as well as a significant positive-relationship between LA volume index and Vp (R²=0.18; p<0.05).

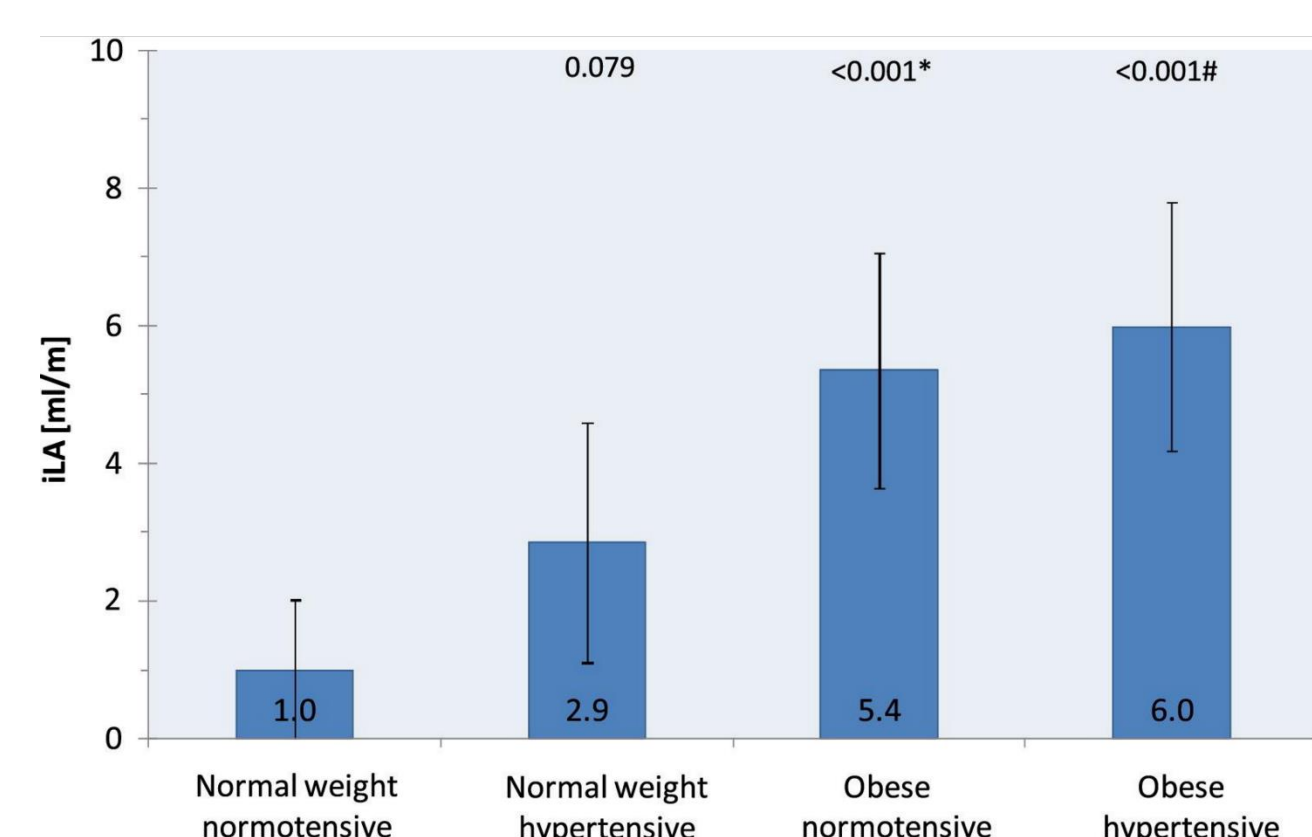
Conclusions: Obese patients have larger LA and markers of elevated LA filling pressures (IVRT and Vp) compared to age matched controls. LAE in obesity may be driven by sub-clinical alterations in diastolic function and left atrial filling pressures.

INTRODUCTION

Background: Obesity is an independent risk factor for atrial fibrillation. This relationship may be mediated by increases in left atrial (LA) size which is common in obesity.

While LA size has been demonstrated to be larger in obese individuals, no mechanism has been proposed to explain this phenomenon.

We investigated whether sub-clinical diastolic dysfunction, specifically elevated LA-LV pressure gradient, could explain increased LA size in obese subjects.



Graph from Stritzke et al showing obesity as an independent risk factor for LA enlargement relative to non-obese controls

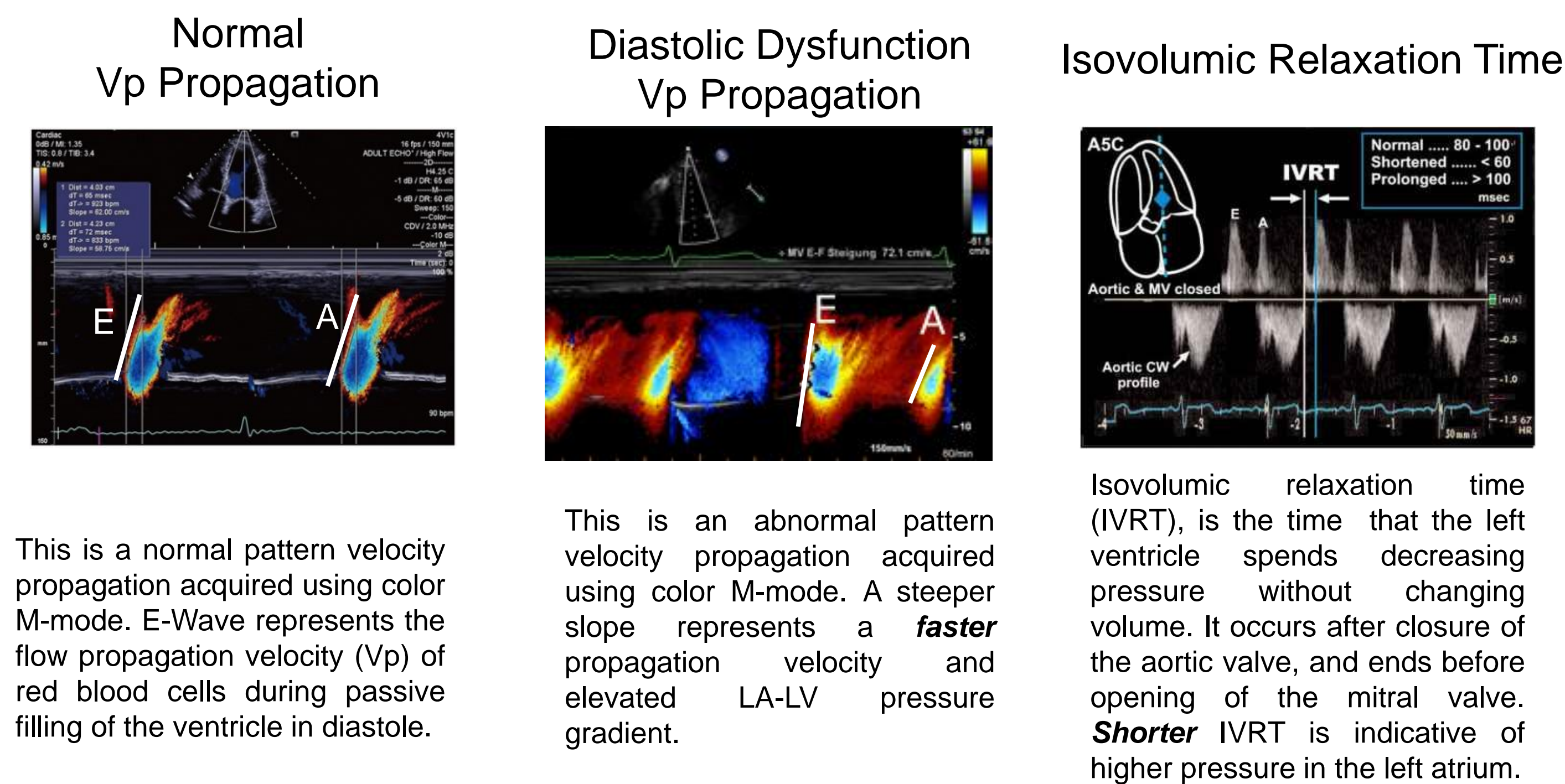
Jan Stritzke, et al. Journal of the American College of Cardiology, Volume 54, Issue 21, 17 November 2009, Pages 1982-1989

PURPOSE

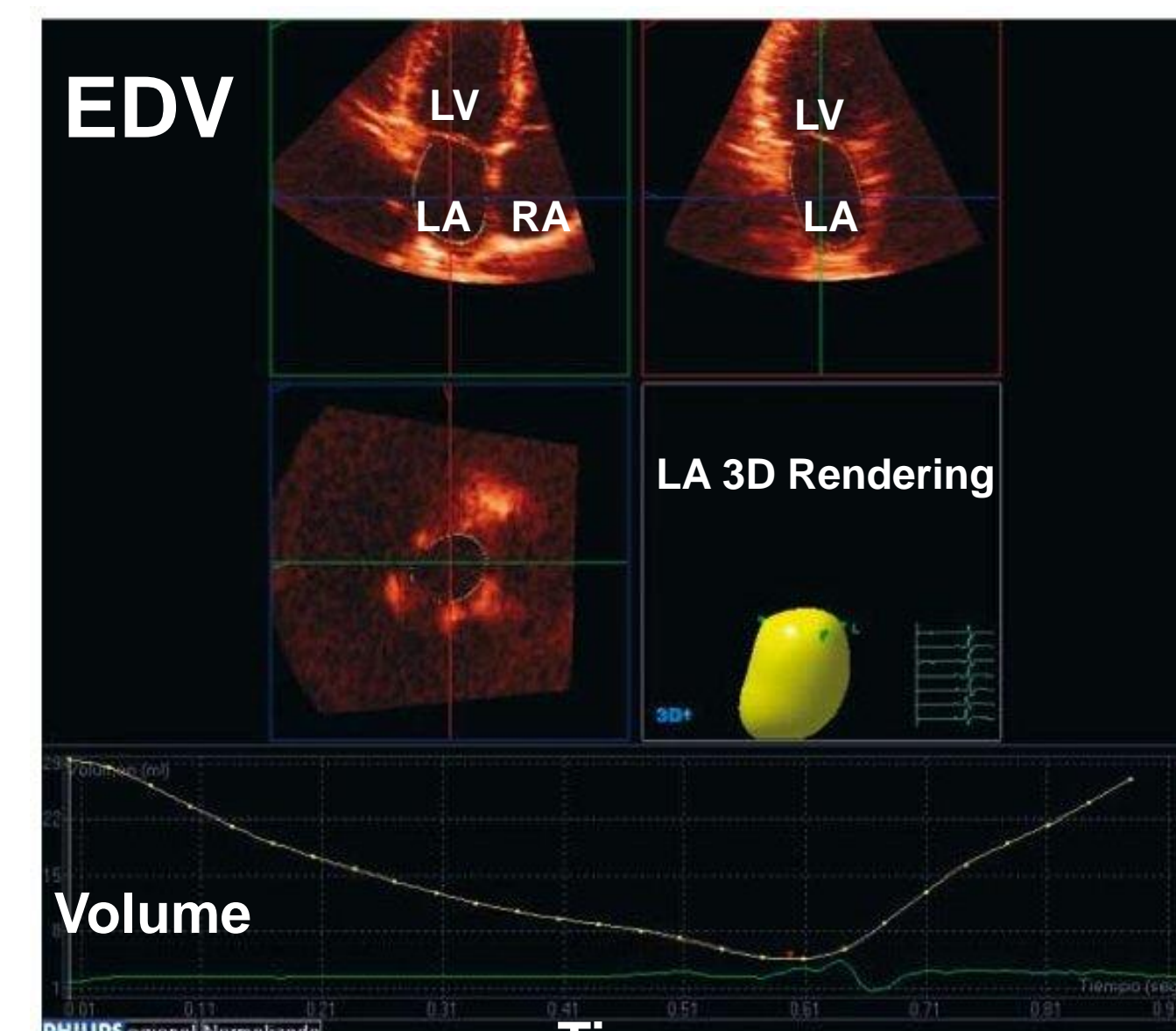
Identify relationships associated with left atrial enlargement in obese individuals using echocardiography

METHODS

Echocardiographic Diastolic Function Parameters



Echocardiographic Volume Measurements



Both LA and LV volume were calculated from 3D echocardiography (Phillips Qlab). End diastolic volumes (EDV) were calculated for each chamber at their maximum volume while end systolic volume (ESV) was calculated just after aortic valve closure (LV ESV) and just before opening of the aortic valve (LA ESV).

Measurements:

- LA Volume Indexed to Height (mL/m): **3D echocardiogram**
- IVRT (msec), Vp (cm/sec) : **Doppler echocardiography**

Subjects: 27 Obese Individuals (8M, 19F) and healthy, non-obese age and sex matched controls

Inclusion criteria:

Controls: BMI <30, Ages 45-64, no insulin dependent DM, CHF, HTN

Obese: BMI >30, <50, Age > 40, <60, Visceral fat >2.5Kg, Elevated serum biomarkers of cTnT, NT-proBNP

Exclusion criteria:

Controls: Previous MI, stroke, BMI >30, sleep apnea, use of CPAP machine, age <45 or >64

Obese: BMI <30 or >45, insulin dependent diabetes, heart failure, severe asthma/COPD, angina or prior myocardial infarction, prior transient ischemic attack or stroke.

RESULTS

1. Obese patients have significantly larger LA and LV volumes when compared to normal age and sex matched controls even when indexed to height. (Table)
2. LA volumes in obese subjects remained higher than controls even after adjusting for LV volume, suggesting a disproportionate increase in LA size to overall heart size. (Table)
3. Shortened isovolumic relaxation time, an indication of increased LA pressure, is related to increased LA volume. (Figure 1)
4. Increased propagation velocity, an indication of increased LA pressure, is related to increased LA volume. (Figure 1)

Table: Left Atrial and Left Ventricular Volume Measurements

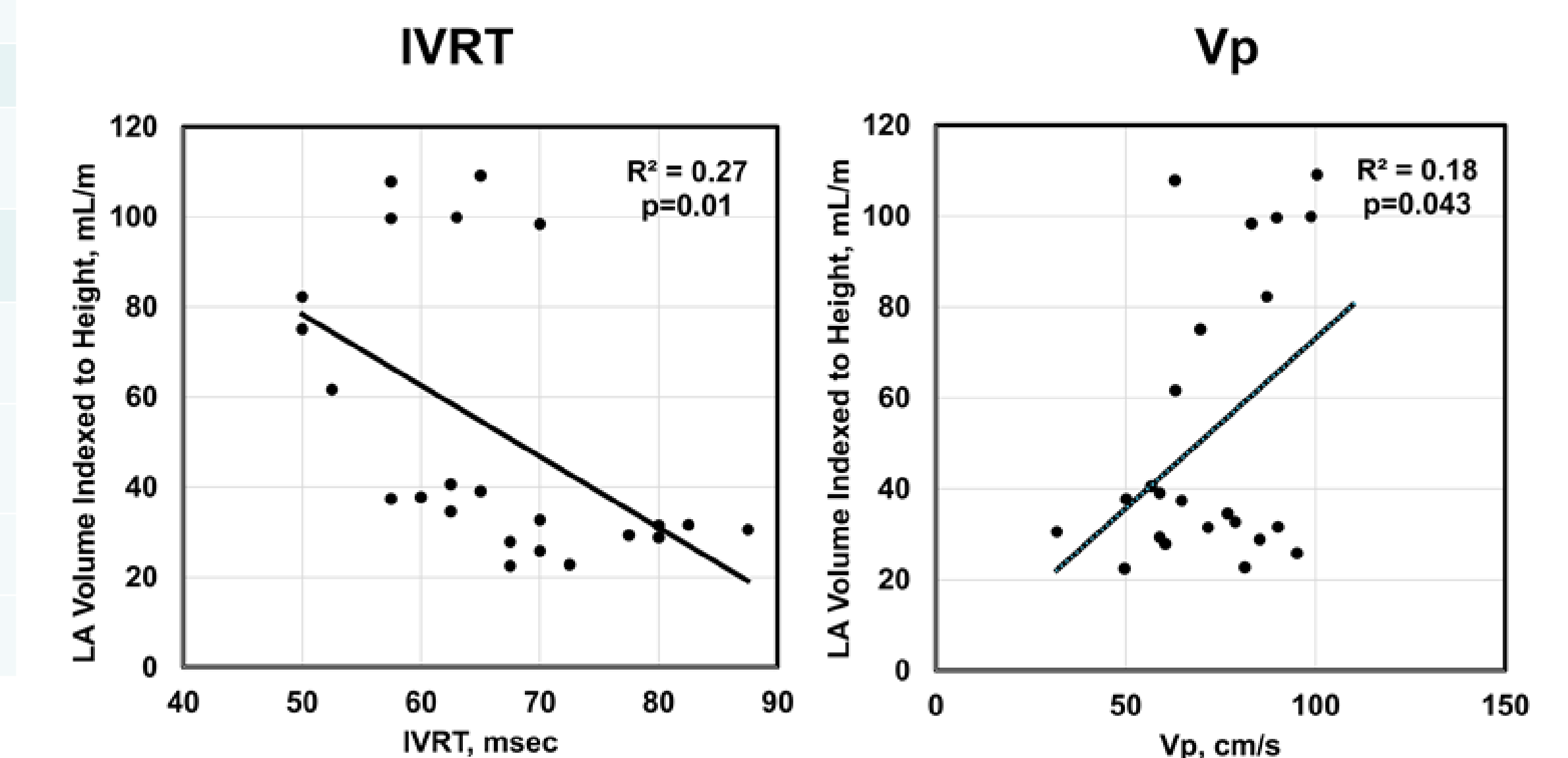
	Obese	Controls
Age (yrs)	49 ± 7	52 ± 5
BMI (kg/m ²)	37.7 ± 5.1	27.0 ± 4.1
LA EDV (mL)	61.2 ± 14.6	33.0 ± 18.7 ‡
LA Volume Indexed to Height (mL/m)	35.9 ± 8.7	19.4 ± 10.5 ‡
LV EDV mL	151.7 ± 20.4	93.8 ± 21.3 ‡
LV Volume Indexed to Height (mL/m)	88.8 ± 7.7	55.1 ± 16.7 ‡
LA EDV Indexed to LV EDV	0.41 ± 0.87	0.32 ± 0.11 *
IVRT (msec)	68 ± 11	100 ± 16 †
Vp (cm/sec)	75 ± 19	56 ± 13 †

Values are mean ± SD. * p < 0.05; † p < 0.01; ‡ p < 0.001

LA Volumes of obese subjects are nearly twice as large as non-obese controls. Larger LA volume is disproportionate to an increase in LV volume, suggesting that this effect is not related to heart size. Obese subjects also have faster Vp and IVRT, markers of diastolic dysfunction and indicative of increased LA to LV pressure gradient (preload).

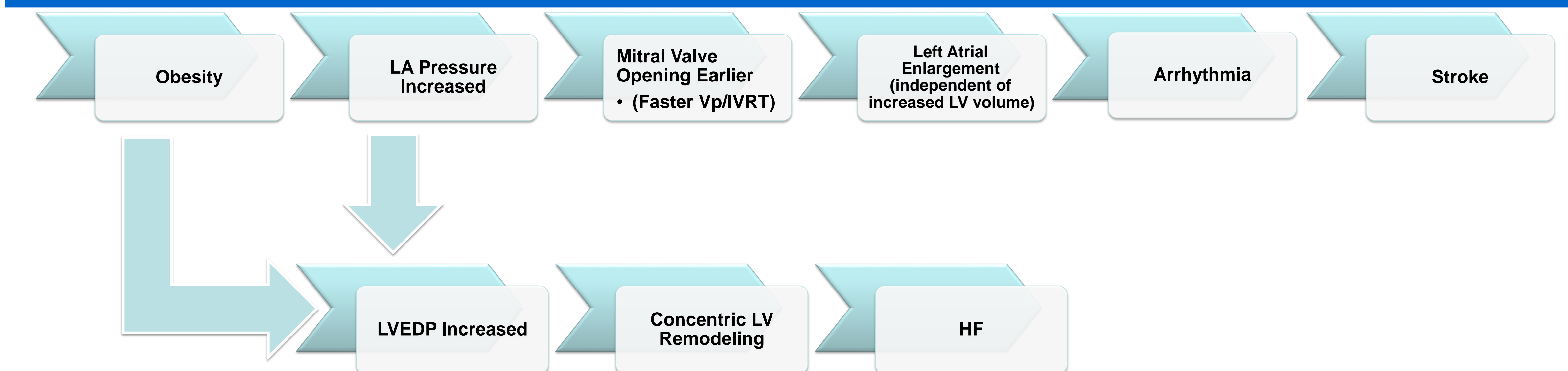
Figure 1

LA volume and Diastolic Parameters (IVRT and Vp)



Relationship between IVRT and LA volume. (Left) As IVRT shortens, LA volume significantly increases. The relationship between LA volume and propagation velocity (right) shows that as Vp increases, LA volume significantly increases.

Discussion



CONCLUSION

Obese patients have larger LA volumes independent of their heart size and markers of elevated LA filling pressures (IVRT and Vp) compared to age matched controls. LAE in obesity may be driven by sub-clinical alterations in diastolic function and left atrial filling pressures.