From Zzz's to MRI's, Echo's and CT's: Advanced Cardiac Imaging in OSA and OHS

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Disclosures:

- None relevant

- I work for Riverside Regional Medical Center as a Cardiac Imager
- I am a Visiting Professor of Kinesiology at UVA

Question for MOC

- A 60 year old male with PMH of diabetes, hypertension, BMI of 28 and hyperlipidemia all of which are currently diet controlled presents for evaluation of OSA. The patient's family attests to daytime somnolence, witnessed apneic episodes and some memory problems for which the patient has insight into. Patient otherwise has been able to continue exercising including daily 30 minute moderate intensity runs. Home study demonstrates an AHI of 4.5. A recent echocardiogram demonstrates normal LVEF, moderate diastolic dysfunction and decreased right ventricular systolic function with a PASP of 45mmHg. Physical exam demonstrates no edema, JVD or rales. What is the next best management choice?
  - A. Cardiac MRI to evaluate for presumed heart failure and Holter monitor for arrhythmia
  - B. Coronary CTA to evaluate for obstructive coronary disease and initiating GLP1 agonist for presumed OSA
  - C. Repeat in-lab PSG and Coronary artery calcium score
  - D. Refer to Cardiology

Why Would I Care about OSA and CPAP?

ESC European Society of Cardiology

Congresses & Events · Journals · Guidelines · Education · Research

Dr Azarbarzin said: "Our findings suggest that CPAP may offer long-term cardiovascular benefit in people with high-risk OSA but may have unintended harmful effects in those without high-risk OSA.

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ESC Press Office

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Treatment for sleep apnoea is good for the heart in some patients but bad for others

06 Aug 2025

Topic(s): *Sleep Disorders; Risk Factors and Prevention;*

A treatment commonly used for obstructive sleep apnoea (OSA), lowers the risk of serious cardiovascular events in some patients but not others, according to research published in the European Heart Journal [1] today.

People with OSA often snore loudly, their breathing starts and stops during the night, and they may wake up several times. Not only does this cause tiredness, but it can also increase the risk of high blood pressure, stroke and heart disease.

Key Question

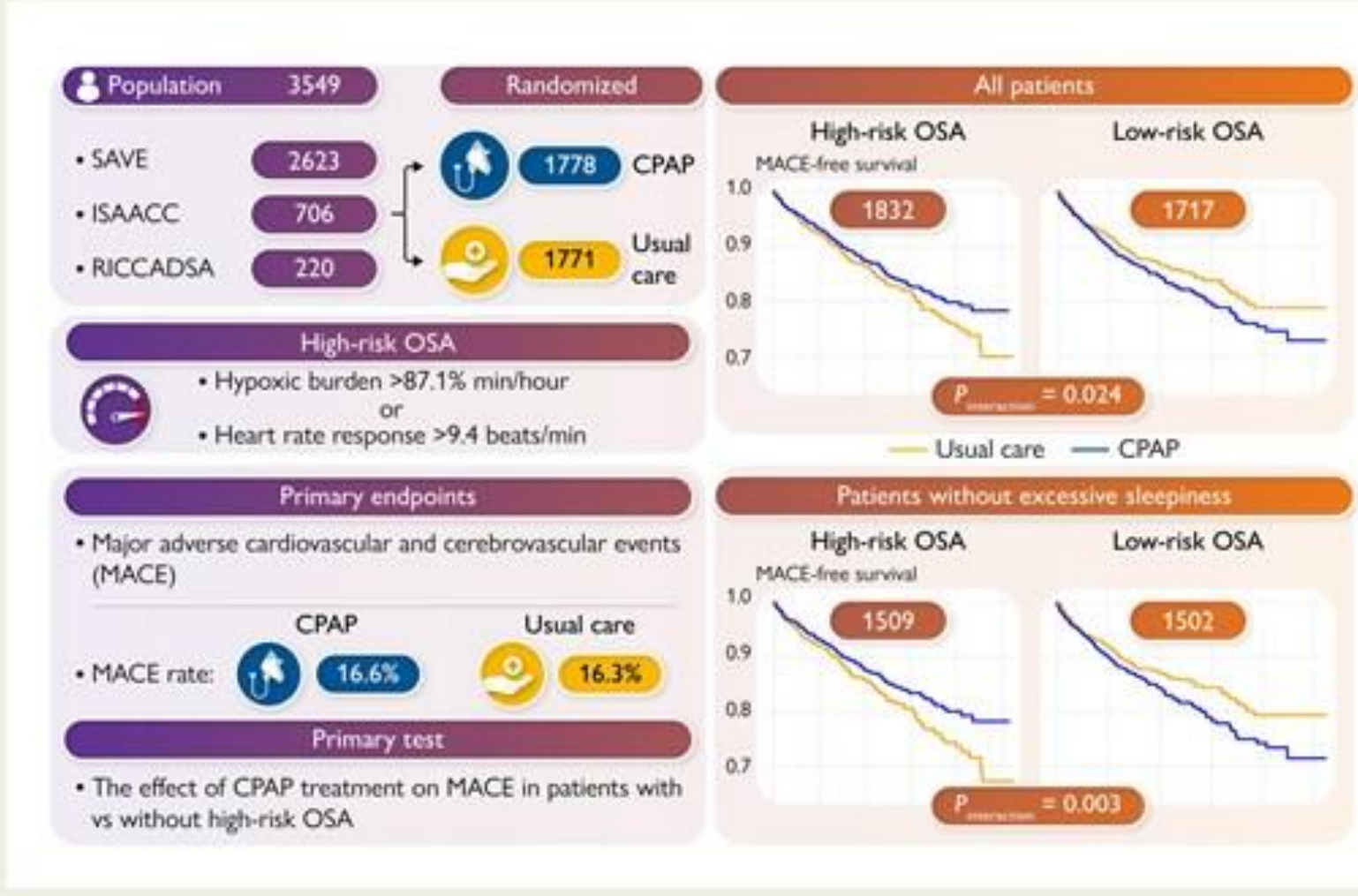
Randomized trials of continuous positive airway pressure (CPAP) have failed to show benefit for secondary cardiovascular outcome prevention. Does CPAP confer cardiovascular benefit preferentially in patients with high-risk obstructive sleep apnoea (OSA)?

Key Finding

In this pooled analysis of three trials, the benefit of CPAP in reducing major adverse cardiovascular and cerebrovascular events was greater in patients with high-risk OSA compared to those with low-risk OSA. In low-risk OSA, CPAP may increase cardiovascular risk.

Take Home Message

Assessment of OSA phenotypes may guide the indication for CPAP to reduce cardiovascular risk.



How Do I Approach Research?

Why Most Published Research Findings Are False

John P. A. Ioannidis

Published: August 30, 2005 • https://doi.org/10.1371/journal.pmed.0020124

Article | Authors | Metrics | Comments | Media Coverage

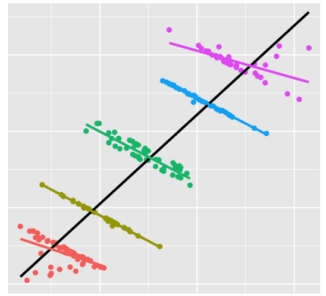
Correction

25 Aug 2022: Ioannidis JPA (2022) Correction: Why Most Published Research Findings Are False. PLOS Medicine 19(8): e1004085. https://doi.org/10.1371/journal.pmed.1004085 | **View correction**

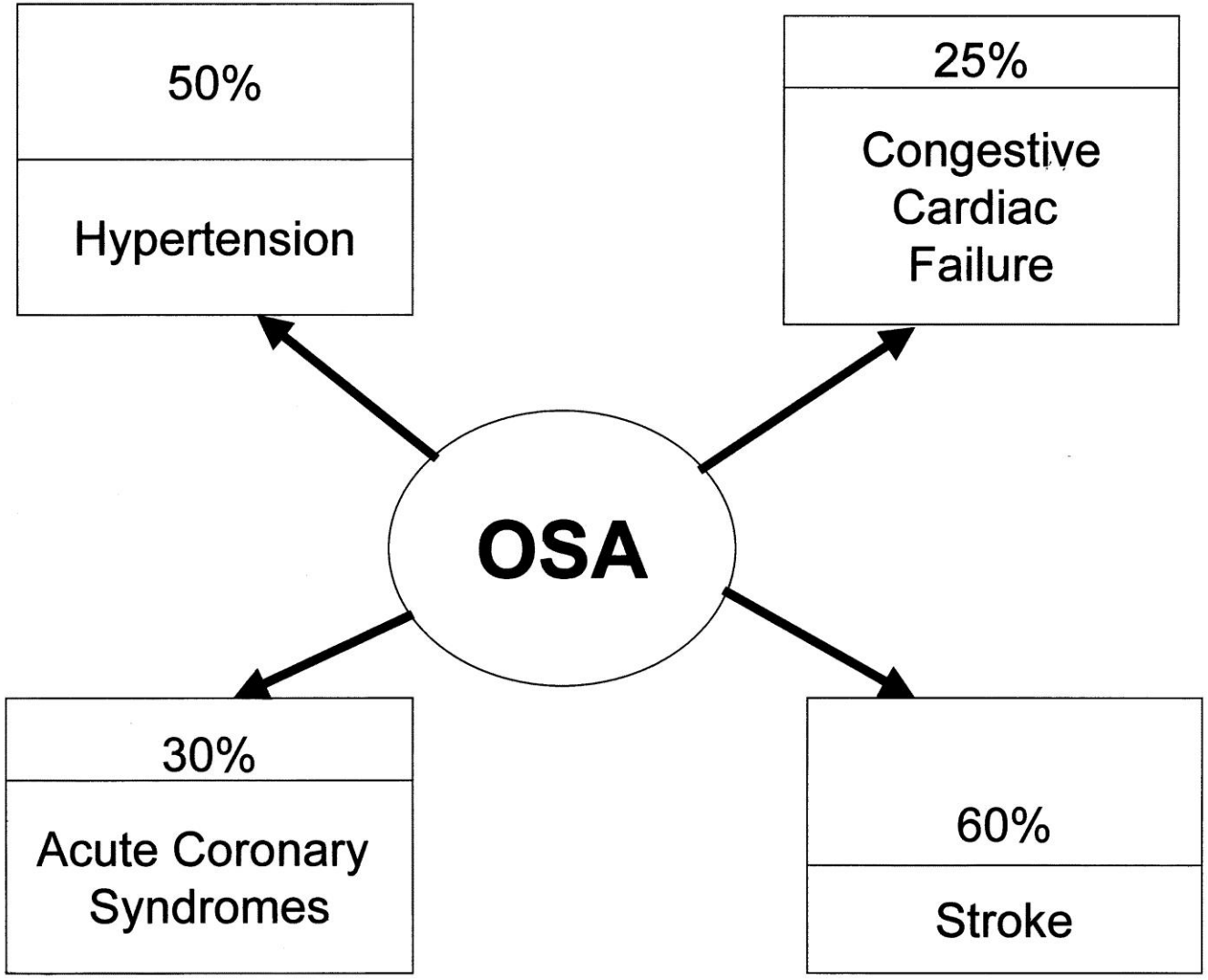
Abstract

Summary

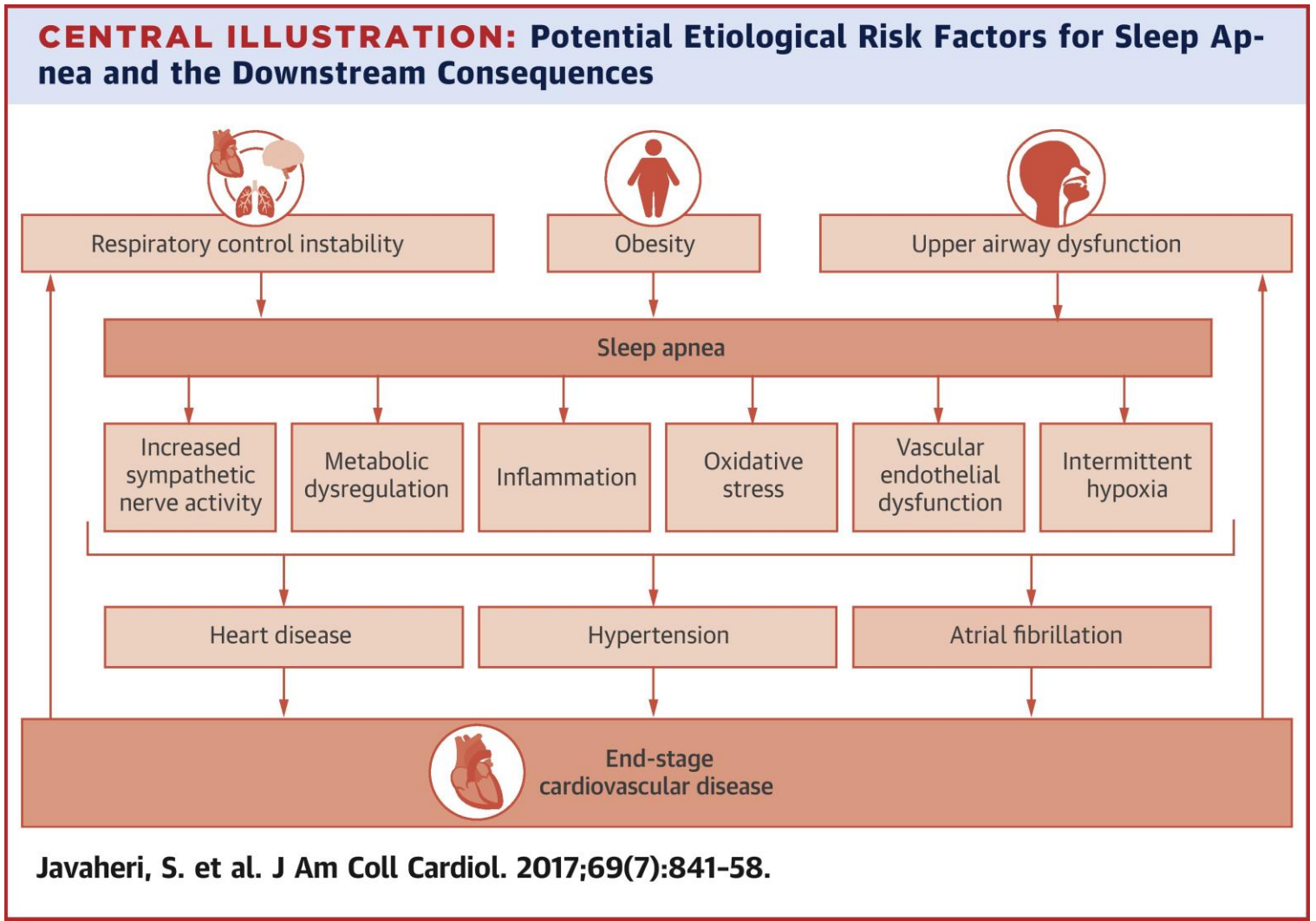
There is increasing concern that most current published research findings are false. The probability that a research claim is true may depend on study power and bias, the number of other studies on the same question, and, importantly, the ratio of true to no relationships among the relationships probed in each scientific field. In this framework, a research finding is less likely to be true when the studies conducted in a field are smaller; when effect sizes are smaller; when there is a greater number and lesser preselection of tested relationships; where there is greater flexibility in designs, definitions, outcomes, and analytical modes; when there is greater financial and other interest and prejudice; and when more teams are involved in a scientific field in chase of statistical significance. Simulations show that for most study designs and settings, it is more likely for a research claim to be false than true. Moreover, for many current scientific fields, claimed research findings may often be simply accurate measures of the prevailing bias. In this essay, I discuss the implications of these problems for the conduct and interpretation of research.



Why ELSE would Cardiologists care?



How does Sleep Apnea affect the Heart



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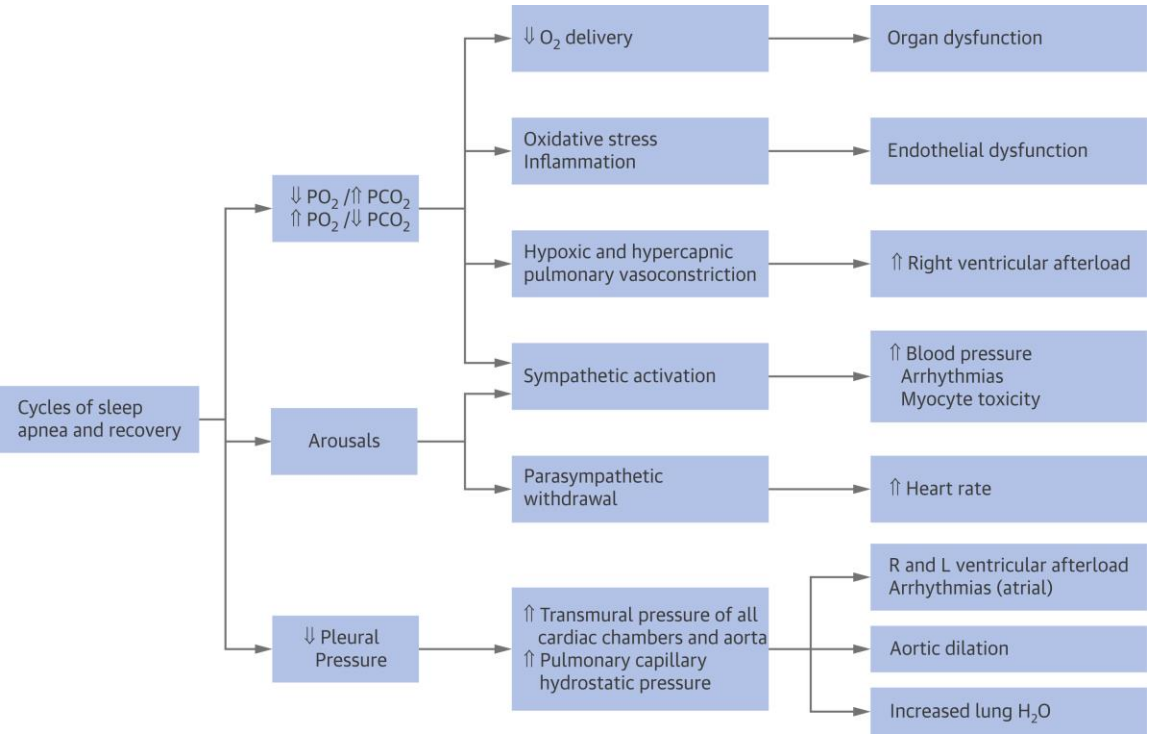

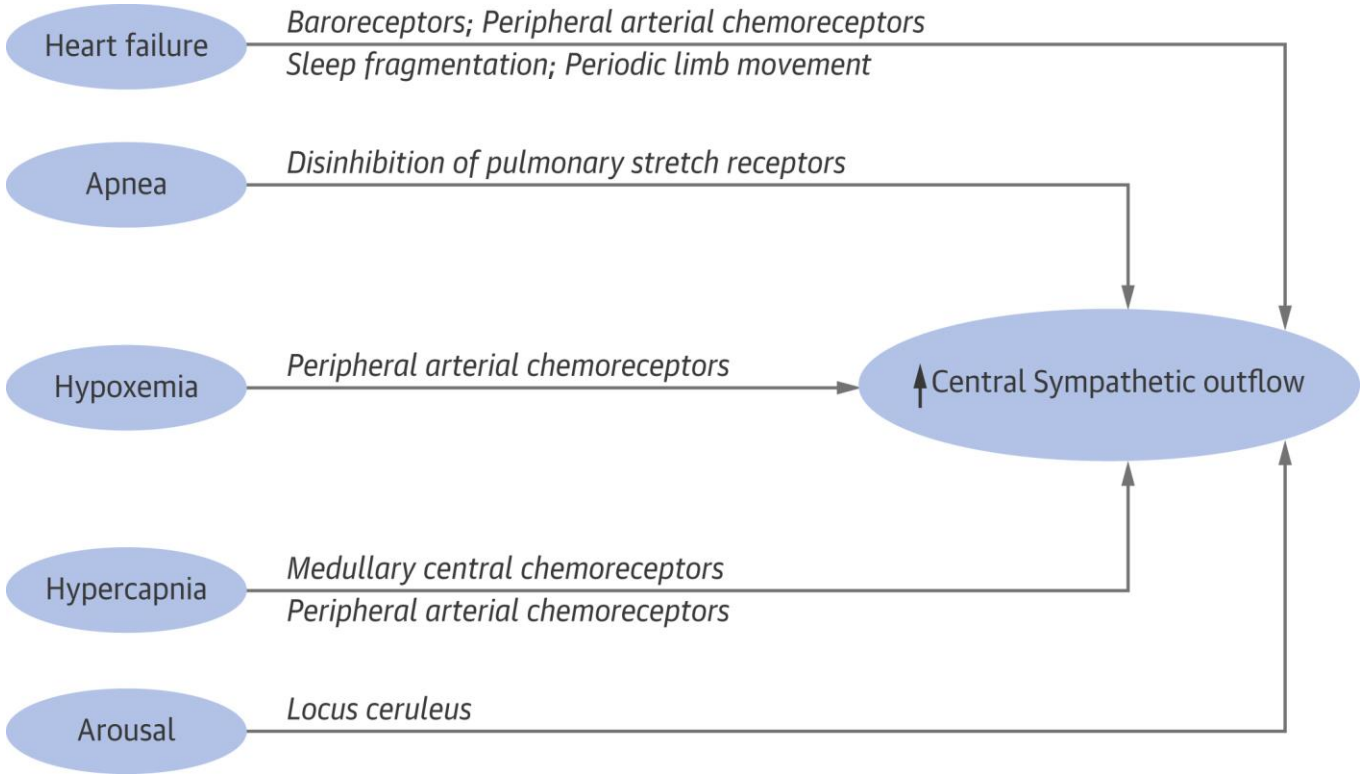

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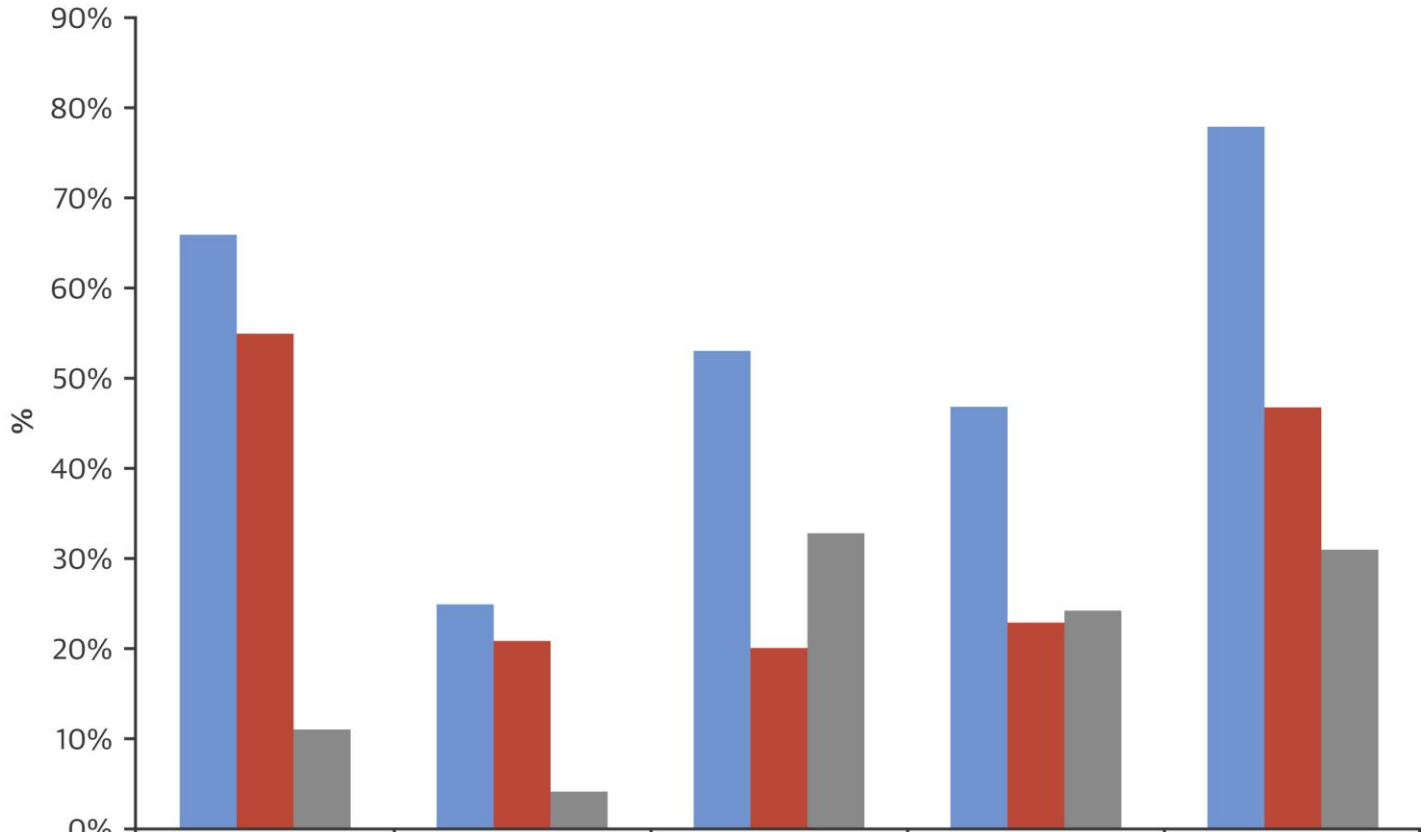
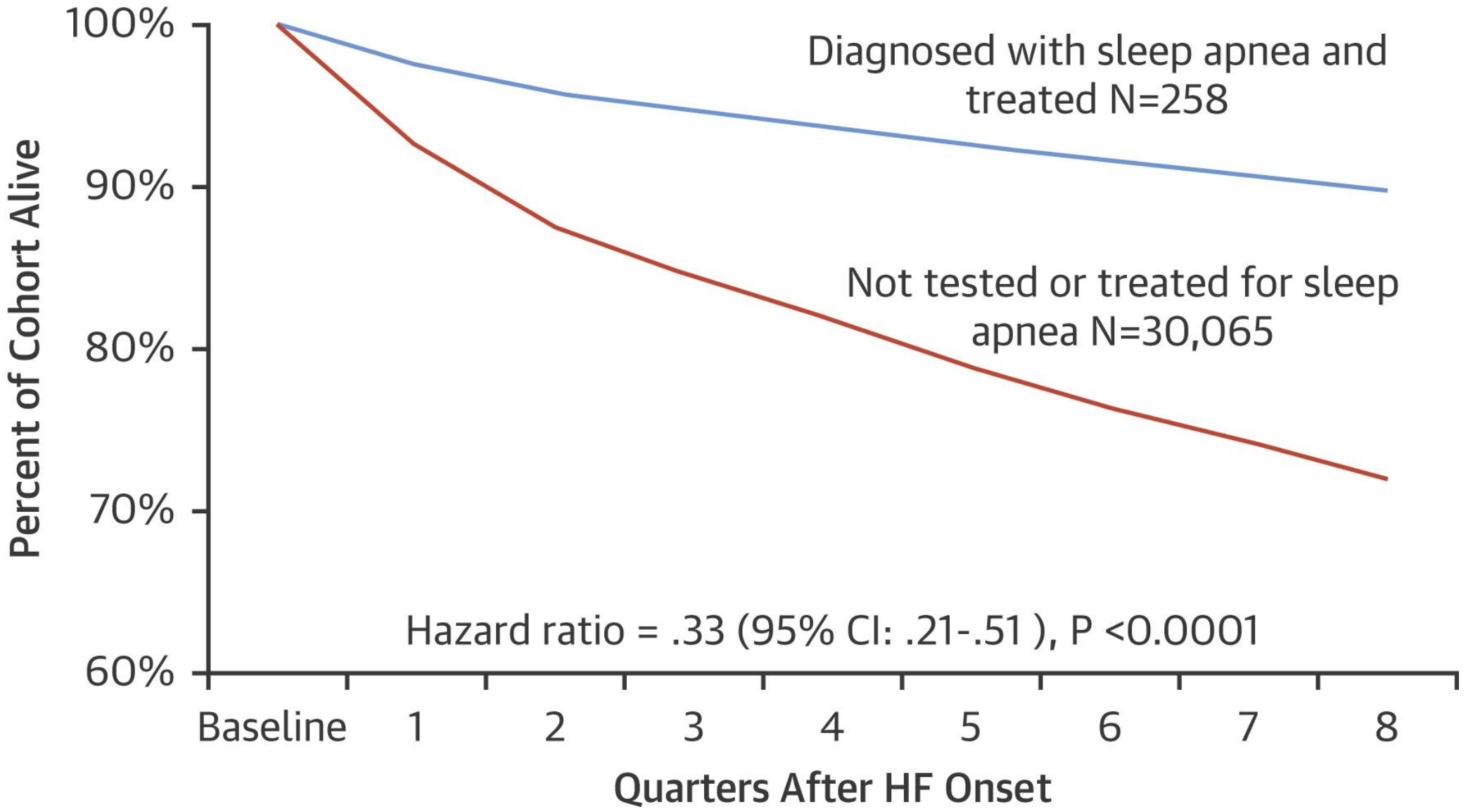


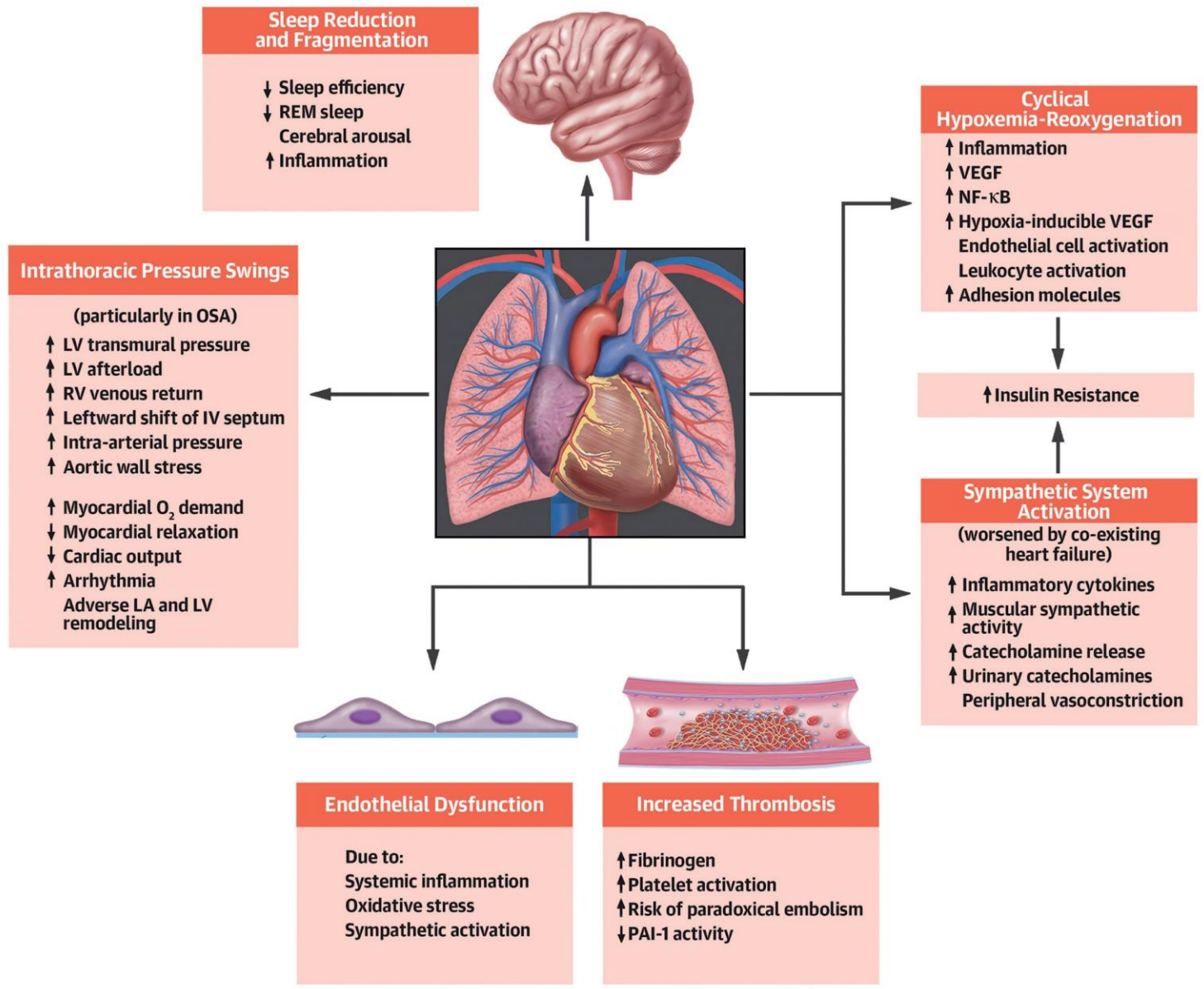
	LVSD Asymptomatic	LVDD Asymptomatic	HFrEF	HFpEF	ADHF
AHI > 15/h	66%	25%	53%	47%	78%
OSA	55%	21%	20%	23%	47%
CSA	11%	4%	33%	24%	31%

AHI > 15/h OSA CSA

How CPAP affects HF with OSA

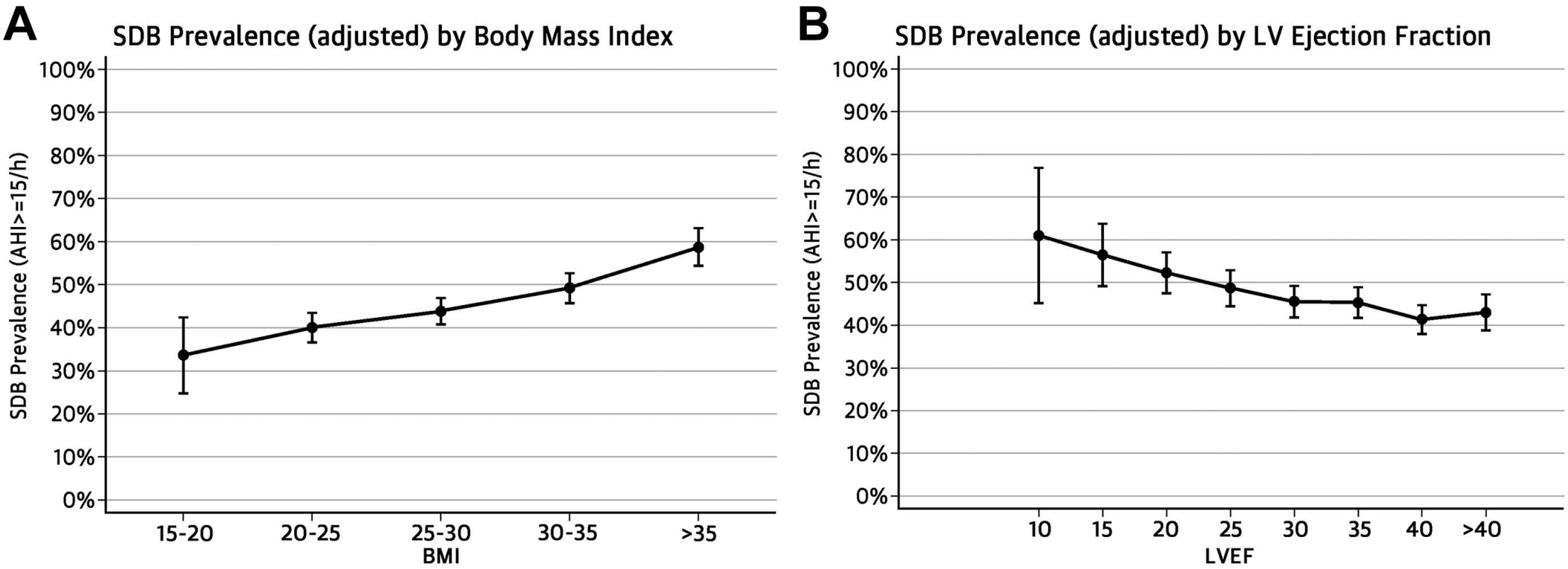


CENTRAL ILLUSTRATION: Pathophysiological Abnormalities in Sleep Disordered Breathing

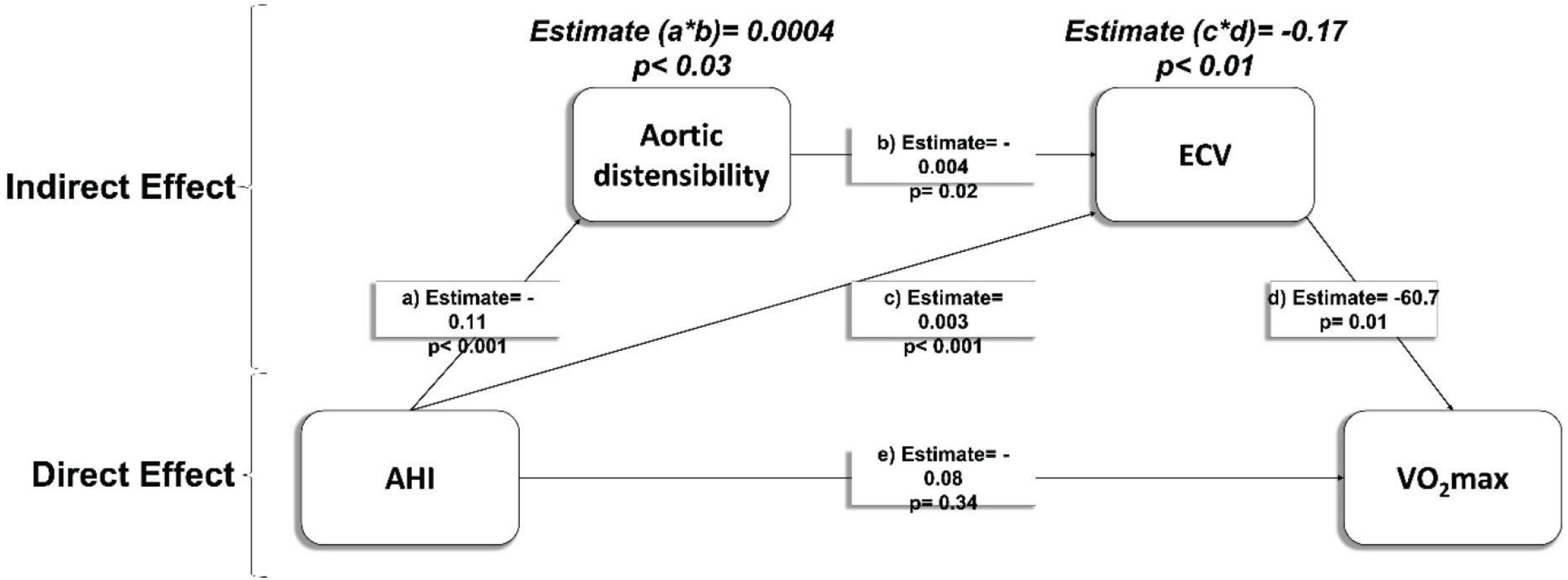


Cowie, M.R. et al. J Am Coll Cardiol. 2021;78(6):608-624.

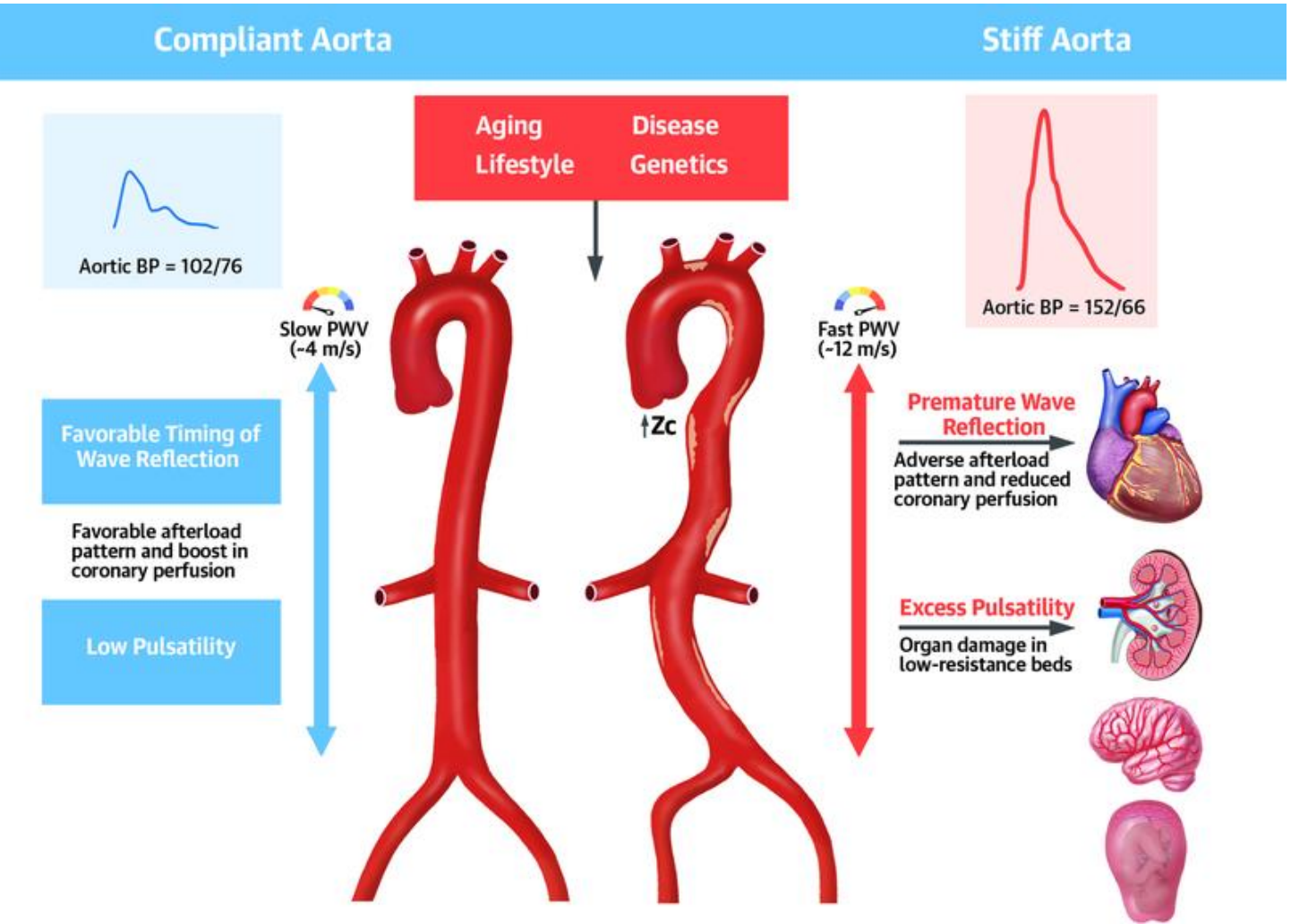
SDB by BMI and EF

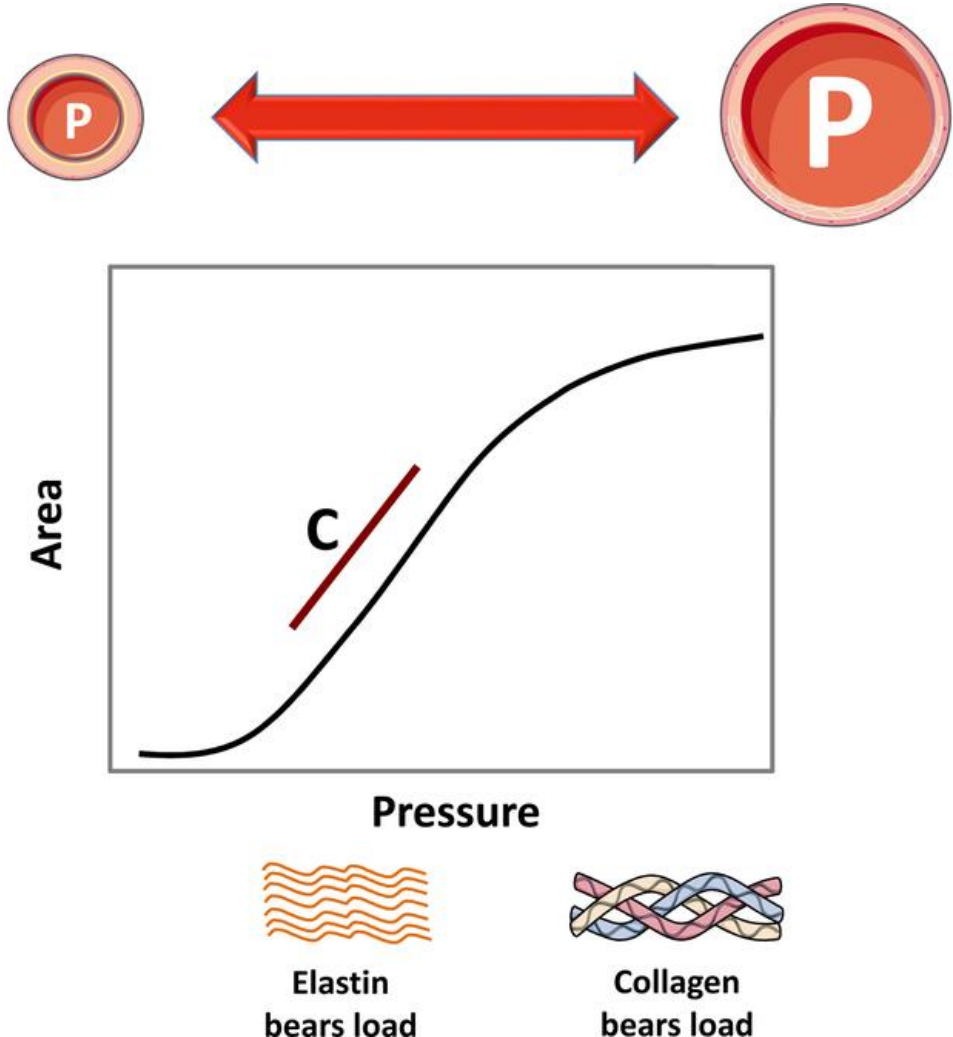


Cardiorespiratory Fitness Among OSA & DM

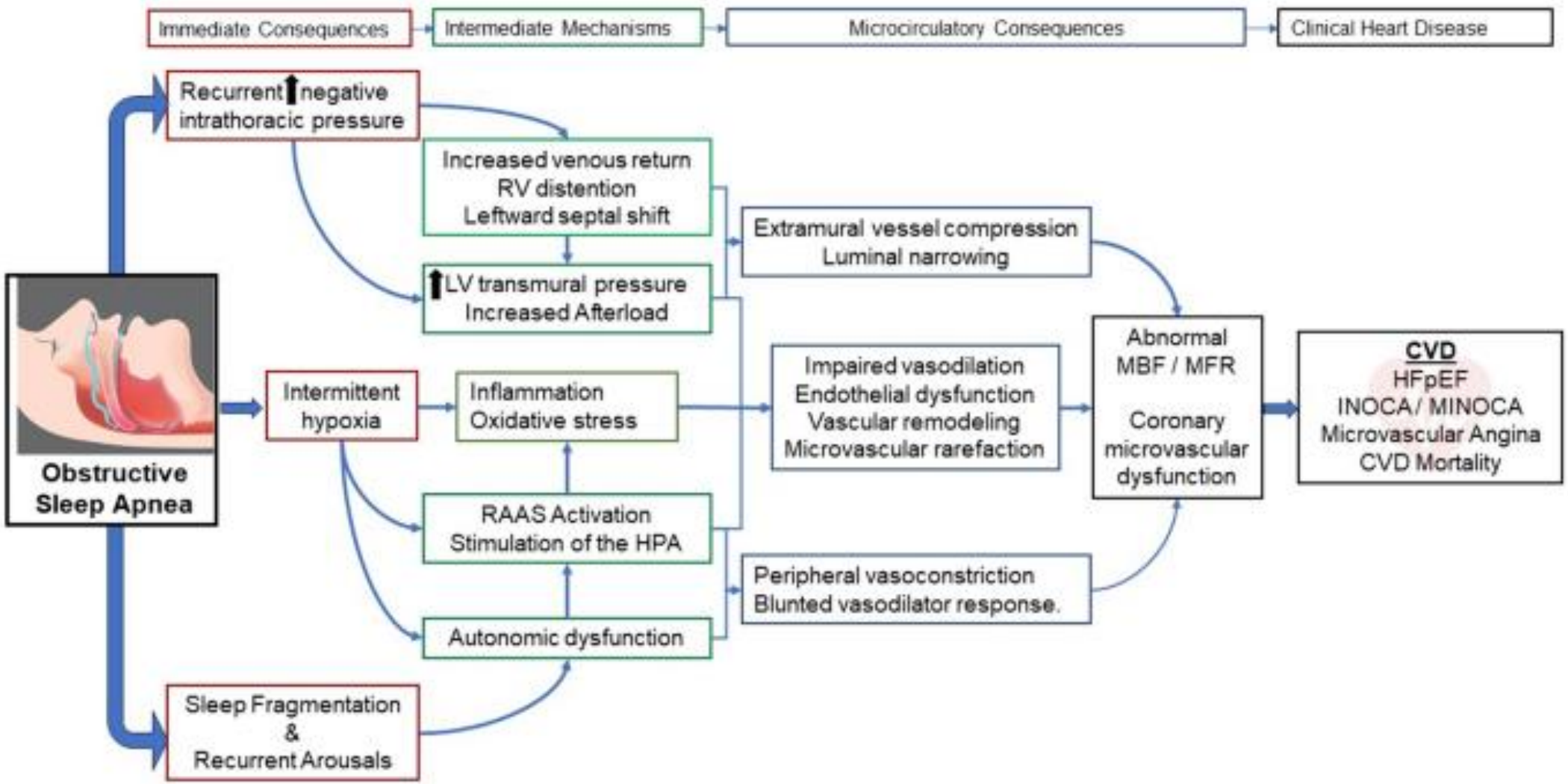


Aortic Distensibility and CVD

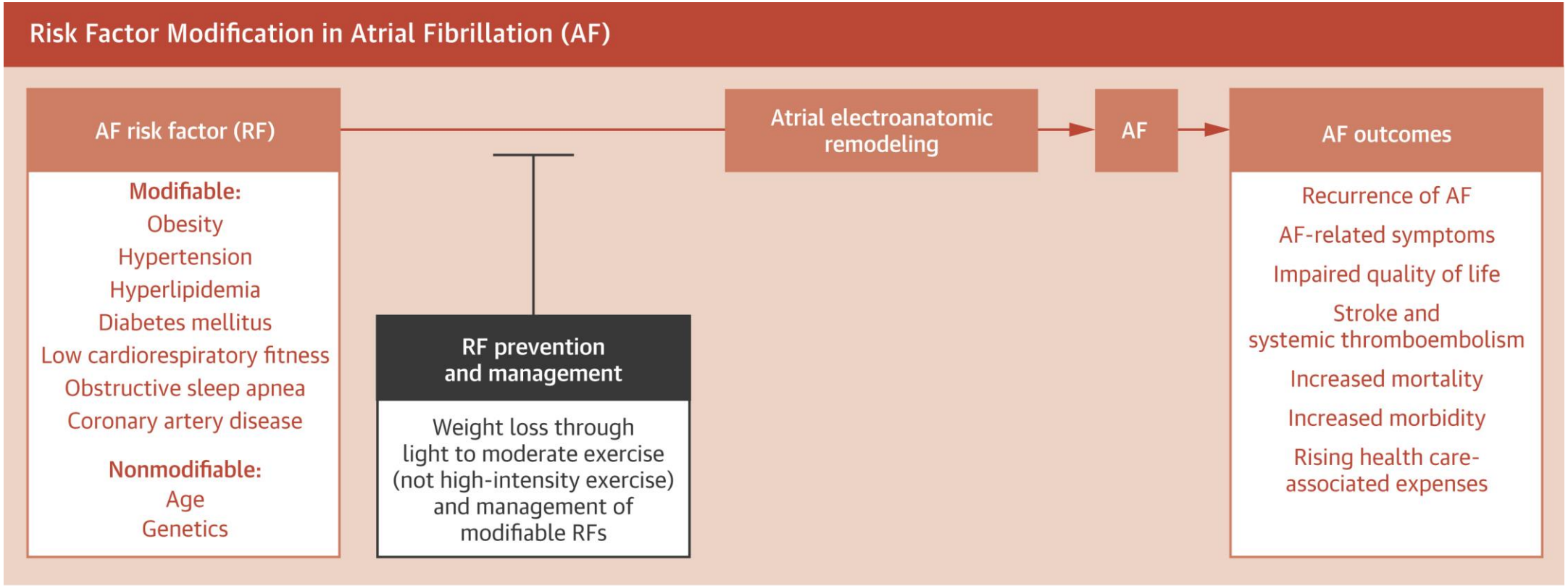




INOCA, CMD and OSA

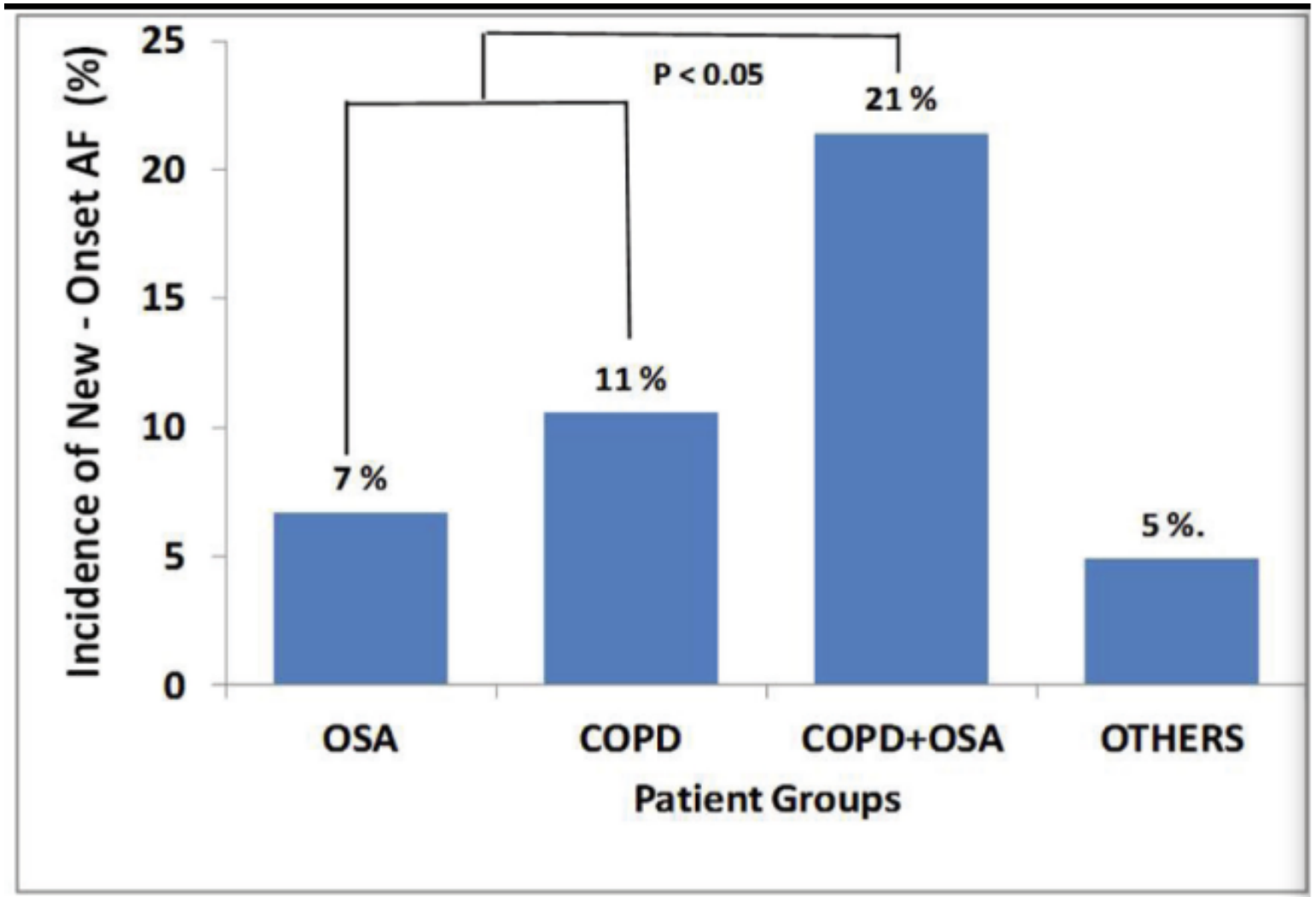


Afib and OSA

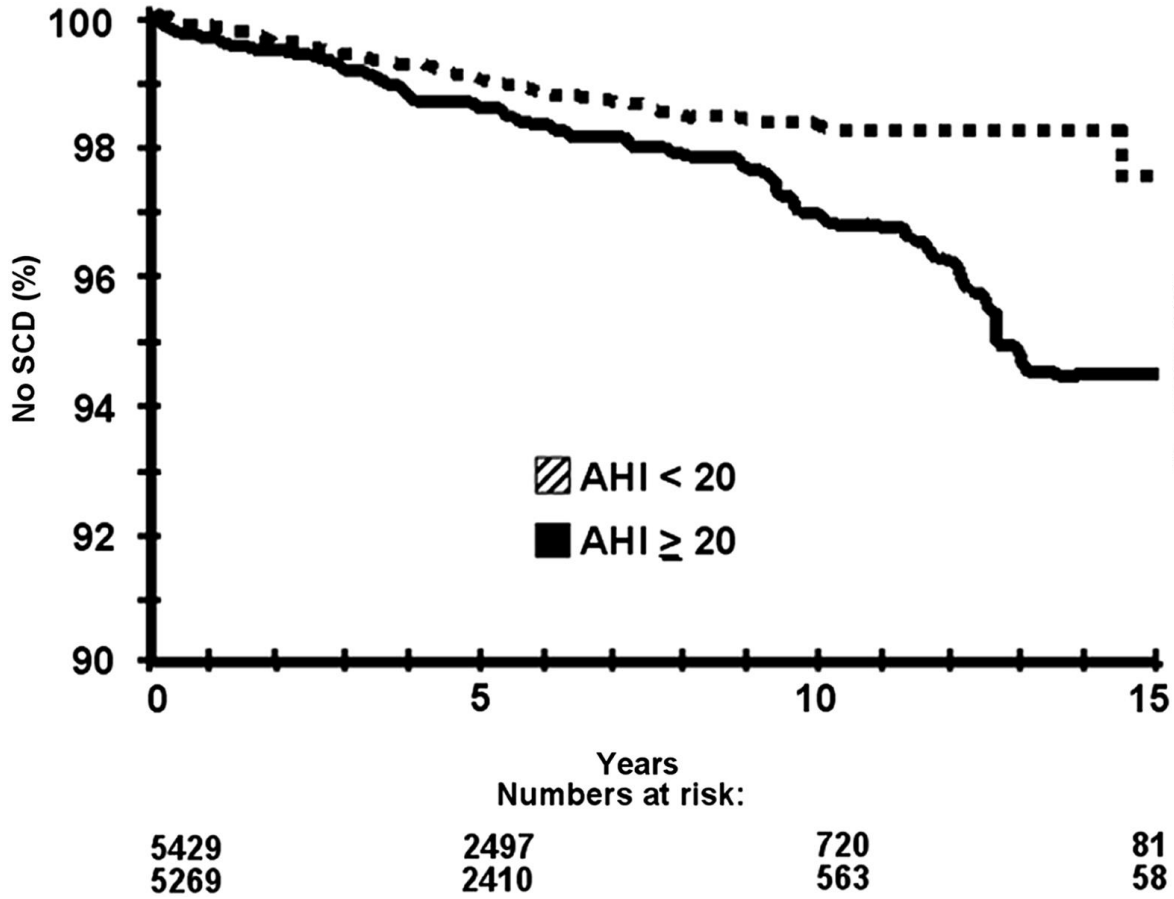


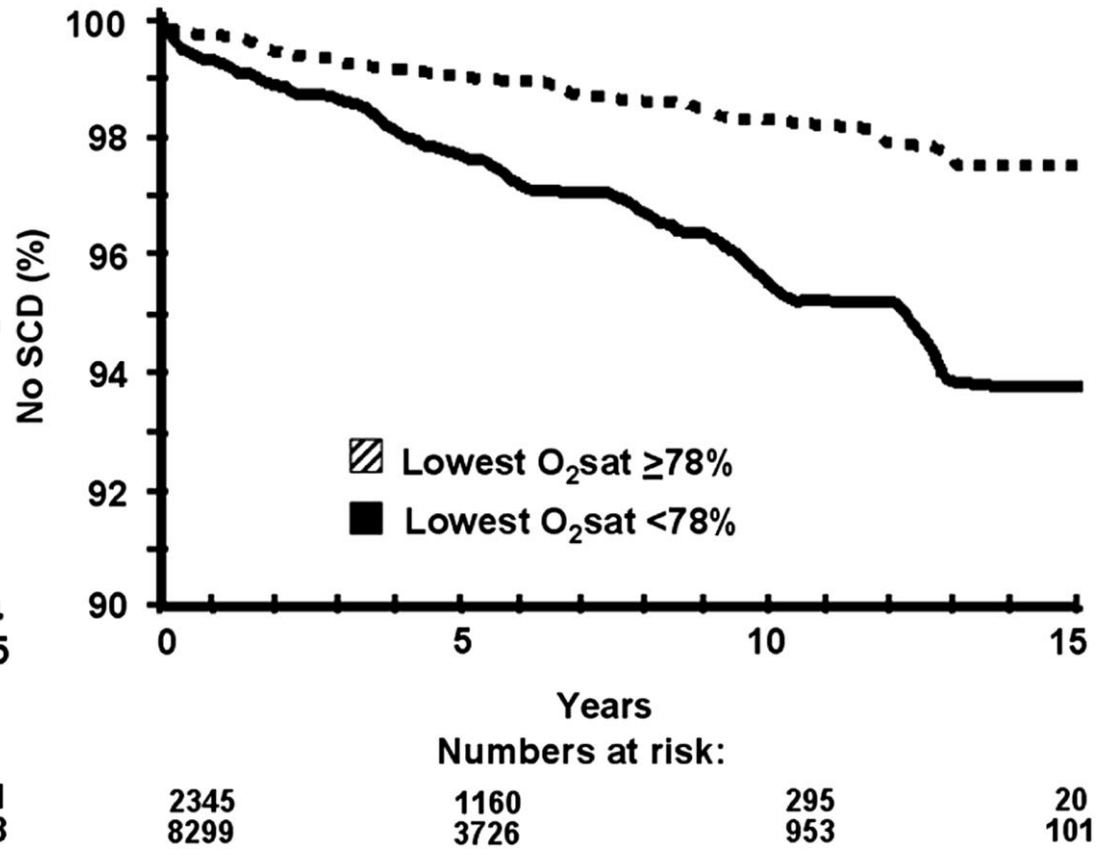
Miller, J.D. et al. J Am Coll Cardiol. 2015; 66(25):2899-906.

Risk of new AF among OSA/COPD

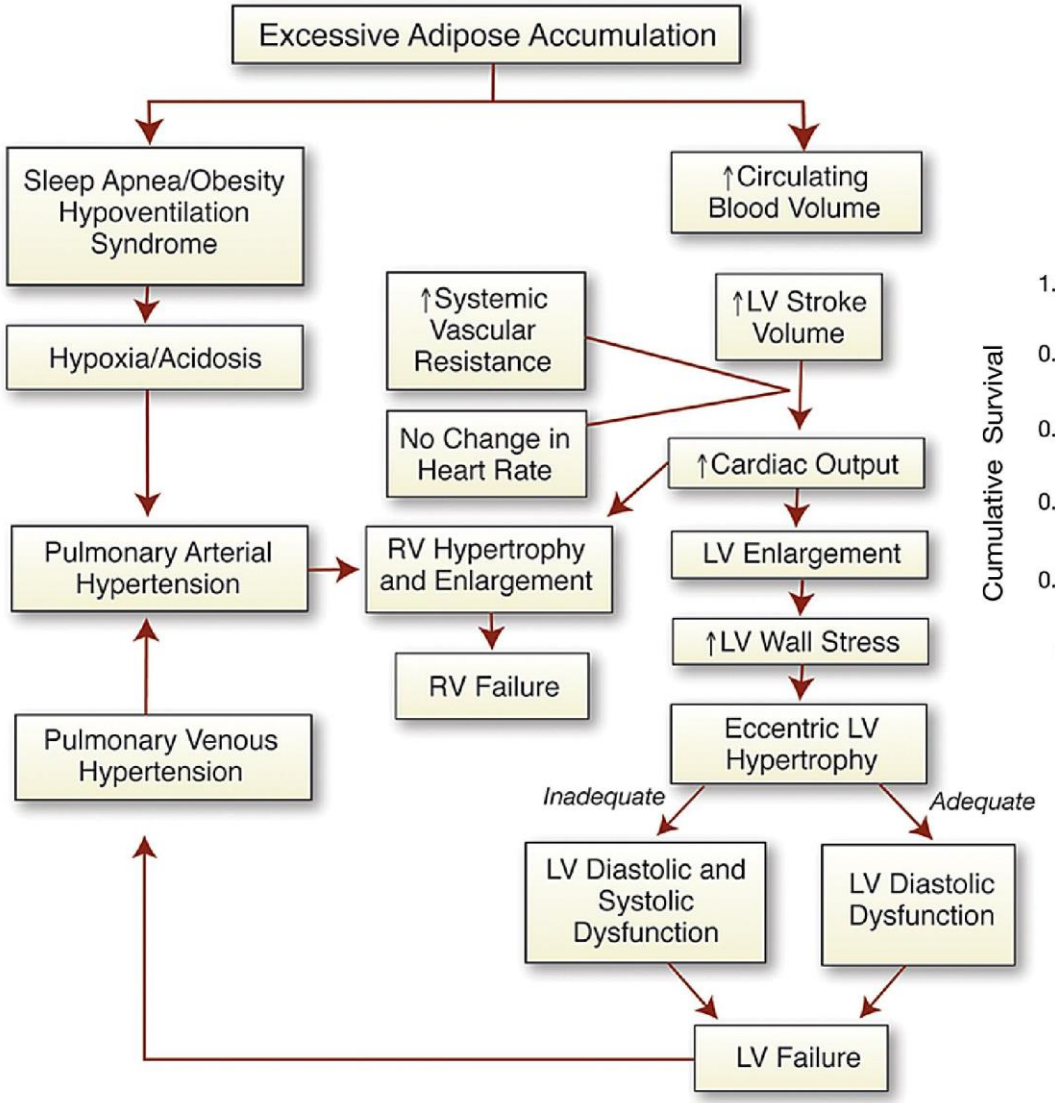


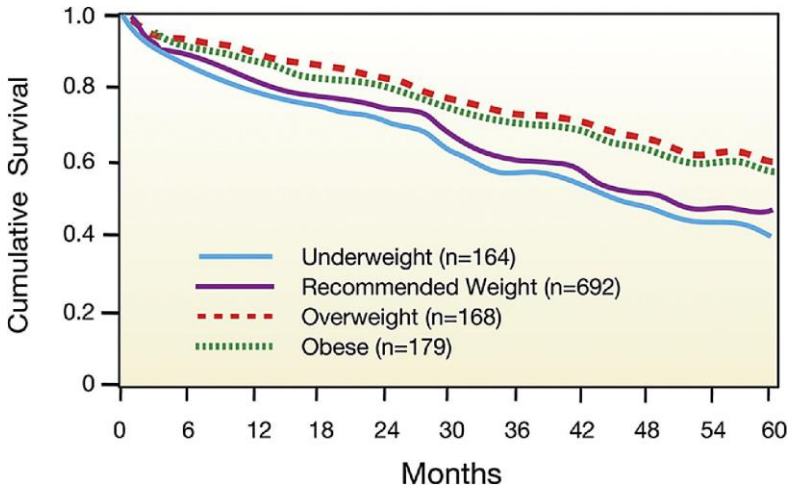
OSA and SCD

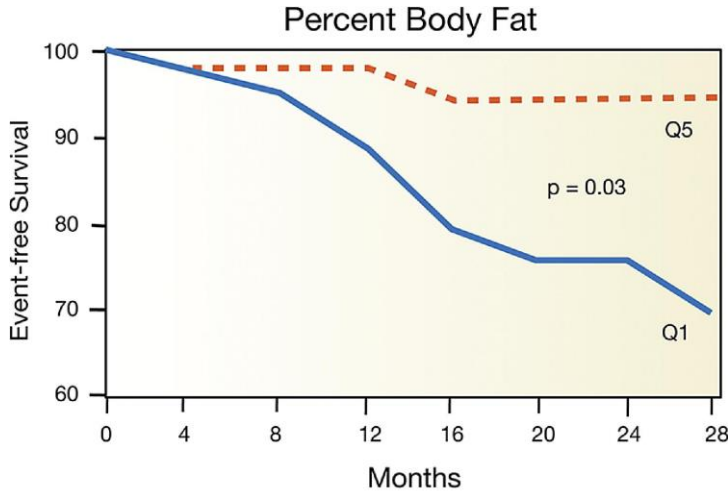

100
98
96
94
92
90
No SCD (%)
AHI < 20
AHI ≥ 20
0
5
10
15
Years
Numbers at risk:
5429
5269
2497
2410
720
563
81
58

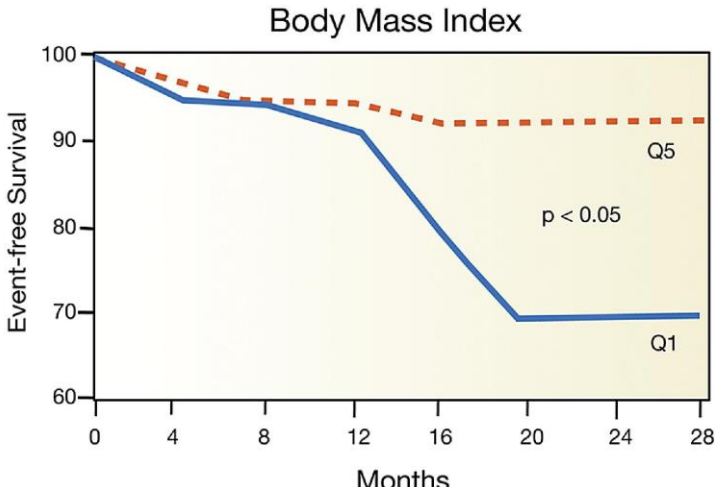

100
98
96
94
92
90
No SCD (%)
Lowest O_2sat ≥78%
Lowest O_2sat <78%
0
5
10
15
Years
Numbers at risk:
2345
8299
1160
3726
295
953
20
101

OHS and CVD

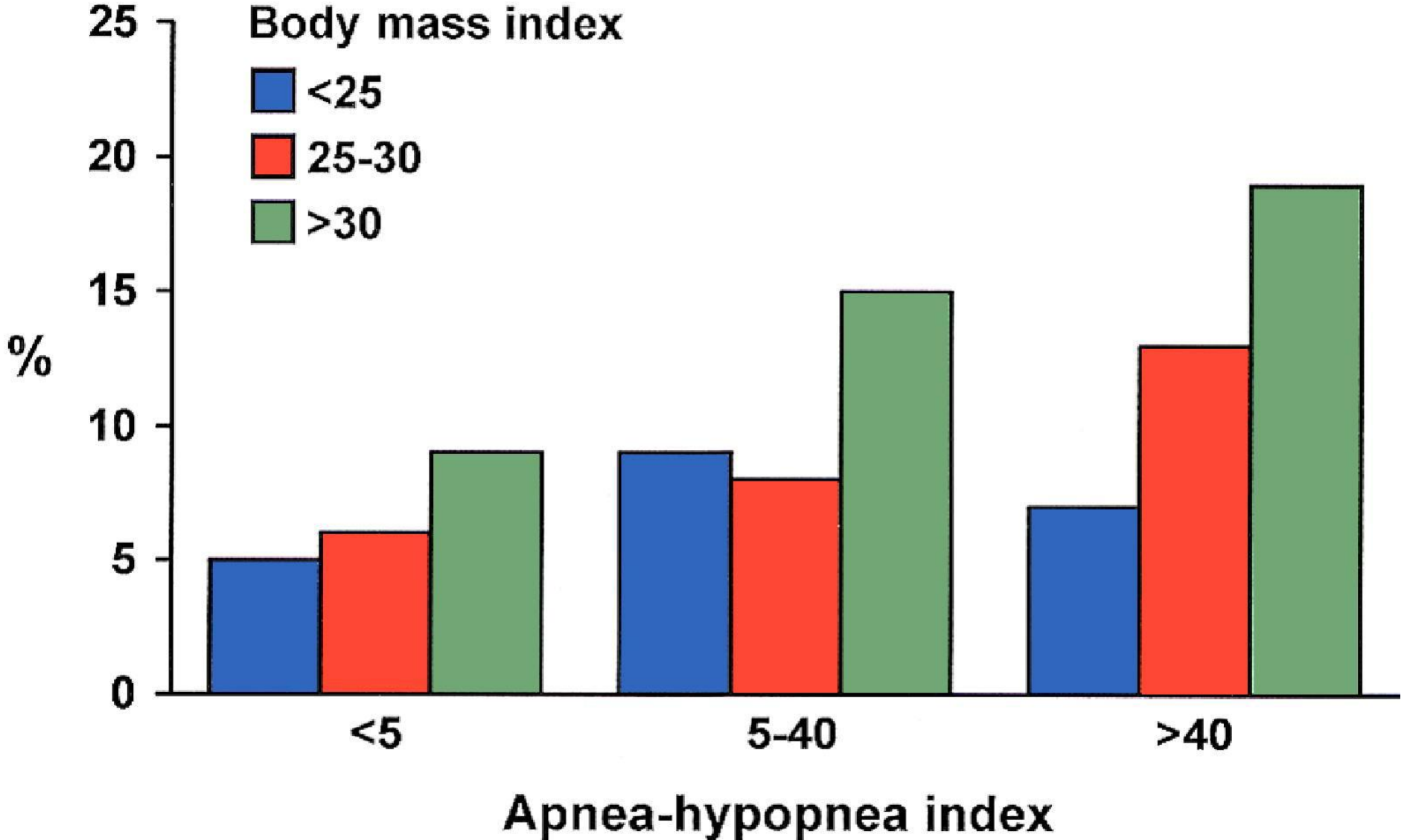

Excessive Adipose Accumulation
Sleep Apnea/Obesity Hypoventilation Syndrome
↑Circulating Blood Volume
Hypoxia/Acidosis
↑Systemic Vascular Resistance
↑LV Stroke Volume
No Change in Heart Rate
↑Cardiac Output
Pulmonary Arterial Hypertension
RV Hypertrophy and Enlargement
LV Enlargement
RV Failure
↑LV Wall Stress
Pulmonary Venous Hypertension
Eccentric LV Hypertrophy
Inadequate
Adequate
LV Diastolic and Systolic Dysfunction
LV Diastolic Dysfunction
LV Failure


Cumulative Survival
1.0
0.8
0.6
0.4
0.2
0
0
6
12
18
24
30
36
42
48
54
60
Months
Underweight (n=164)
Recommended Weight (n=692)
Overweight (n=168)
Obese (n=179)

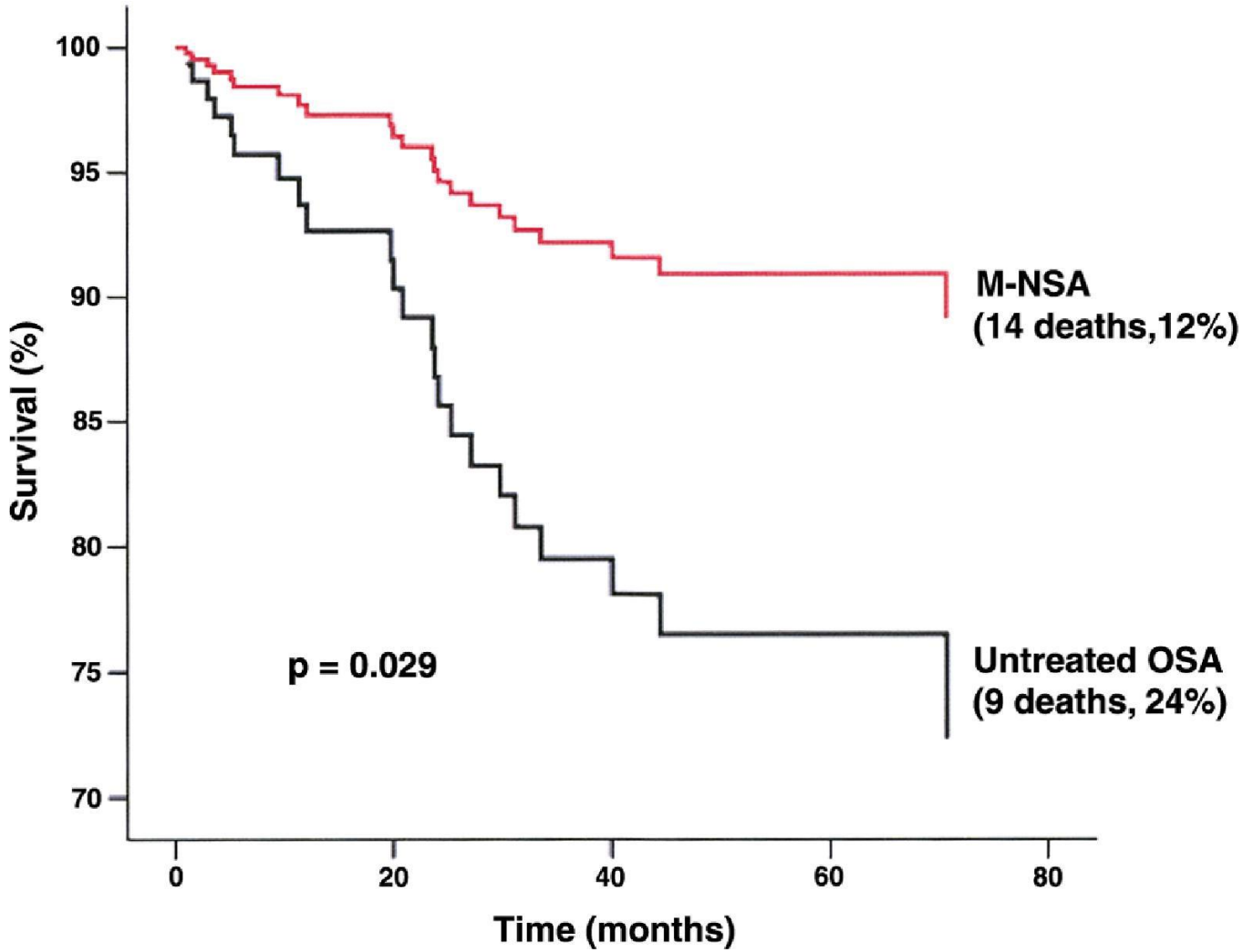

Percent Body Fat
Event-free Survival
100
90
80
70
60
0
4
8
12
16
20
24
28
Months
Q5
p = 0.03
Q1


Body Mass Index
Event-free Survival
100
90
80
70
60
0
4
8
12
16
20
24
28
Months
Q5
p < 0.05
Q1

Incidence of Afib by OSA severity and BMI



Impact of OSA on mortality in HF



CPAP for OSA receiving CABG

Table 1. Primary and Secondary Outcomes in Patients with OSA with CPAP undergoing CABG

	Mortality	LOS (Days)	Total Healthcare Charges (US$)	Total Hospital Cost (US$)
Crude Odds Ratio (P value)				
OSA with CPAP and CABG	0.47 (P: 0.21)	0.04 (P:0.90)	5, 752.49 (P: 0.68)	4,413.08 (P:0.046)
Adjusted Odds Ratio (P value)				
OSA with CPAP and CABG	0.38 (0.11)	-0.72 (**P:< 0.01**)	-11, 364.68 (P:0.38)	-21.64 (P: 0.99)

Table 2. Unadjusted and Adjusted OR for CABG complications for patients with OSA and CPAP

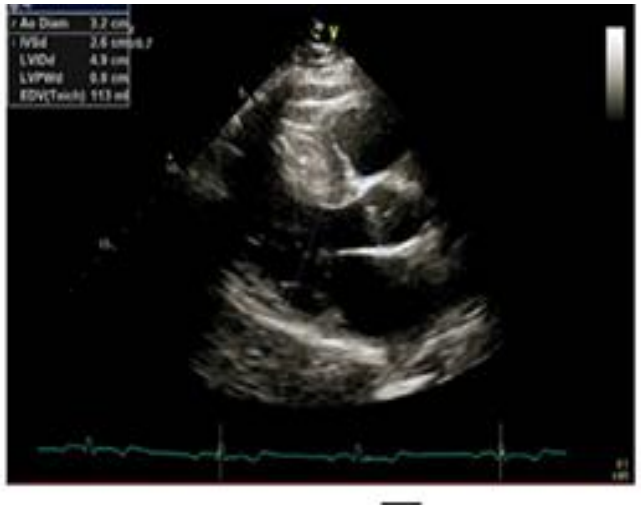
	Unadjusted OR (OR)	P value	Adjusted OR (aOR)	P value
Complications				
Endotracheal Intubation	1.00	0.99	0.63	0.07
Mechanical Ventilation <24 hours (%)	1.19	0.63	1.06	0.87
Mechanical Ventilation 24 – 96 hours	1.15	0.64	0.84	0.60
Mechanical Ventilation >96 hours	0.73	0.45	0.34	**0.03**
Tracheostomy	0.60	0.47	0.19	0.12
Post Procedure Shock	0.91	0.75	0.72	0.32
Post Procedure VTE	1.09	0.92	1.04	0.96

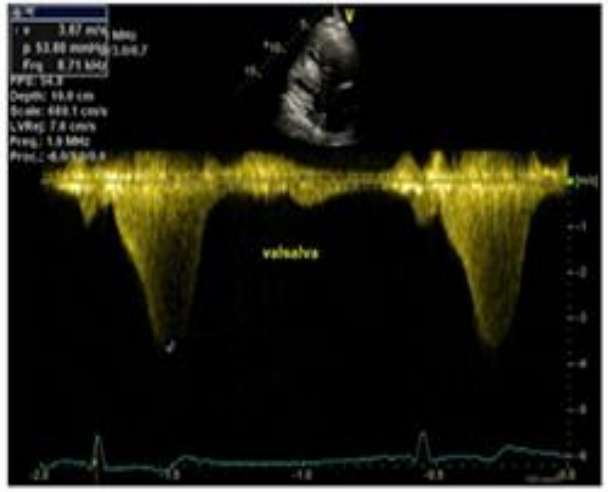
Summary So Far

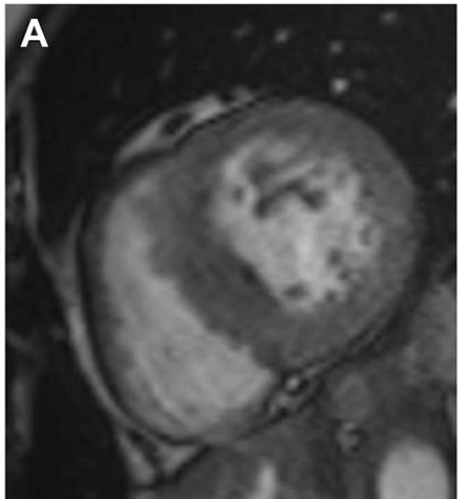
- OSA is a risk factor for most Cardiac Pathologies
- Mechanisms are likely related to sympathetic activation, increased LV and RV afterload, and ultimately changes in cardiac fibrosis
- This is likely directly associated with AHI
- CPAP probably helps but data is classically inconsistent

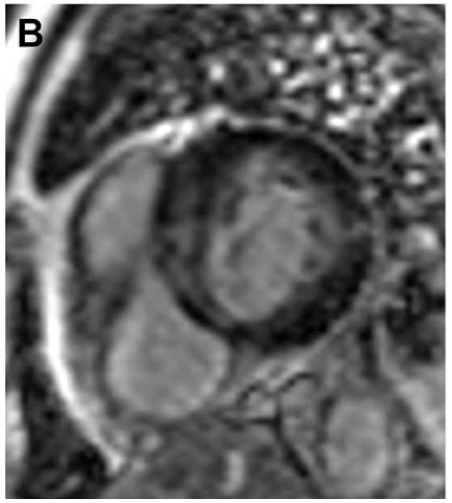
- How can we be sure? Is there a manner to detect positive or negative effects of cardiac pathologies?

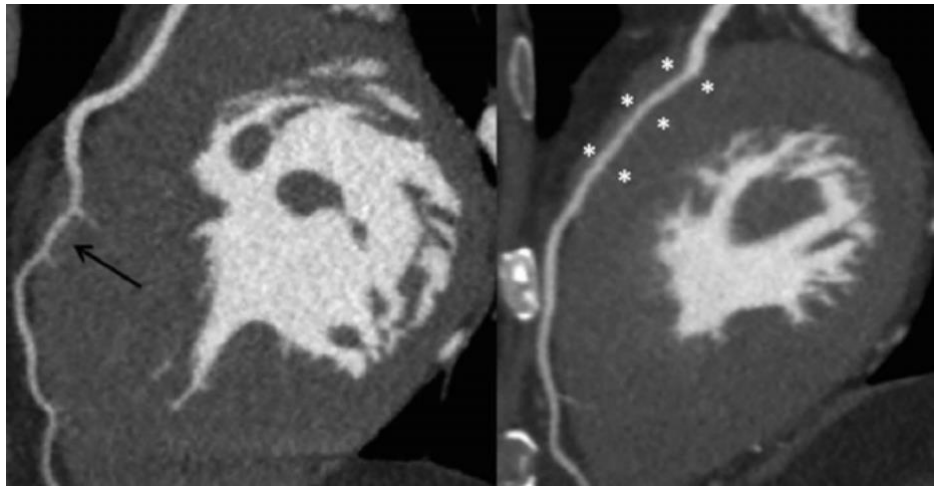
Advanced Cardiac Imaging and OSA/OHS





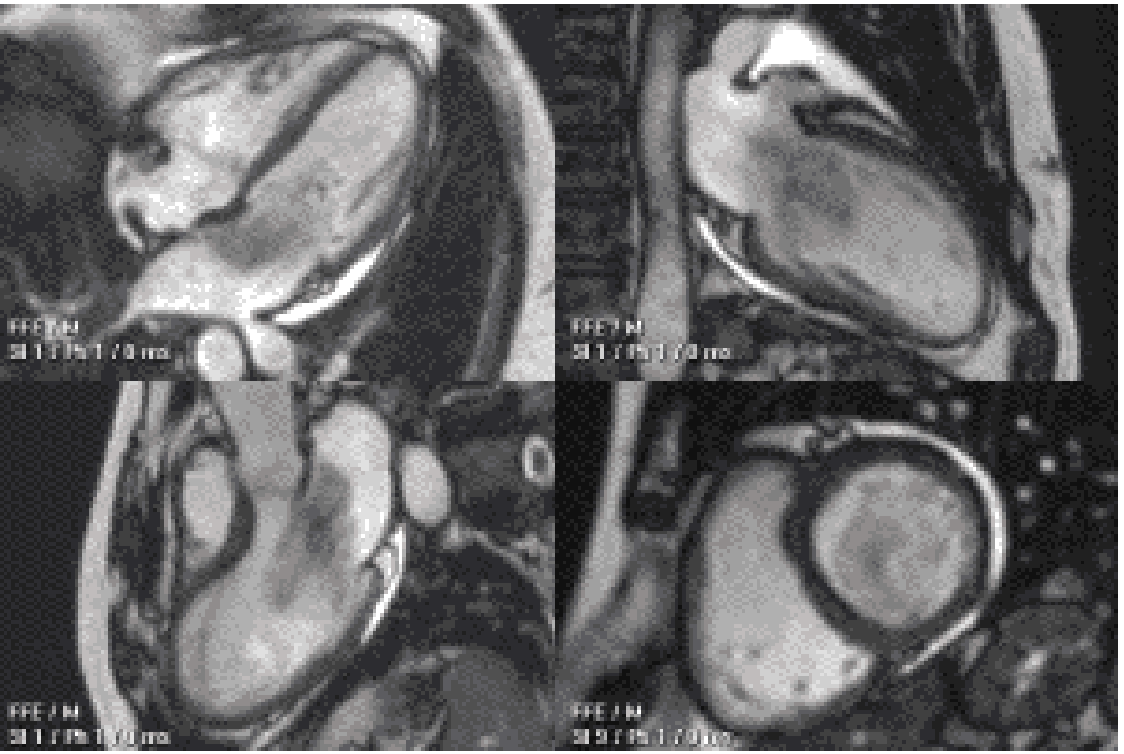


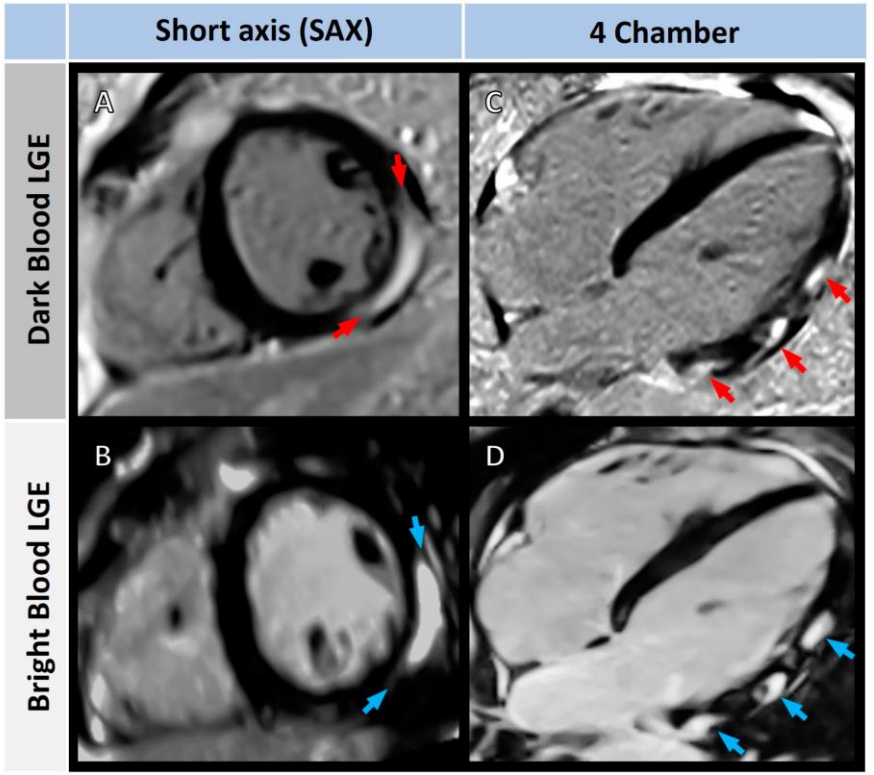


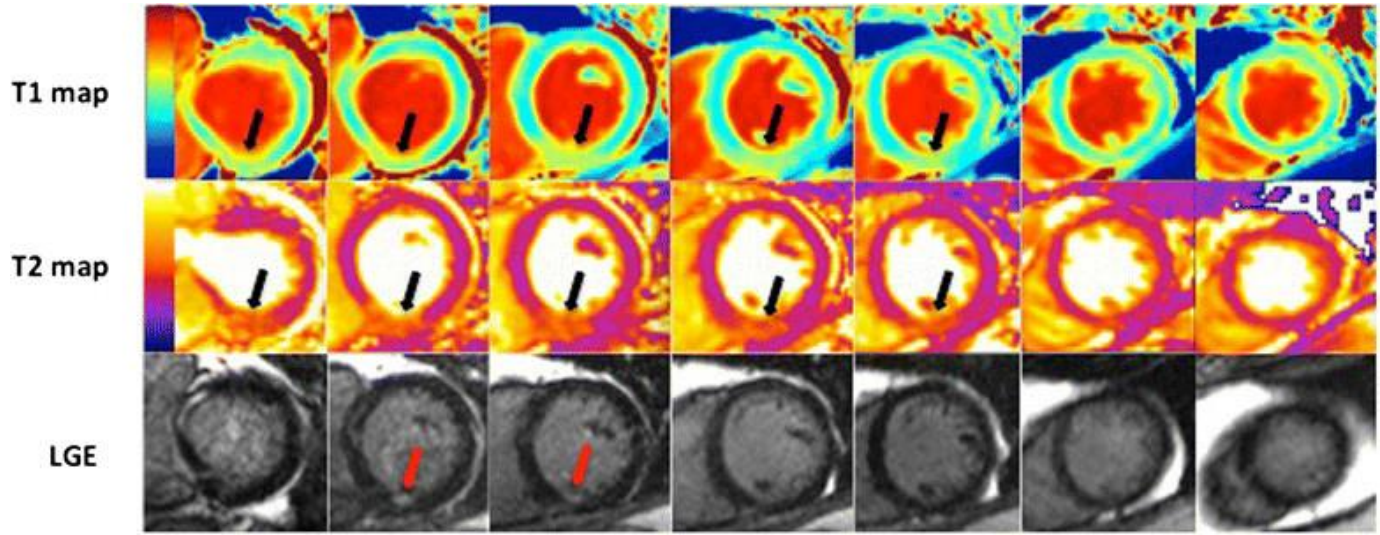


Cardiac MRI

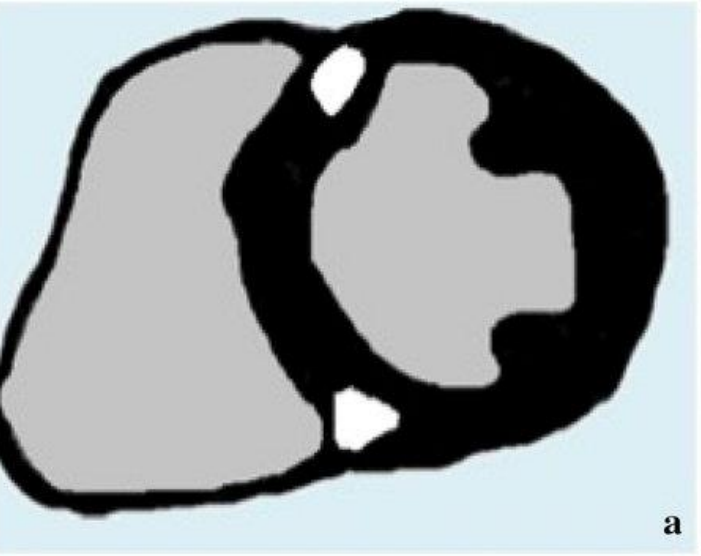
What is Cardiac MRI

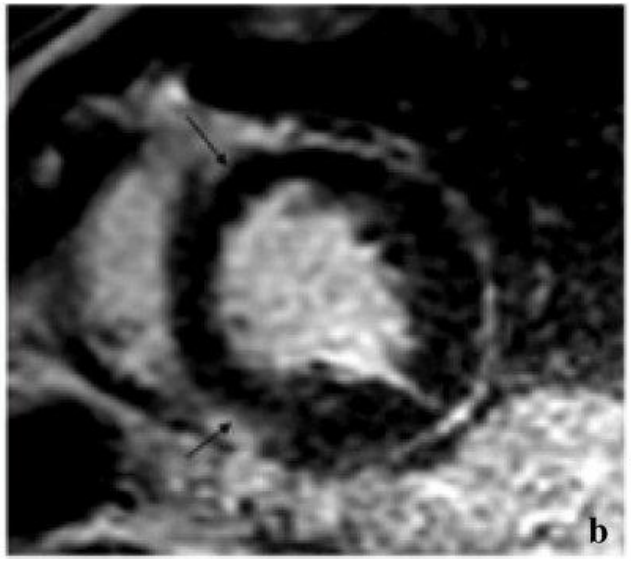


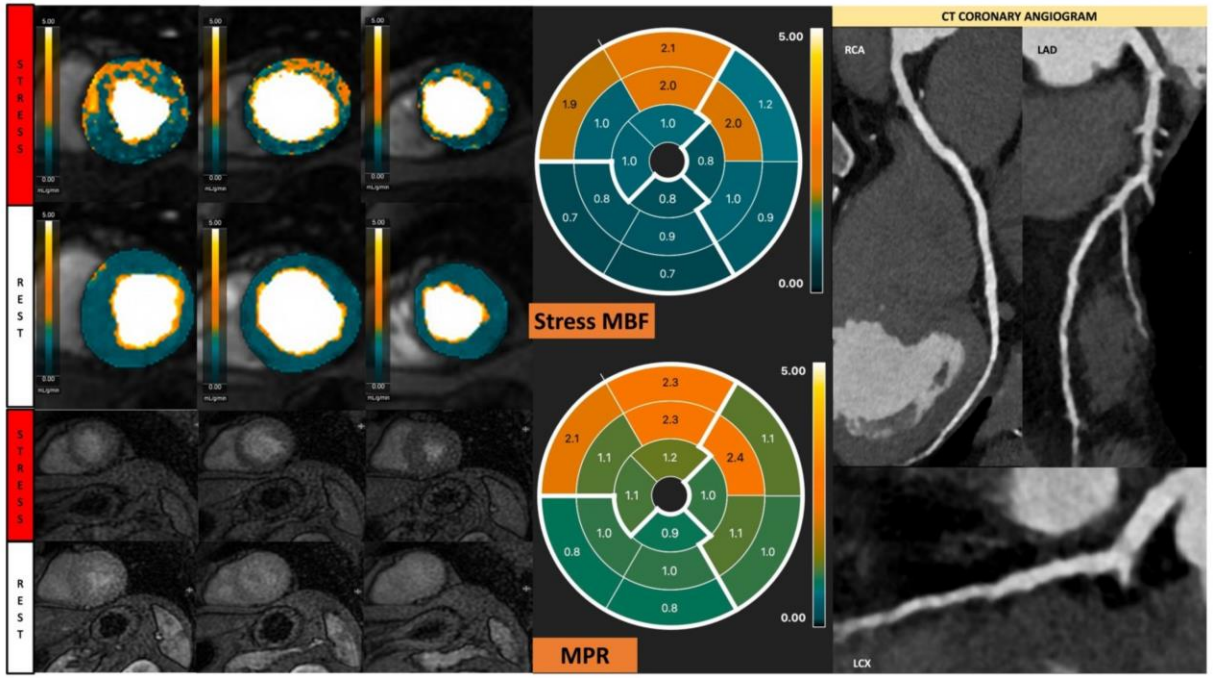


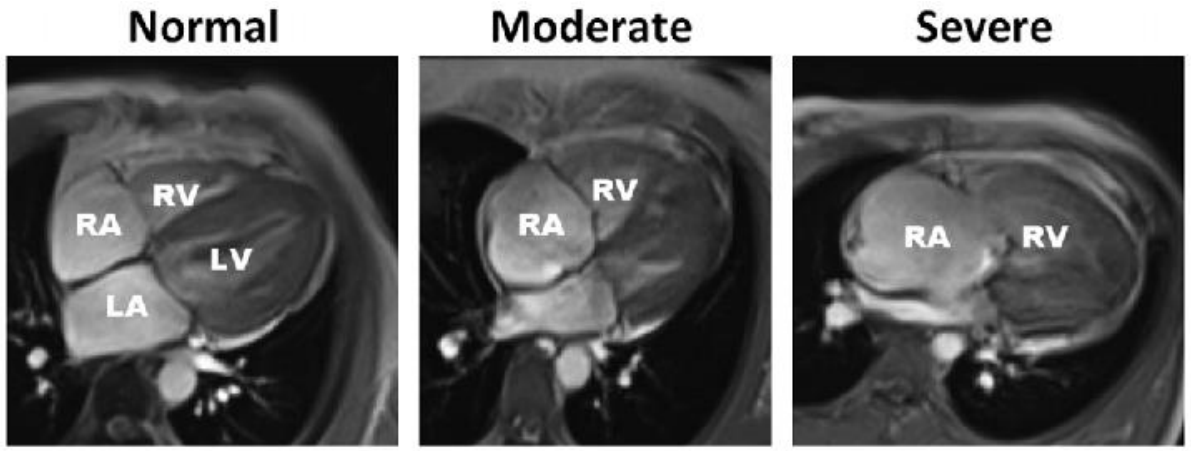


Classic OSA CMR patterns

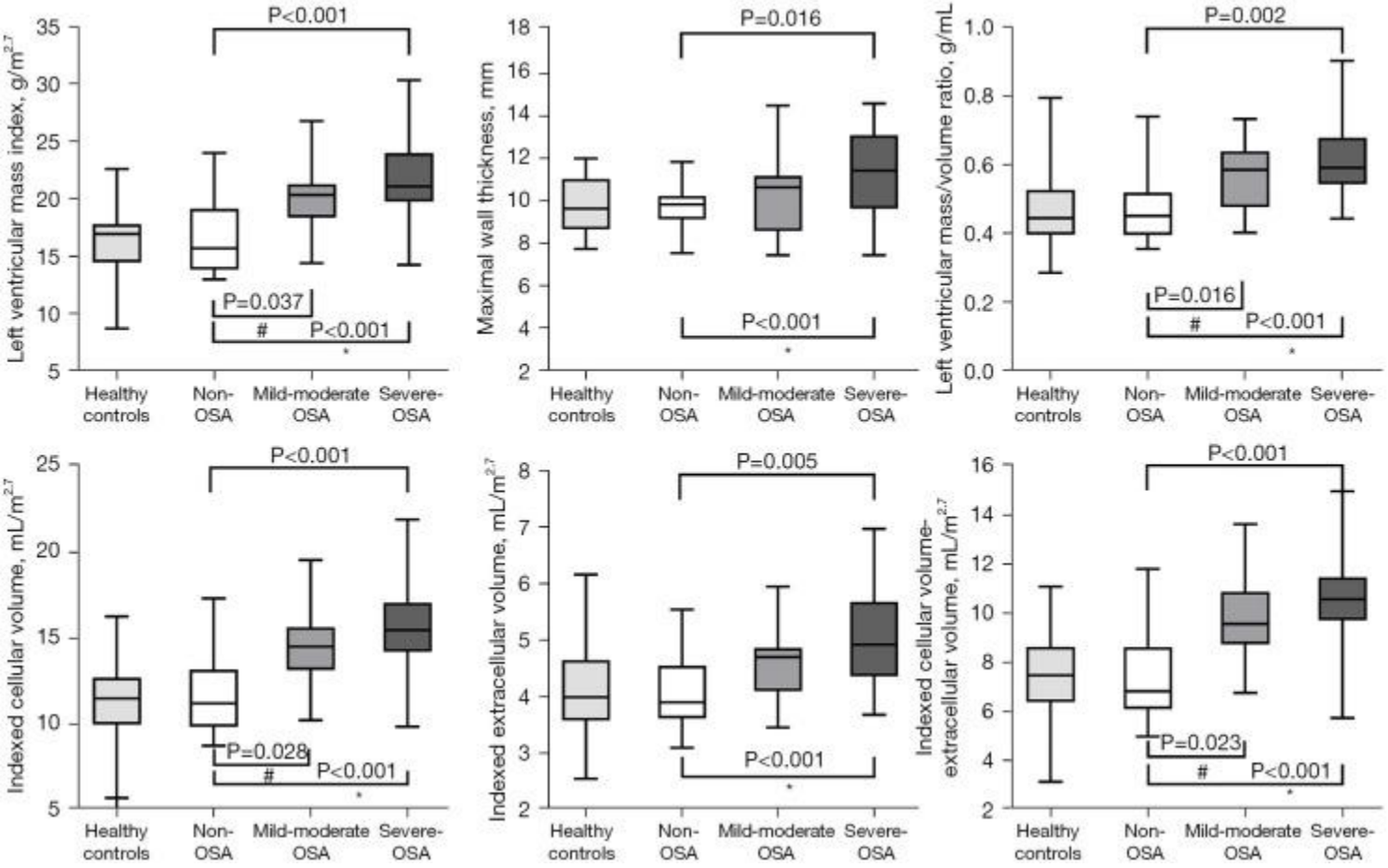


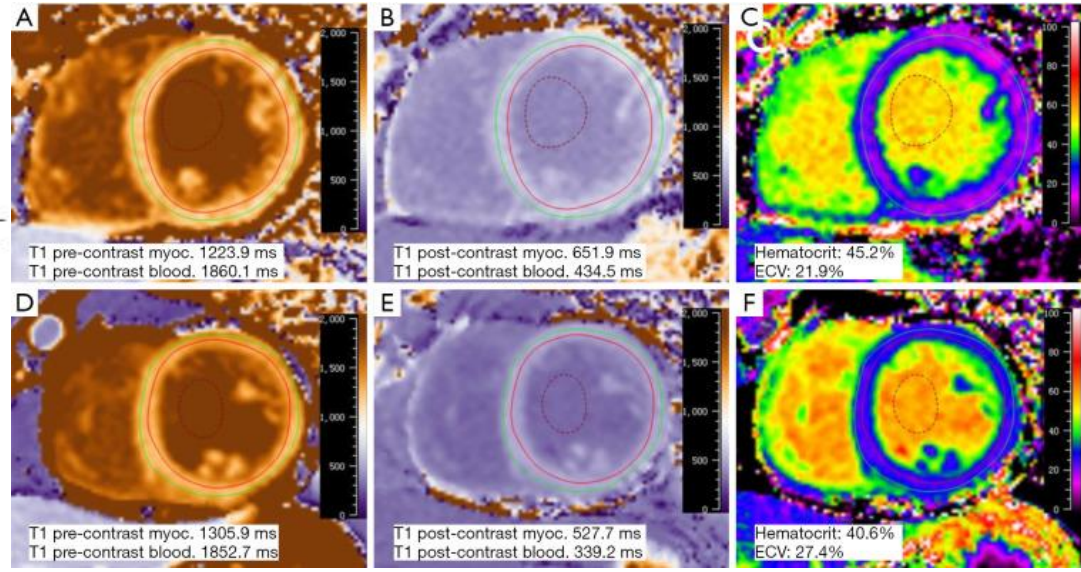




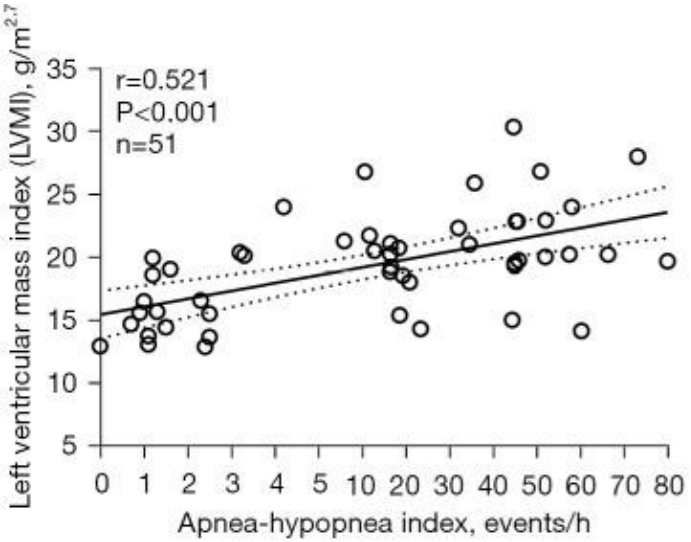


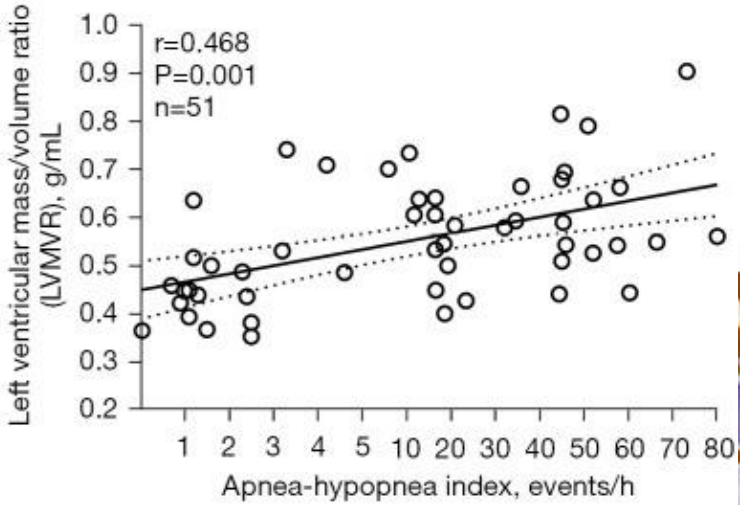
LV and RV remodeling in OSA by CMR

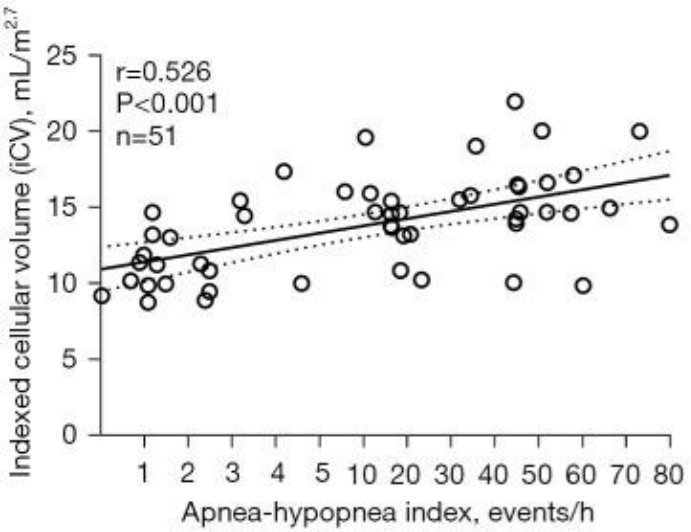


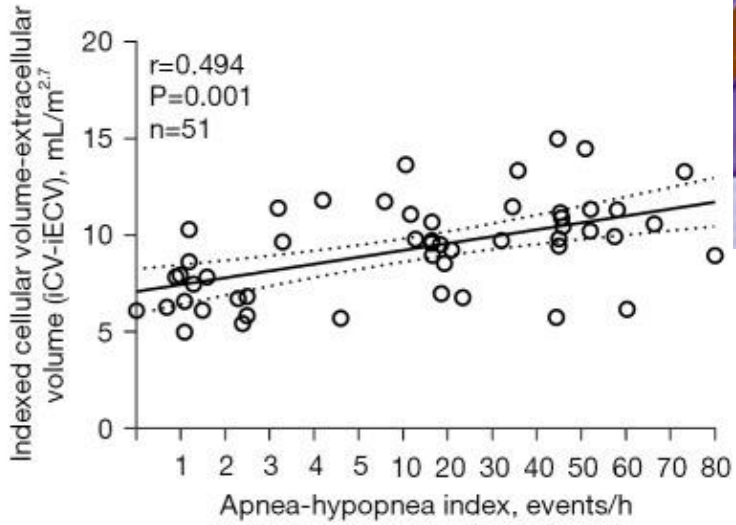


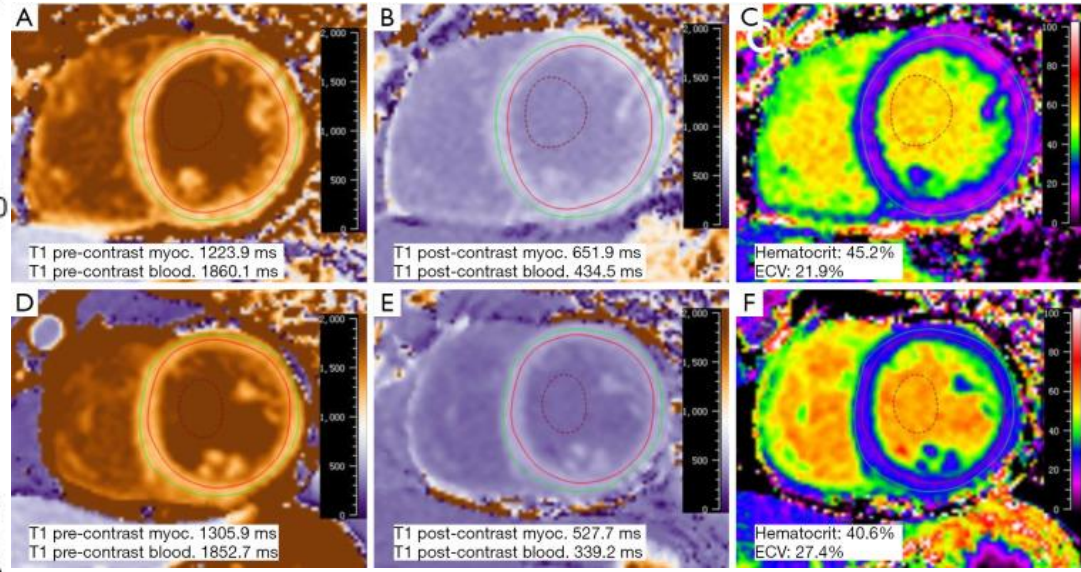
LV and RV remodeling in OSA by CMR



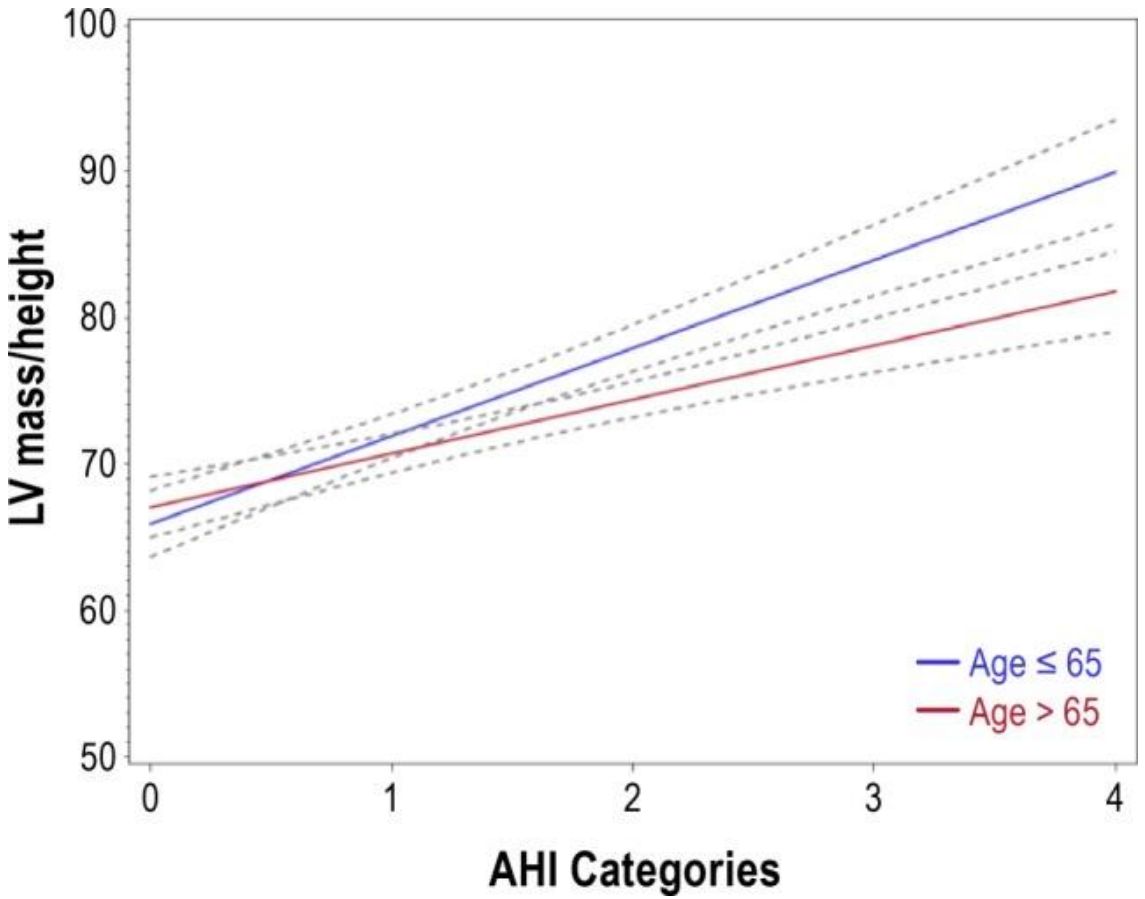


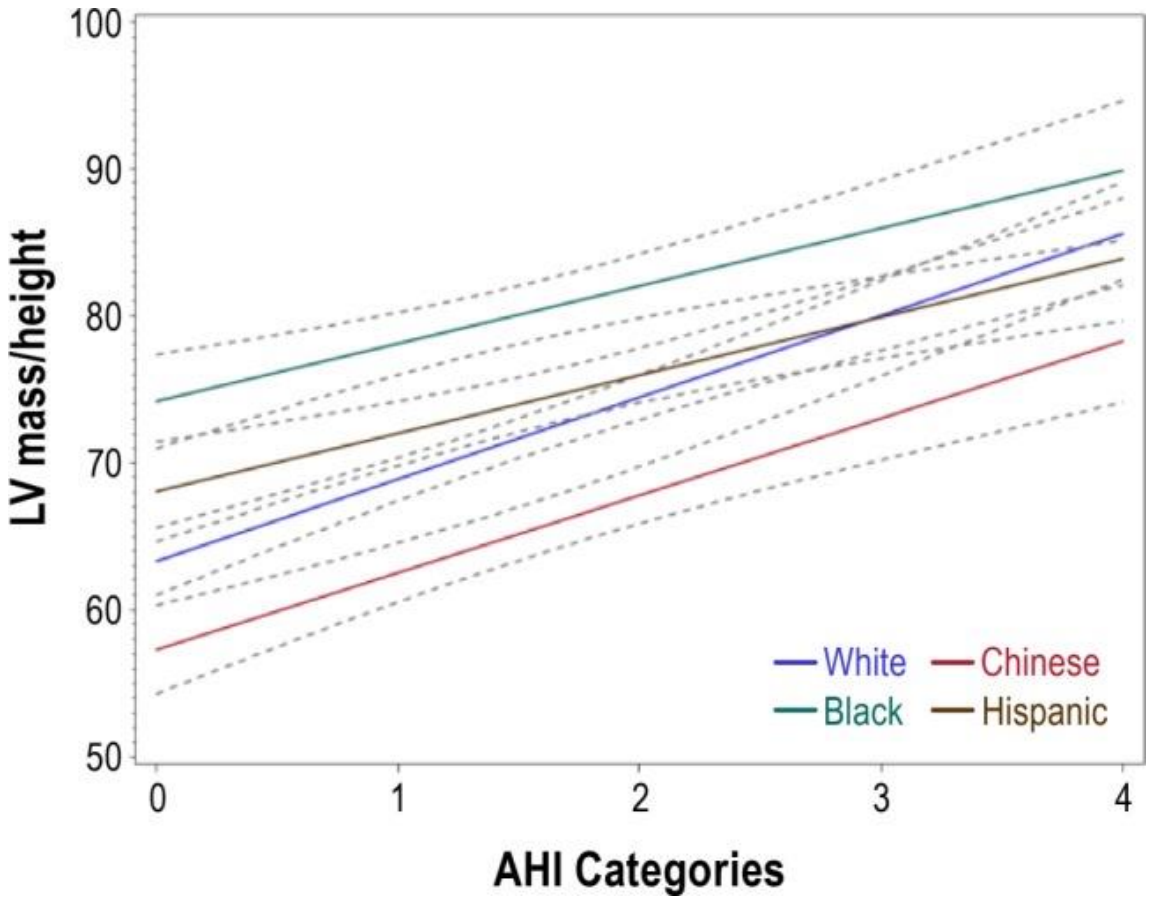




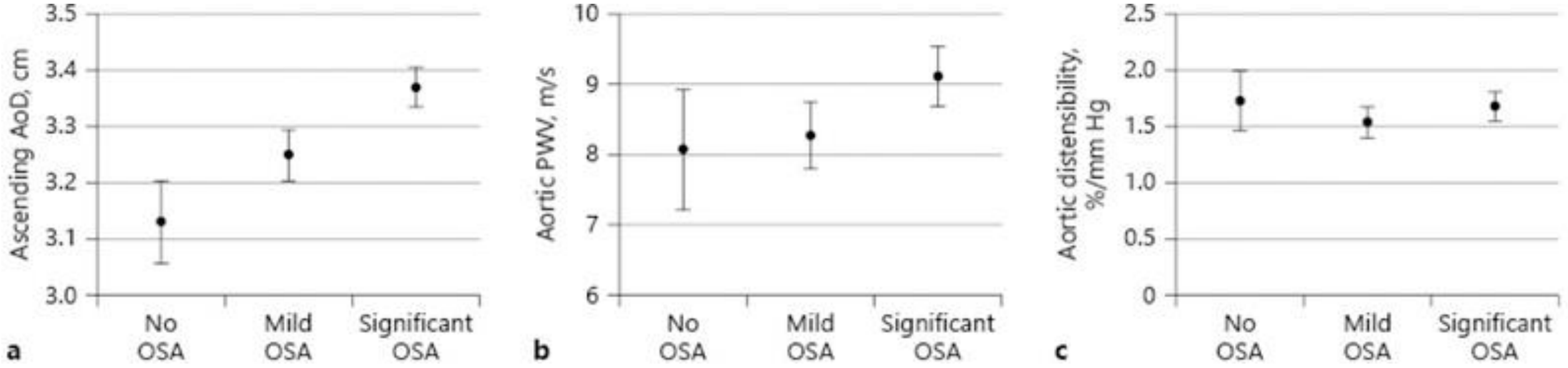


MESA: OSA and LV structure

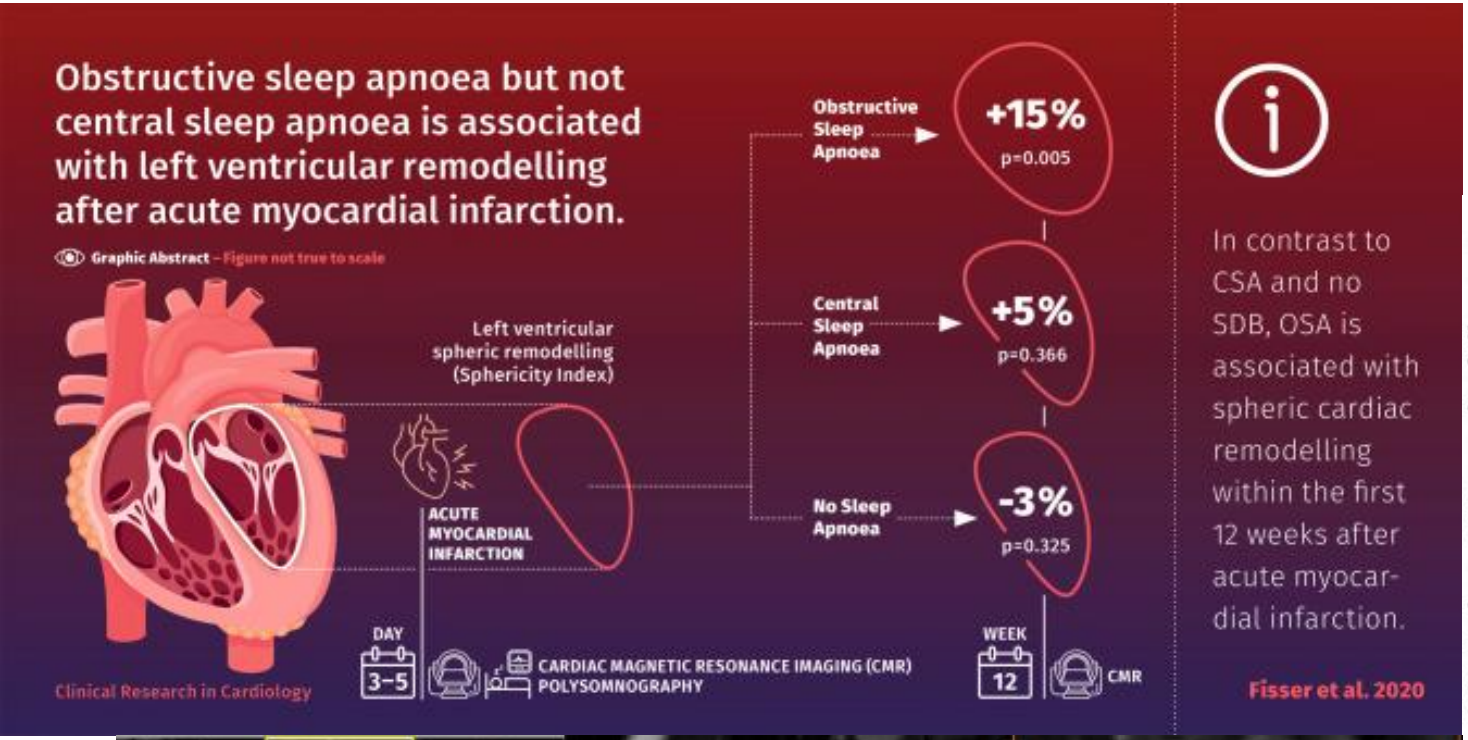


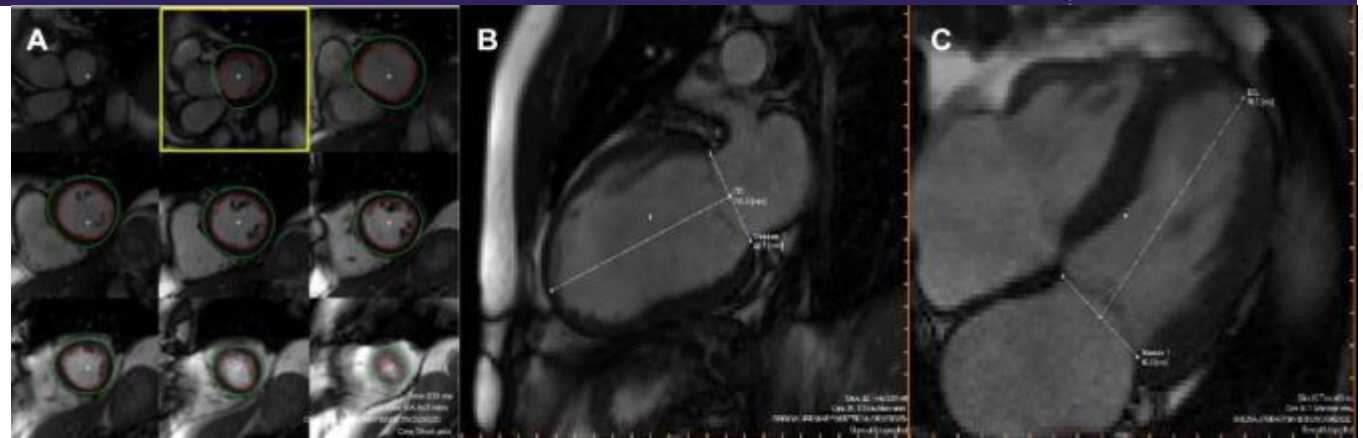


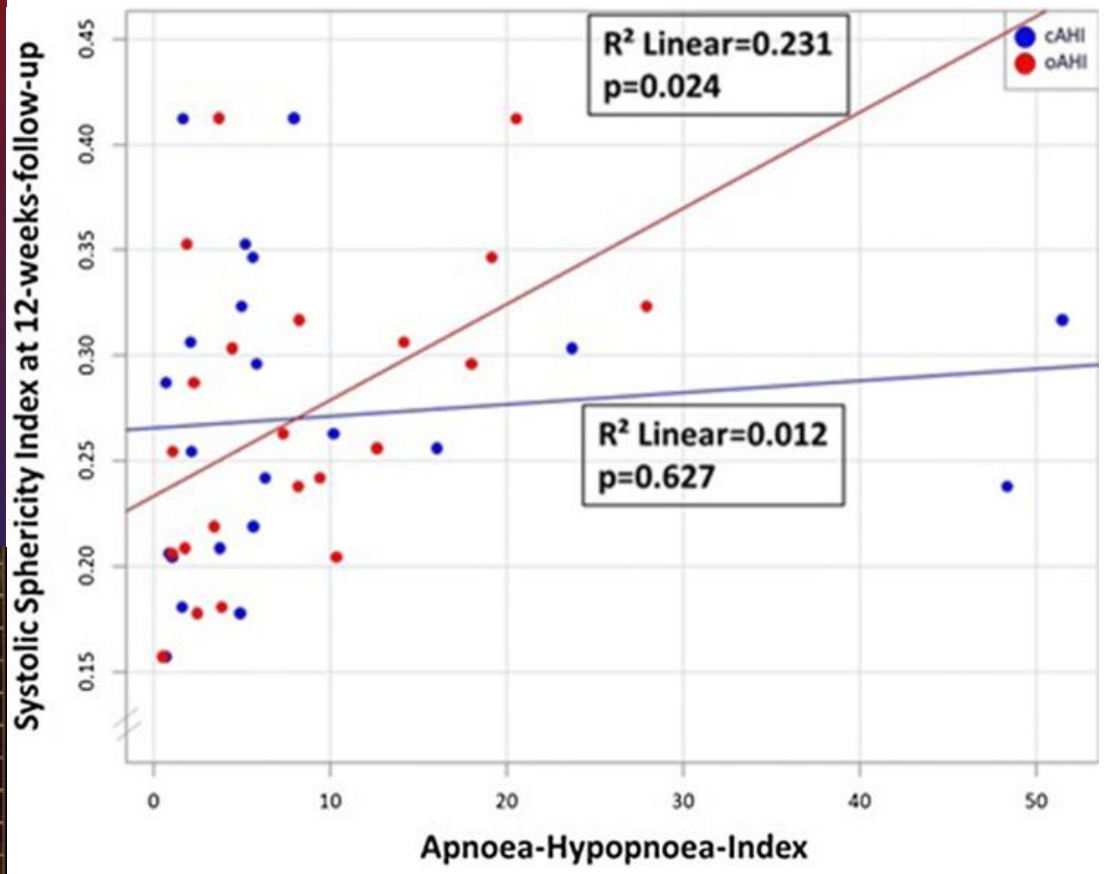
MESA: OSA and Thoracic Aorta assessment



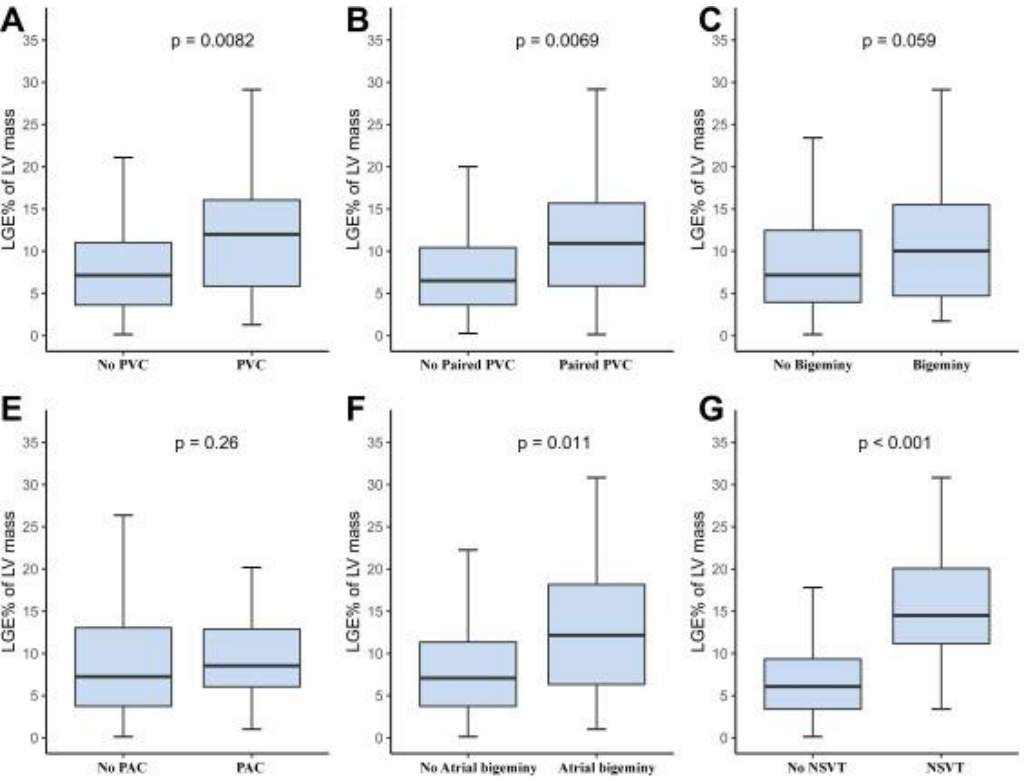
Sphericity by CMR and OSA

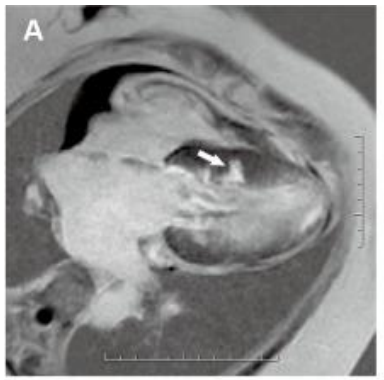


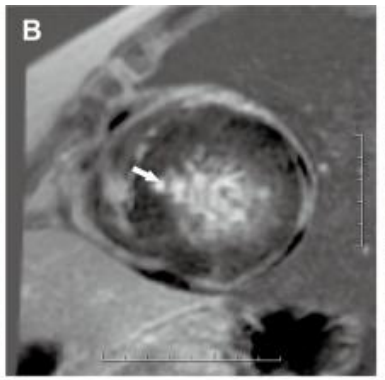


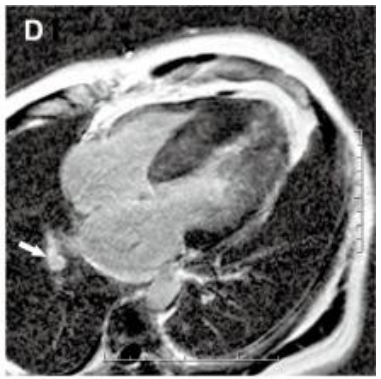


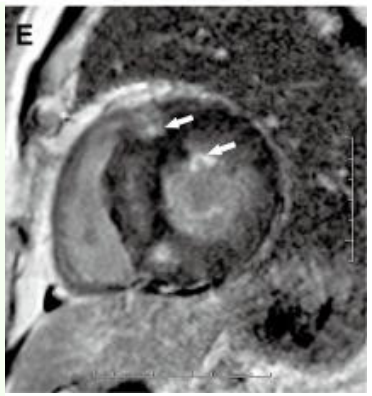
HCM, OSA and LGE mediating Arrhythmia







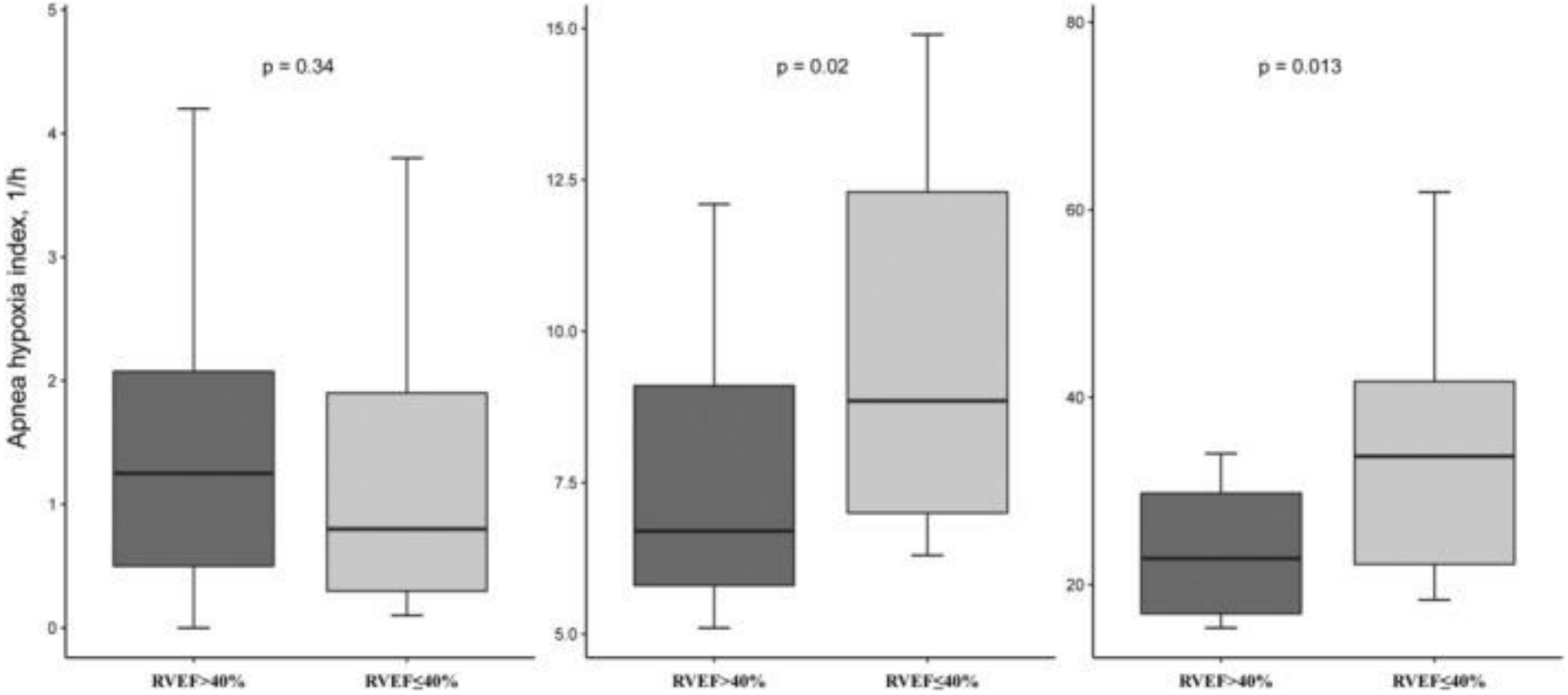




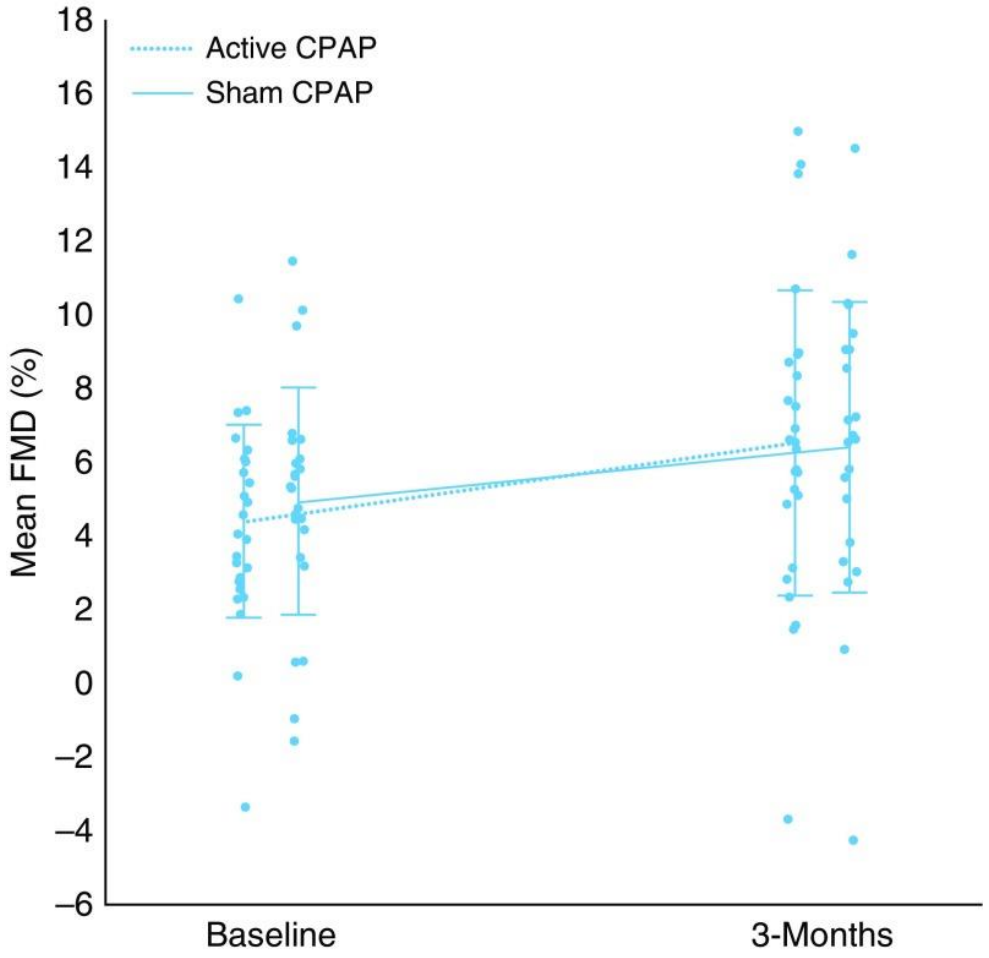
Clinical Variables Associated with LGE% from Univariate and Multivariate Analysis

Variable	Univariate		Multivariate Adjusted R^2=0.264	
	r	p	β	p
Age	−0.025	0.751		
Sex	−0.177	0.021		
History of syncope	0.159	0.040		
NYHA class	0.211	0.006	0.174	0.018
Left atrial diameter	0.142	0.066		
IVST	0.161	0.037	0.163	0.026
Apnea hypopnea index, 1/h	0.278	<0.001	0.286	<0.001
Oxygen desaturation index, 1/h	0.247	0.001		
Time ratio of SpO2 <90%, %	0.163	0.034		

OSA and RVEF among HOCM

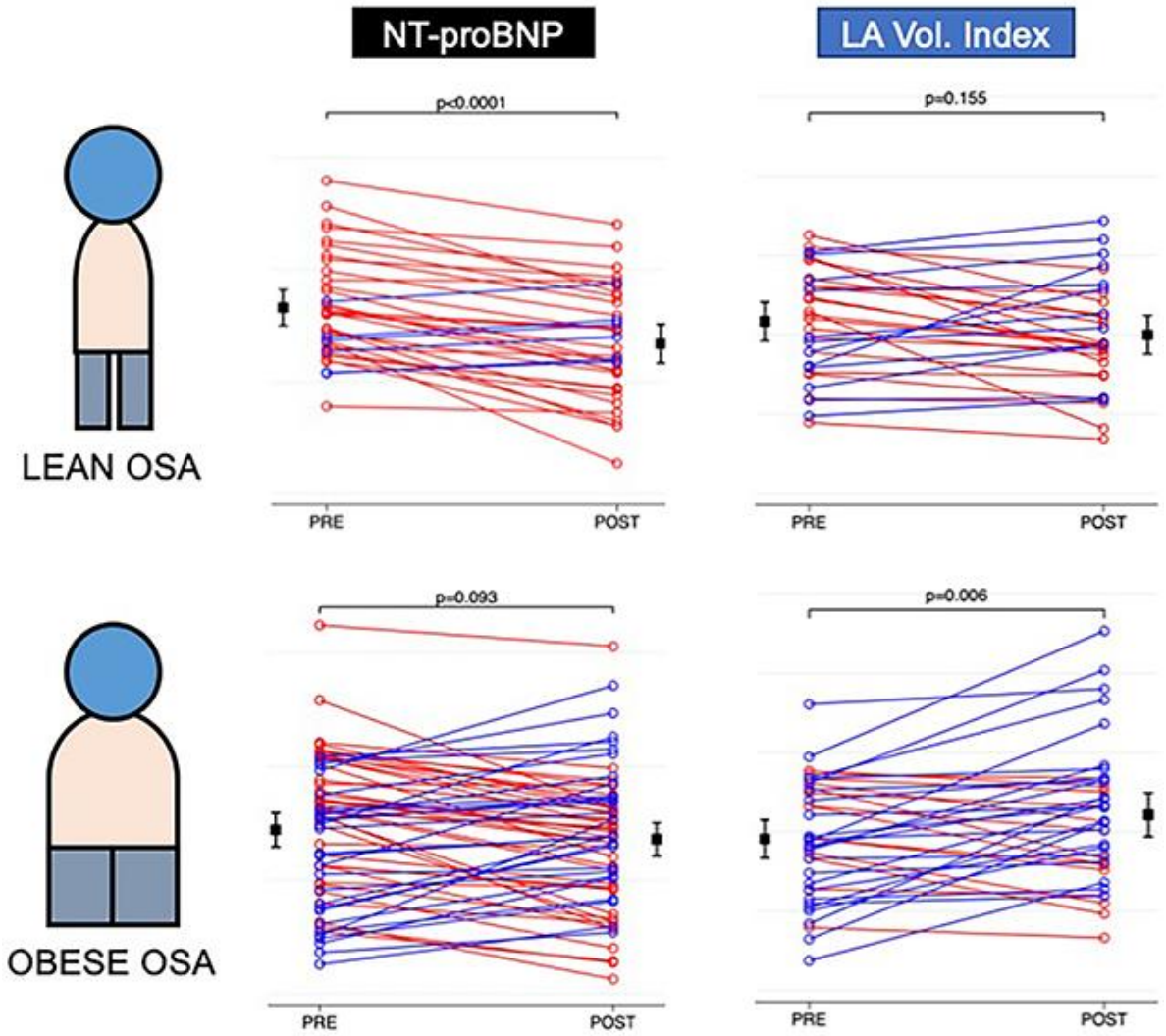


CPAP on FMD and Chamber sizes in OSA and DM2

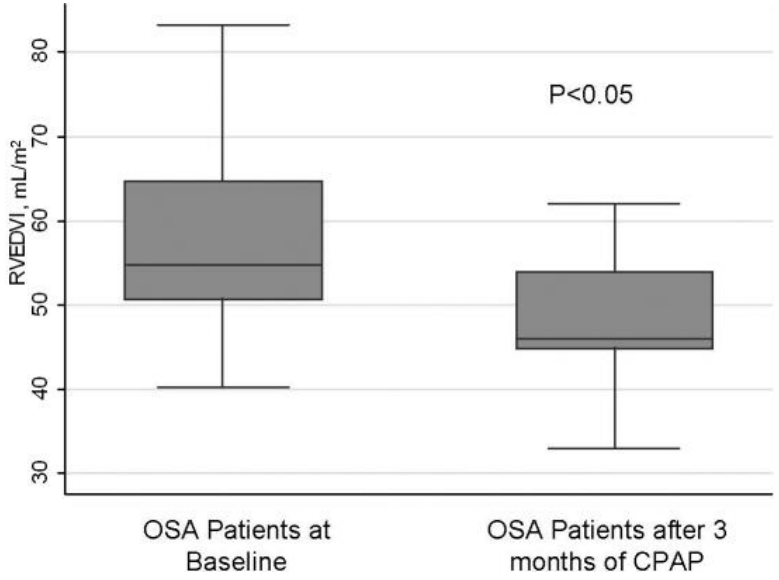


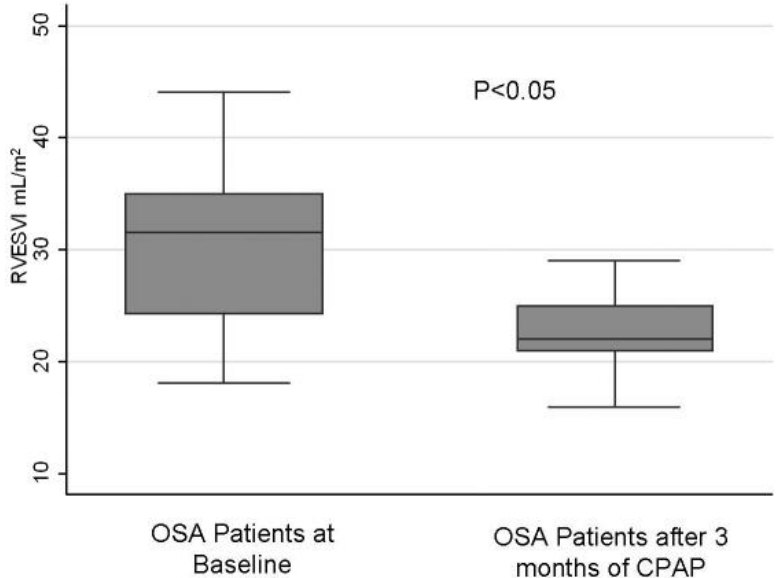
Variable	Control Subjects (*n* = 28)	T2DM Only (*n* = 27)	OSA Only (*n* = 29)	T2DM + OSA (*n* = 57)	OSA Main Effect	T2DM Main Effect	OSA × T2DM Interaction
Macrovascular reactivity							
Flow-mediated dilation, %	6.1 ± 4.0	7.3 ± 3.6	6.8 ± 4.5	4.8 ± 2.9	0.9 (−1.1 to 2.9)	1.3 (−0.7 to 3.3)	−3.1 (−5.6 to −0.6)*
NTG-induced dilation, %	16.5 ± 8.2	17.0 ± 6.9	15.3 ± 8.4	13.6 ± 7.5	−1.2 (−4.1 to 1.7)	−0.3 (−2.8 to 2.2)	
Microvascular reactivity							
ACh-induced dilation, %	29.9 ± 22.9	36.9 ± 29.9	44.7 ± 36.1	31.2 ± 26.2	4.6 (−7.0 to 16.2)	−4.0 (−14.4 to 6.4)	
SNP-induced dilation, %	28.9 ± 30.4	31.9 ± 27.0	35.7 ± 26.7	33.6 ± 29.7	9.4 (−2.0 to 20.8)	2.5 (−7.7 to 12.7)	
CMR imaging							
LV mass, g	104.6 ± 24.8	96.0 ± 19.3	98.0 ± 32.5	109.1 ± 32.8	−4.0 (−13.4 to 5.4)	1.1 (−7.3 to 9.5)	
LV end-diastolic volume, ml	158.0 ± 32.0	135.3 ± 22.9	129.5 ± 30.2	139.6 ± 33.4	−27.7 −43.2 to −12.2)†	−16.8 (−30.9 to −2.7)*	22.4 (3.2 to 41.6)*
LV end-systolic volume, ml	62.5 ± 18.7	52.4 ± 14.3	47.9 ± 16.9	51.2 ± 19.2	−6.6 −13.1 to −0.1)*	−1.9 (−7.6 to 3.8)	
LV ejection fraction, %	61.0 ± 6.5	61.6 ± 5.8	63.6 ± 6.7	64.2 ± 6.6	1.0 (−1.5 to 3.5)	0.0 (−2.4 to 2.4)	
RV mass, g	34.4 ± 7.0	29.0 ± 5.1	28.8 ± 9.3	30.1 ± 8.2	−2.6 (−5.7 to 0.5)	−1.5 (−4.2 to 1.2)	
RV end-diastolic volume, ml	174.1 ± 31.7	144.8 ± 26.5	141.4 ± 45.1	146.4 ± 32.0	−32.0 (−49.1 to −14.9)†	−21.6 (−36.5 to −6.7)†	23.2 (2.6 to 43.8)*
RV end-systolic volume, ml	79.6 ± 20.0	61.3 ± 16.2	54.5 ± 18.3	58.8 ± 21.7	−20.4 (−30.8 to −10.0)†	−13.0 (−22.0 to −4.0)†	14.4 (1.9 to 26.9)*

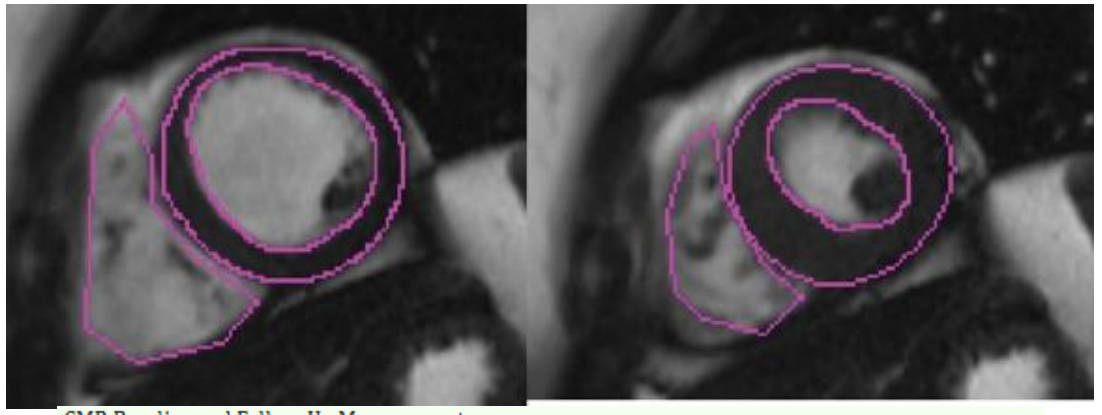
CPAP effect on BNP and LAVI by CMR



CPAP reduces RVEDV in severe OSA by CMR







CMR Baseline and Follow-Up Measurements

	Baseline	After 3 Months of CPAP
LVEDVI, mL/m^2	54.2 ± 19.5	50.4 ± 8.1
LVESVI, mL/m^2	22.4 ± 9.0	20.5 ± 3.5
">LV free wall, mm	8.0 ± 1.1	8.0 ± 1.1
LVEF, %	58.5 ± 8.2	58.9 ± 5.4
RVEDVI, mL/m^2	57.6 ± 11.4	47.8 ± 14.4[1]
RVESVI, mL/m^{2*}	30.0 ± 7.7	22.2 ± 5.5[a]
RVEF, %	47.5 ± 12.1	52.5 ± 8.6
RV free wall, mm	4.4 ± 1.3	4.2 ± 1.2
IVS, mm	7.9 ± 2.2	8.0 ± 2.4
MPRI	0.94 ± 0.14	0.83 ± 0.63

Short CPAP improves Remodeling

CPET data.

	Before Treatment (n = 17)	3 Months after CPAP Treatment (n = 17)	p-Value *
Maximum load (%)	79.86 (15.8)	85.5 (16.35)	0.093
Maximum load (W)	170.36 (39.25)	174.29 (45.18)	0.662
VO_2 (max. load) (L/min)	2.14 (0.41)	2.23 (0.44)	0.706
VO_2 (max. load) (%)	84.36 (18.08)	87.21 (17.48)	0.706
VO_2 (max. load) (mL/kg/min)	17.52 (3.79)	18.6 (3.40)	0.255
VO2/kg (max. load) (%)	83.77 (18.68)	88.92 (18.36)	0.195
VCO_2 (max. load) (L)	2.61 (0.61)	2.67 (0.67)	0.889
VCO_2 (max. load) (%)	93 (20.21)	93.86 (18.63)	1
HR rest (beats/min)	86.08 (10.10)	88.83 (12.47)	0.906
HR max (beats/min)	135.5 (20.66)	132.38 (18.13)	0.432
HR max (%)	90.25 (12.74)	88 (12.14)	0.283
O_2 pulse (max. load) (mL/beat)	16.69 (3.11)	16.46 (3.66)	0.925
O_2 pulse (max. load) (%)	72.64 (16.57)	72.14 (16.53)	0.826
VE_{AT} (L/min)	44.51 (10.59)	38.60 (7.52)	0.03 *
VE_{AT} (%) (reference value)	43.21 (21.19)	37.86 (17.77)	0.028 *
VE max load	74.53 (20.04)	69.81 (17.77)	0.51
VE max load (%)	68.84 (22.18)	66.36 (24.24)	0.615
VE/VCO_2 slope (peak value)	23.47 (2.73)	20.63 (3.52)	0.042 *
RER max. load	1.19 (0.14)	1.21 (0.09)	0.109

Changes in cardiac MRI parameters in OSA group patients after treatment with CPAP.

	Before Treatment (n = 17)	3 Months after CPAP Treatment (n = 17)	p-Value *
	Mean (Standard Deviation)		
LA and LV parameters			
LV EDV (mL)	182.68 (43.81)	197.93 (30.28)	0.102
LV EDVI (mL/m^2)	75.92 (17.51)	81.79 (10.67)	0.136
LV ESV (mL)	70.03 (21.58)	77.66 (16.17)	0.163
LV ESVI (mL/m^2)	29.12 (8.86)	32.26 (7.04)	0.193
LV SV (mL)	114.43 (26.31)	122.47 (20.14)	0.210
LV SVI (mL/m^2)	47.49 (10.14)	50.63 (7.26)	0.246
LV EF (%)	61.78 (4.89)	62.08 (5.94)	0.906
LV mass	125.65 (30.39)	120.39 (26.13)	0.266
LA (cm^2)	31.61 (9.16)	25.65 (5.13)	0.724
LV GLS (%)	−23.76 (4.10)	−24.29 (3.91)	0.981
LV GCS (%)	−35.42 (4.79)	−34.88 (5.51)	0.850
LA GLS (%)	20.45 (7.25)	26.05 (14.00)	0.043 *

Cardiac MRI parameters of control and OSA group patients.

	OSA (n = 32)	Control (n = 17)	p-Value *
	Mean (Standard Deviation)		
LV parameters			
LV EDVI (mL/m^2)	79.44 (21.02)	71.58 (15.59)	0.438
LV ESVI (mL/m^2)	30.89 (11.69)	27.47 (10.94)	0.438
LV EF (%)	61.62 (5.28)	62.71 (9.45)	0.506
LV GLS (%)	−24.86 (4.85)	−23.64 (4.89)	0.532
LV GCS (%)	−35.14 (4.67)	−39.11 (7.01)	0.063

Changes in cardiac MRI parameters in OSA group patients after treatment with CPAP.

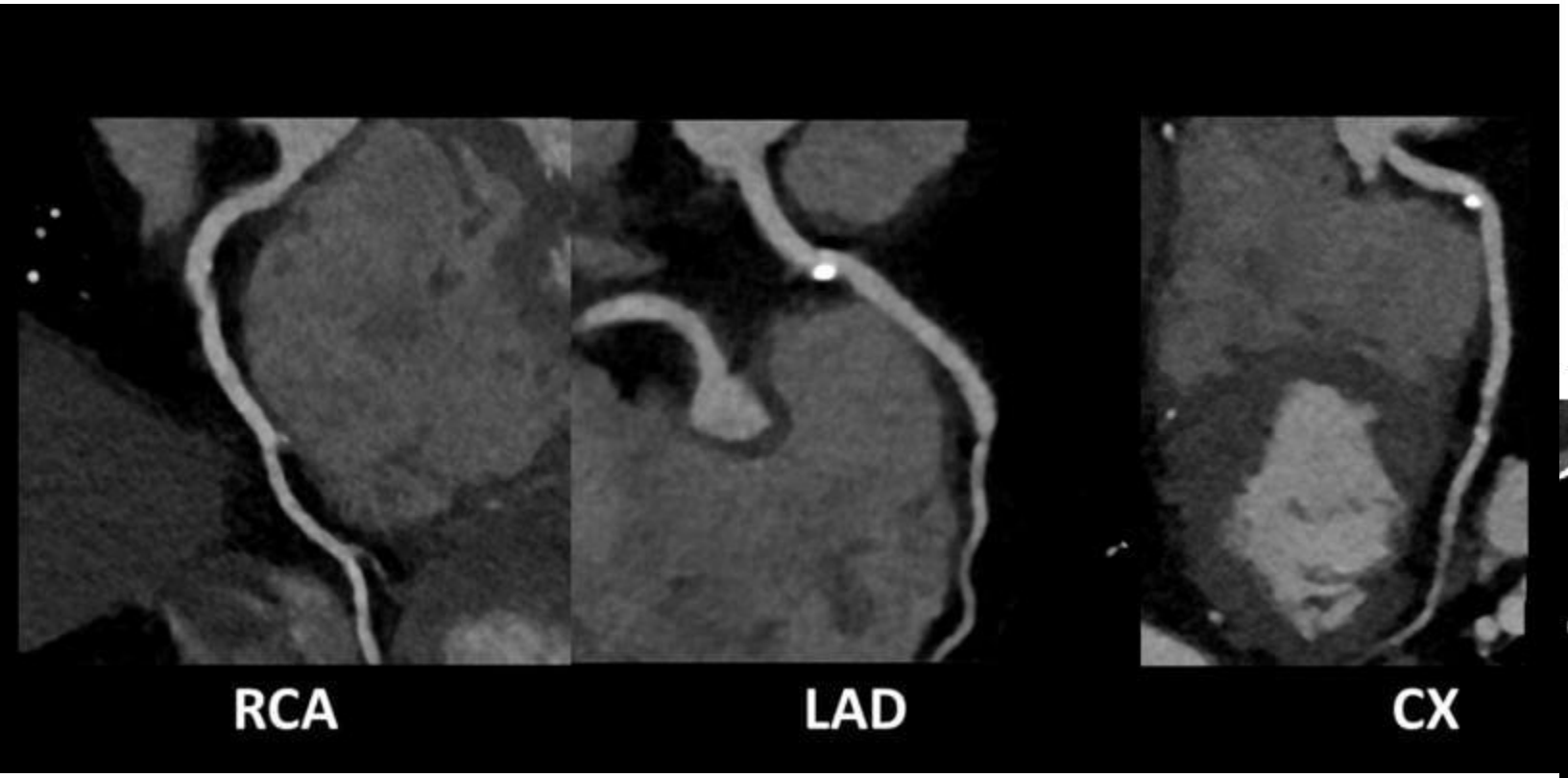
	Before Treatment (n = 17)	3 Months after CPAP Treatment (n = 17)	p-Value *
	Mean (Standard Deviation)		
RA and RV parameters			
RV EDV (mL)	164.82 (32.57)	180.16 (39.09)	0.042 *
RV EDVI (mL/m^2)	68.82 (12.74)	74.81 (14.74)	0.067
RV ESV (mL)	78.33 (23.64)	77.45 (21.77)	0.846
RV ESVI (mL/m^2)	32.53 (9.27)	32.94 (8.21	0.791
RV EF (%)	53.35 (9.36)	57.09 (7.51)	0.151
RA (cm^2)	21.94 (2.68)	23.48 (4.19)	0.129
RV GLS (%)	−24.21 (7.37)	−27.31 (5.19)	0.480
RA GLS (%)	21.04 (7.14)	26.18 (7.17)	0.043 *

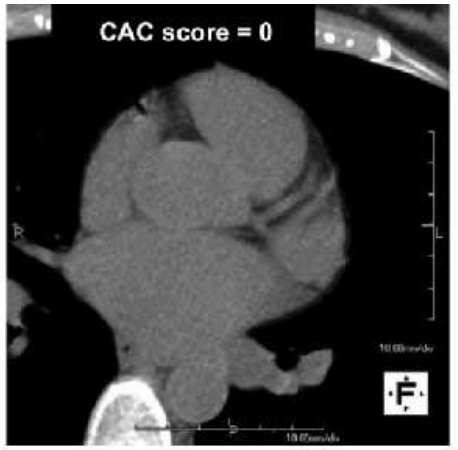
Cardiac MRI parameters of control and OSA group patients.

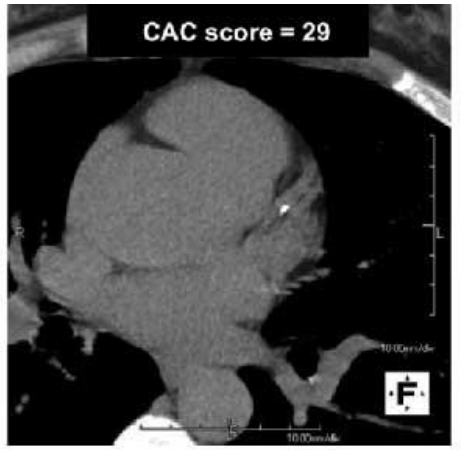
	OSA (n = 32)	Control (n = 17)	p-Value *
	Mean (Standard Deviation)		
RV parameters			
RV GLS (%)	−25.72 (10.93)	−21.54 (5.87)	0.096
RV EDVI (mL/m^2)	70.46 (14.77)	58.65 (14.31)	0.042 *
RV ESVI	33.56 (11.67)	22.88 (9.19)	0.069
RV EF (%)	53.69 (8.91)	61.35 (9.08)	0.016 *

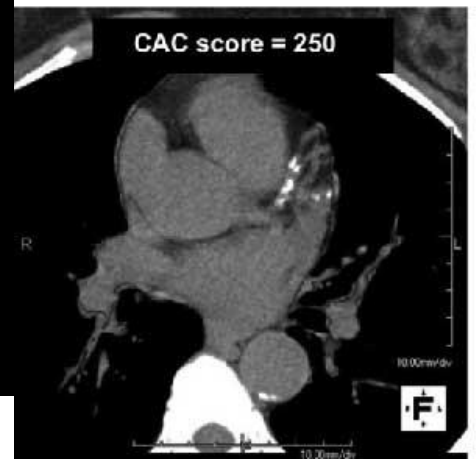
Cardiac CTA

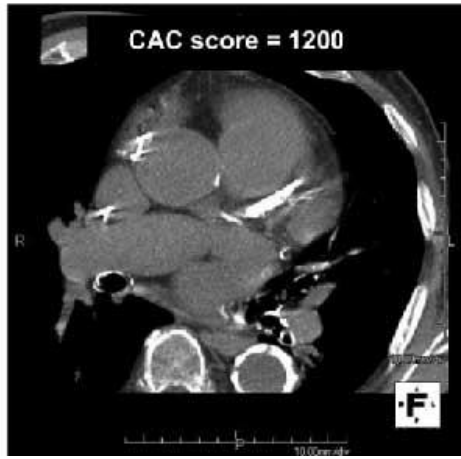
What is Cardiac CT? Is there an OSA phenotype?



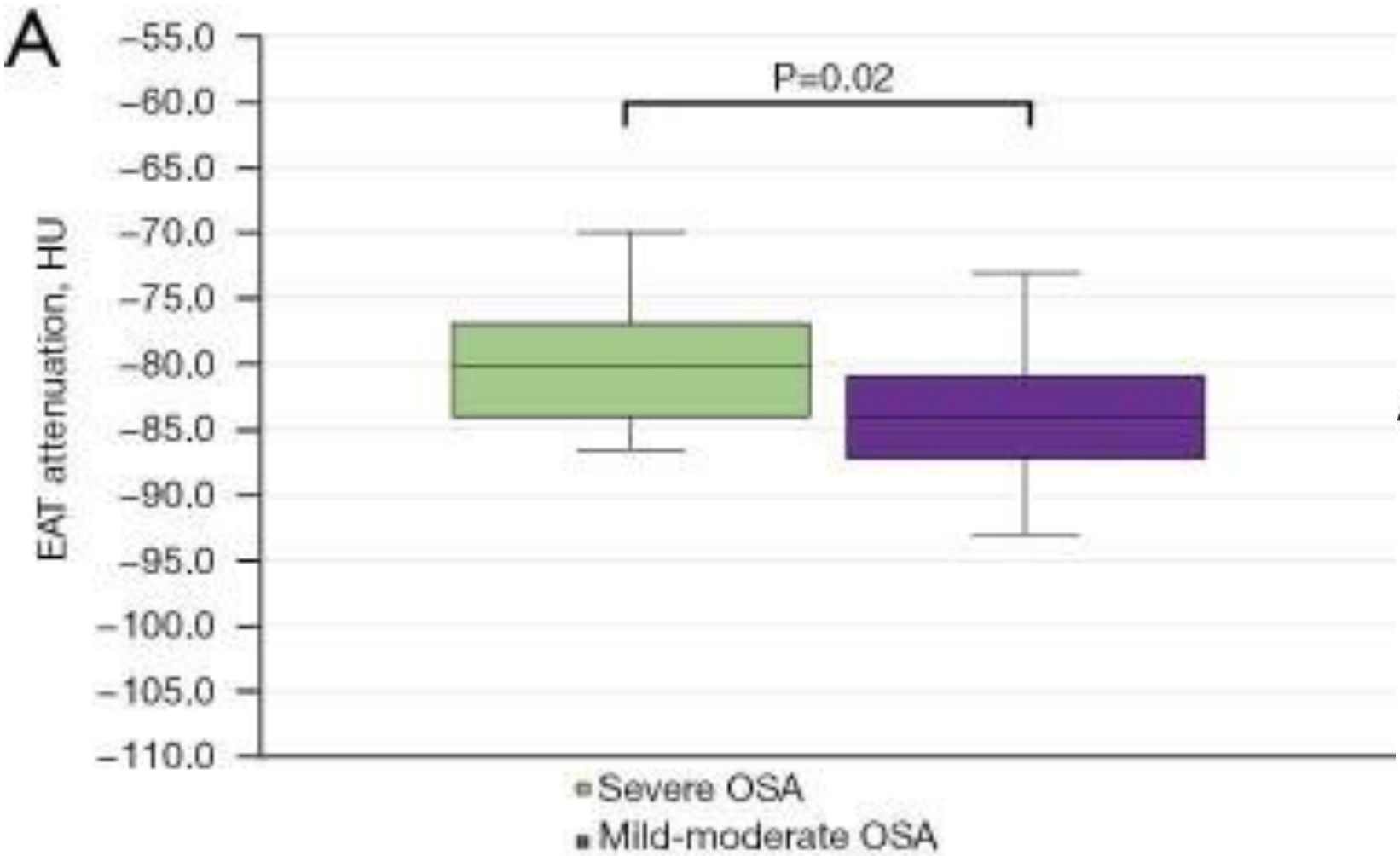


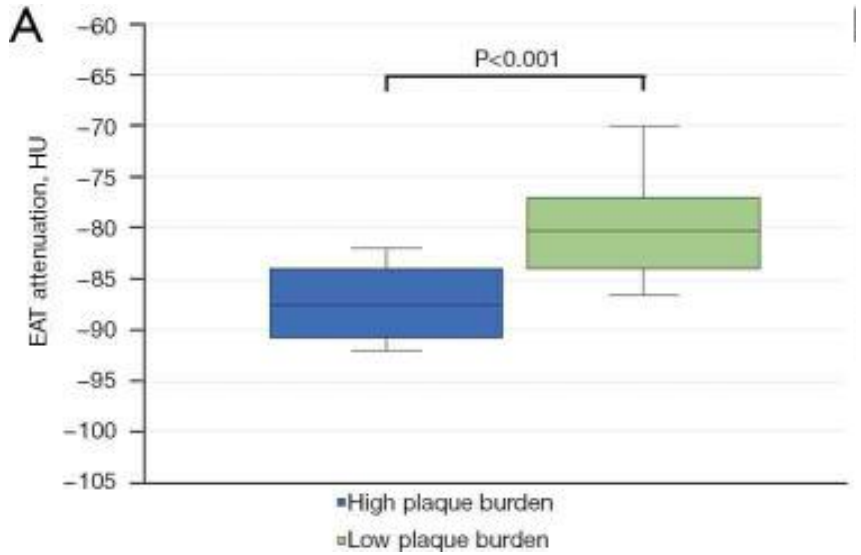


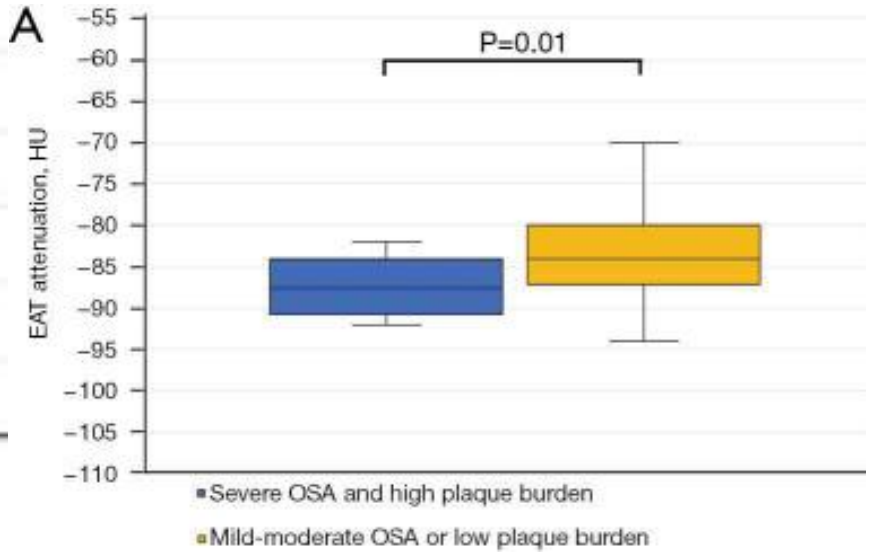




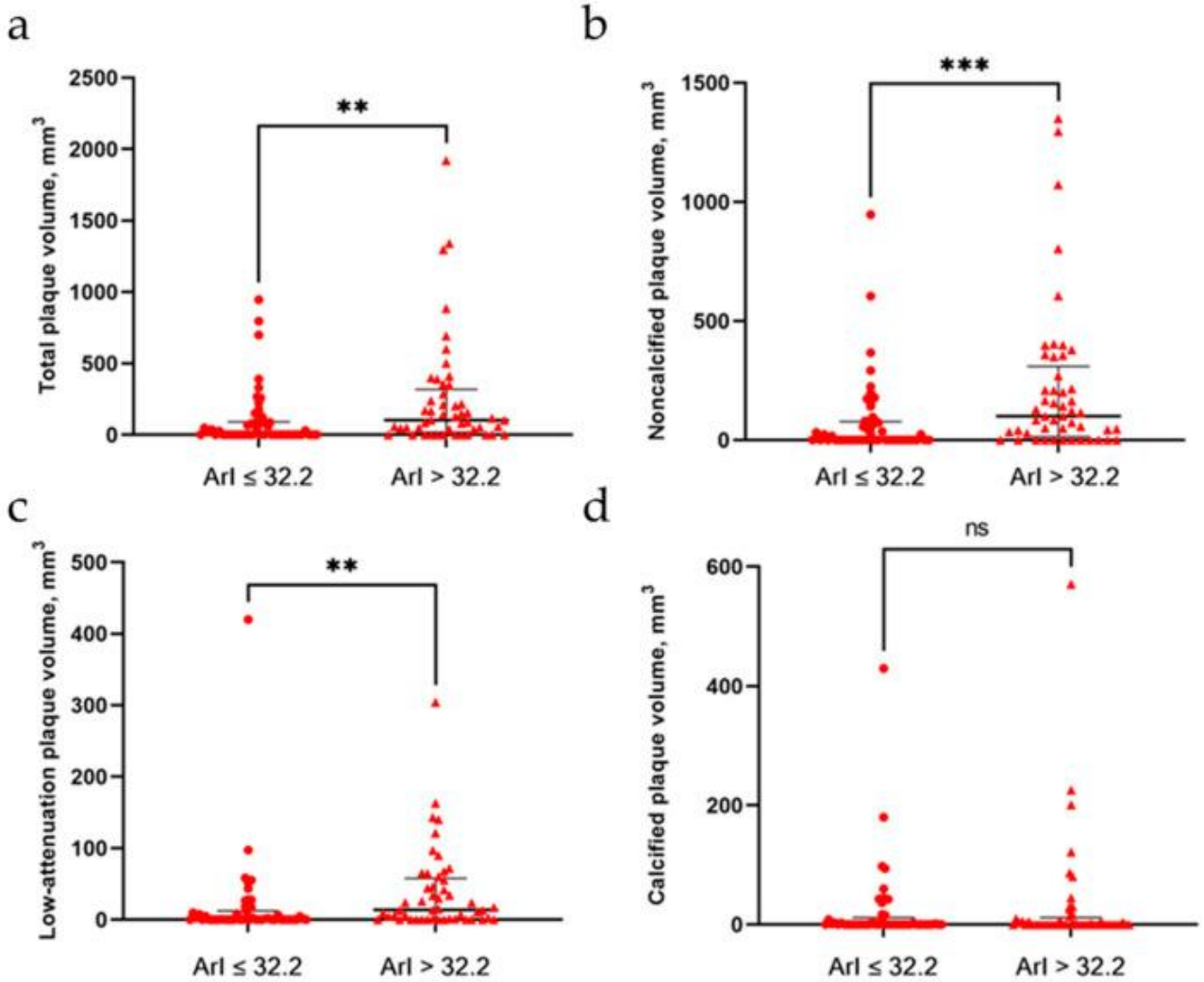
CCTA evaluation of EAT inflammation in OSA



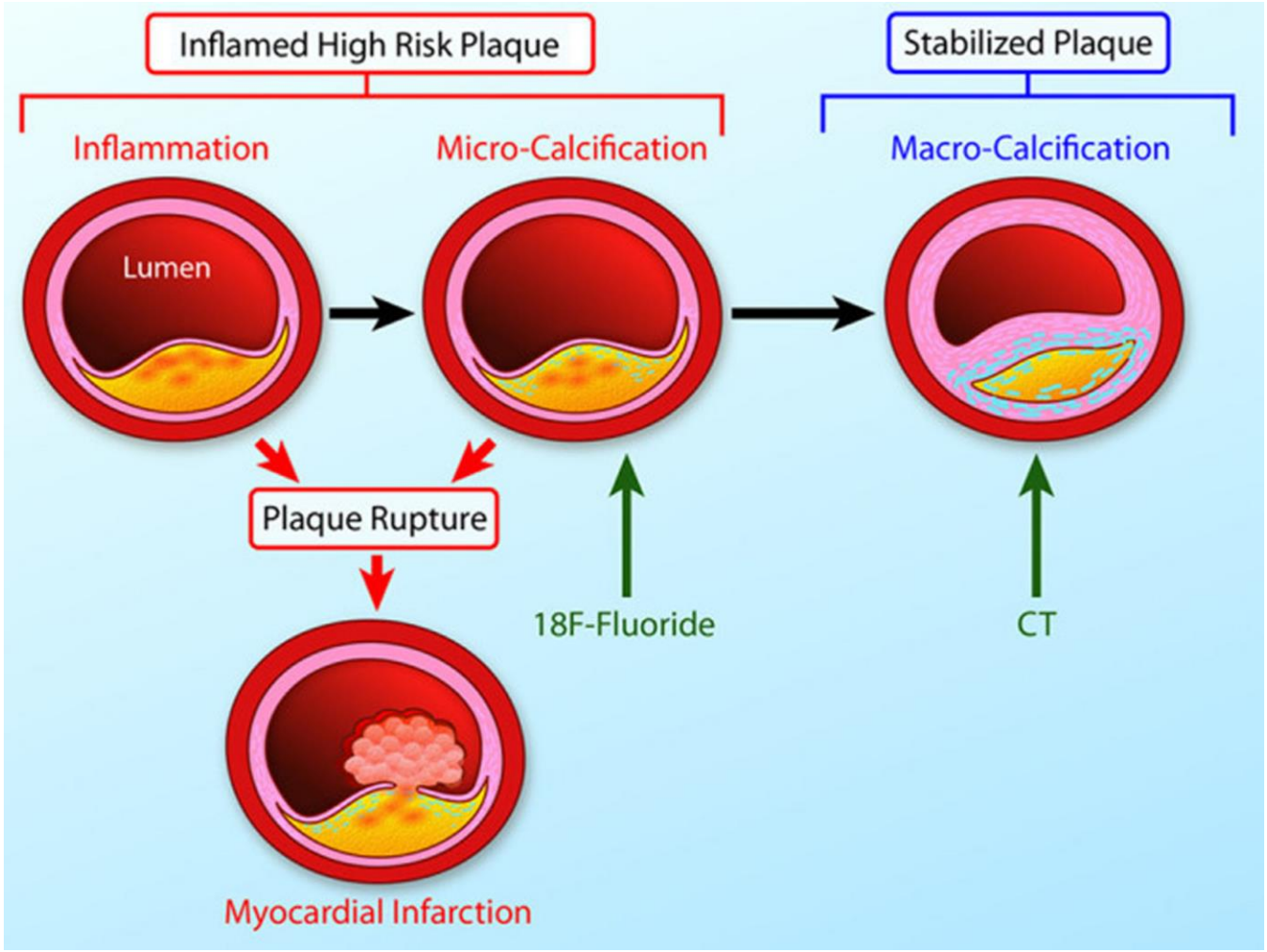


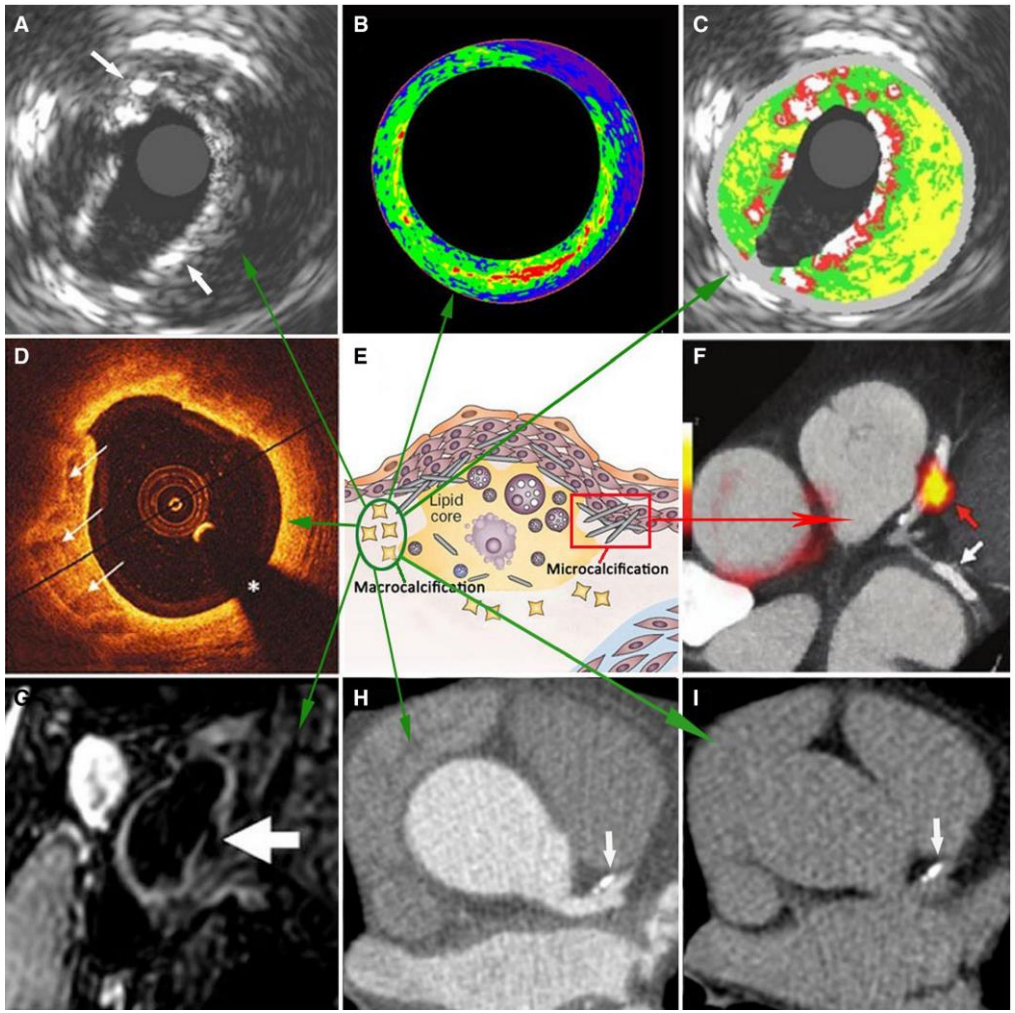


Arousals among OSA and CAD by CCTA



Mechanism of CAD – Calcified vs. Noncalcified





OSA and Calcium scoring

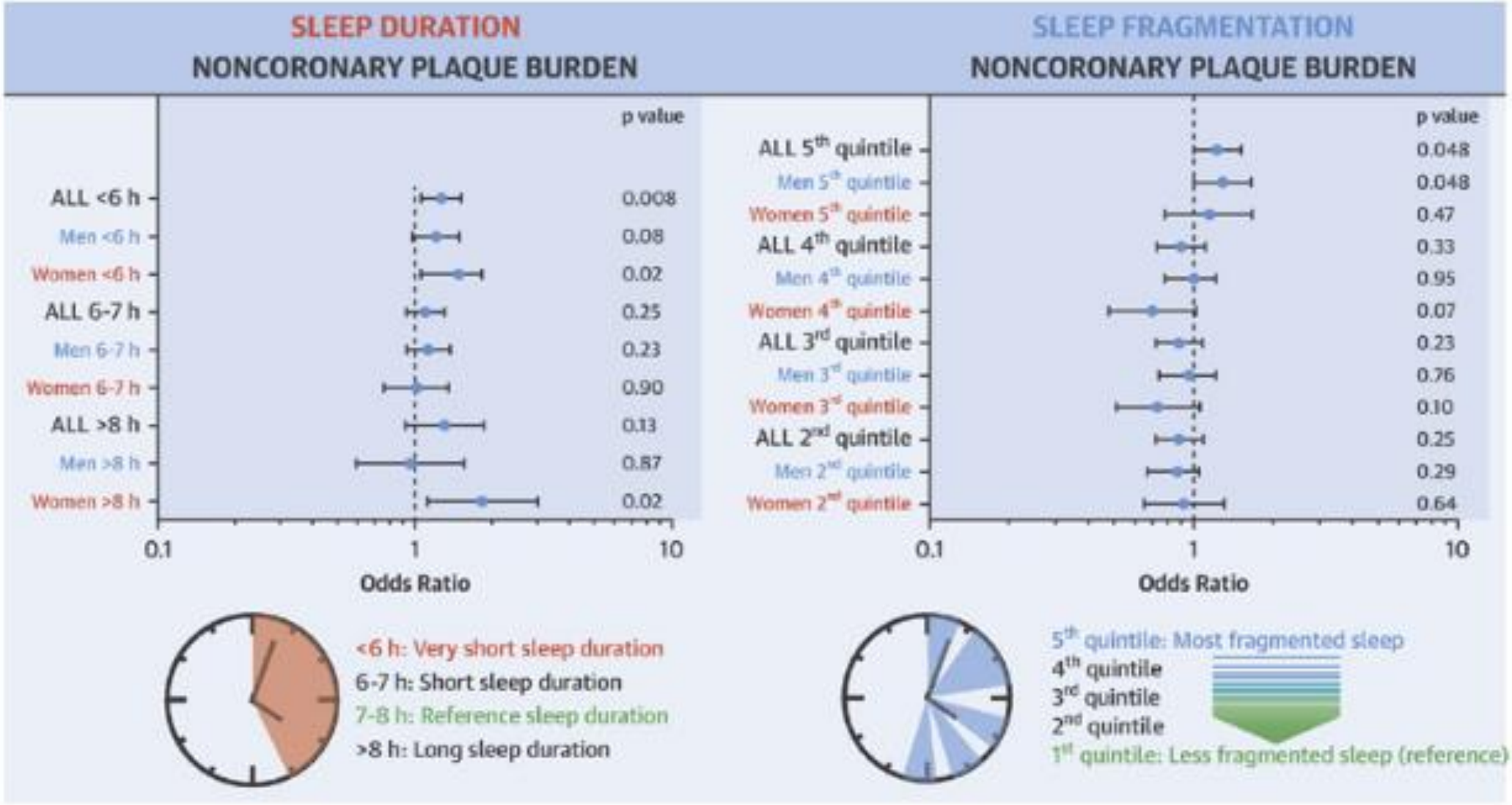
Table 1. Odds ratio, confidence intervals and AUC for OSA severity and other CAD risk factors in predicting CACS>400.

	OR	95% CI	P-value	AUC
Age	1.13	1.04-1.22	<0.01	0.78
OSA severity	1.04	1.01-1.06	<0.01	0.77
Gender	0.83	0.19-3.71	0.81	0.53
BMI	1.05	0.95-1.17	0.33	0.57
Hypertension	22.14	1.24-296	0.04	0.77
Diabetes Mellitus	3.16	0.65-15.5	0.16	0.57
Hyperlipidemia	2.01	0.55-7.42	0.29	0.59
Smoking history Current smoker vs Never smoked Ex-smoker vs Never smoked	3.45 3.01	0.67-17.77 0.59-15.23	0.14 0.18	0.61
Family history of CAD	0.70	0.11-4.28	0.70	0.54

Adjusted hazard ratios for CAC scores and cardiovascular disease events stratified by sleep apnea severity (binary; AHI ≥ or < 15 events/h).

	CVD			
	AHI < 15 events/h (n = 92)		AHI ≥ 15 events/h (n = 68)	
	HR* (95% CI)	*P*	HR* (95% CI)	*P*
CAC density	0.509 (0.323–0.801)	.0035	0.783 (0.434–1.410)	.4146
Agatston score	1.427 (1.238–1.644)	< .01	1.447 (1.221–1.715)	< .01
CAC volume	1.786 (1.478–2.158)	< .01	1.617 (1.270–2.058)	< .01

CENTRAL ILLUSTRATION: Sleep Duration and Quality Versus Subclinical Atherosclerotic Burden

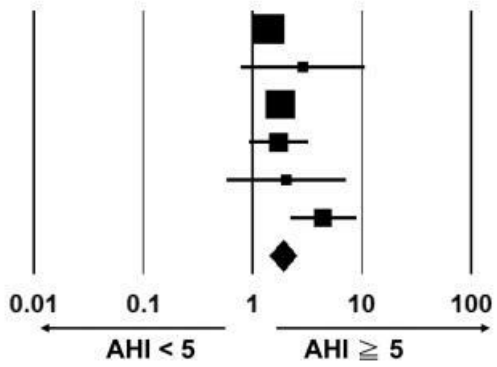


Domínguez, F. et al. J Am Coll Cardiol. 2019;73(2):134–44.

Association for AHI and CAC: meta-analysis

(A)

Study name 1st author (year)	Odds ratio	Lower limit	Upper limit	Z-value	p-Value	Relative weight
Kim (2020)	1.424	1.174	1.729	3.58	<.001	33.59
Bikov (2019)	2.917	0.791	10.761	1.61	0.108	4.31
Lutsey (2015)	1.811	1.451	2.261	5.25	<.001	32.06
Luyster (2014)	1.731	0.917	3.265	1.69	0.090	13.49
Arik (2013)	2.029	0.582	7.078	1.11	0.267	4.66
Sorajja (2008)	4.443	2.214	8.916	4.20	<.001	11.89
Pooled	**1.896**	**1.423**	**2.526**	**4.37**	**<.001**	

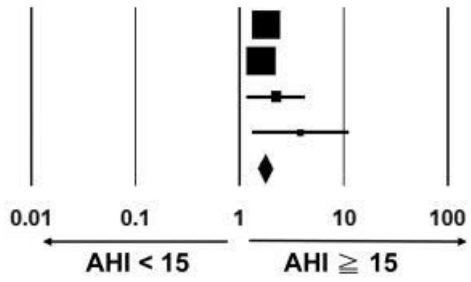


Heterogeneity test:

Q-value = 11.498, df=5, p-value= 0.042, I-square=56.51%

(B)

Study name 1st author (year)	Odds ratio	Lower limit	Upper limit	Z-value	p-Value	Relative weight
Kim (2020)	1.815	1.427	2.308	4.86	<.001	44.85
Lutsey (2015)	1.602	1.266	2.027	3.92	<.001	46.75
Luyster (2014)	2.230	1.165	4.267	2.42	0.015	6.16
Arik (2013)	3.818	1.302	11.199	2.44	0.015	2.24
Pooled	**1.763**	**1.501**	**2.071**	**6.90**	**<.001**	

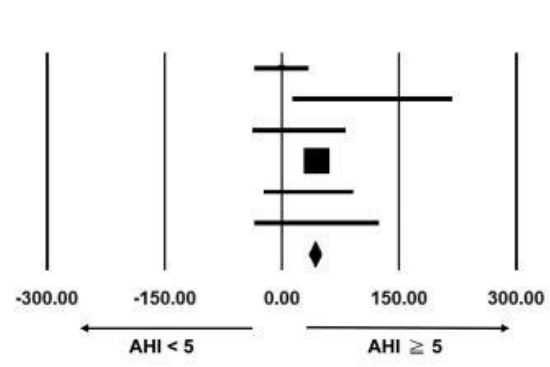


Heterogeneity test:

Q-value = 3.176, df=3, p-value=0.365, I-square=5.54%

(A)

Study name 1st author (year)	Difference in means	Lower limit	Upper limit	Z-value	p-Value	Relative weight
Kim (2020)	0.000	-34.627	34.627	0.000	1.000	4.44
Bikov (2019)	116.500	15.185	217.815	2.254	0.024	0.52
Luster (2014)	22.490	-36.816	81.796	0.743	0.457	1.52
Weinreich (2013)	44.760	37.106	52.414	11.462	<.001	90.98
Arik (2013)	34.850	-21.331	91.031	1.216	0.224	1.69
Kepez (2011)	44.520	-34.443	123.483	1.105	0.269	0.85
Pooled	**42.636**	**35.336**	**49.936**	**11.447**	**<.001**	

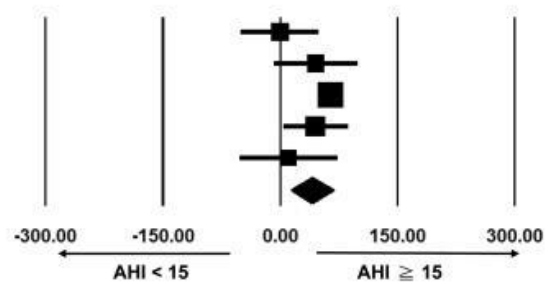


Heterogeneity test:

Q-value = 8.681, df=5, p-value= 0.122, I-square=42.40%

(B)

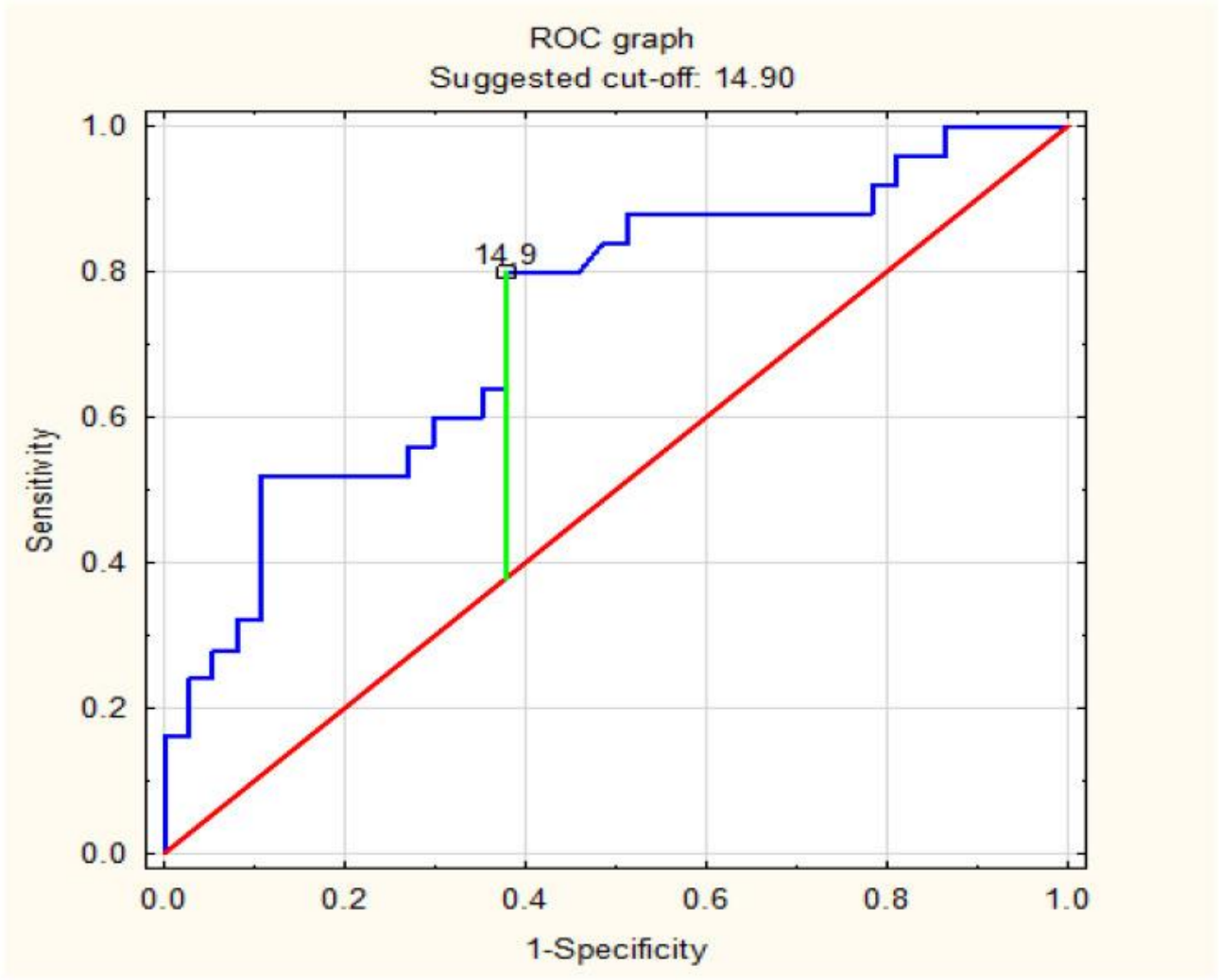
Study name 1st author (year)	Difference in means	Lower limit	Upper limit	Z-value	p-Value	Relative weight
Kim (2020)	0.000	-49.305	49.305	0.000	1.000	16.61
Luster (2014)	46.020	-6.526	98.566	1.717	0.086	15.48
Weinreich (2013)	65.160	59.858	70.462	24.089	<.001	35.38
Arik (2013)	45.810	4.897	86.723	2.195	0.028	19.94
Kepez (2011)	10.820	-51.410	73.050	0.341	0.733	12.60
Pooled	**40.673**	**13.250**	**68.096**	**2.907**	**0.004**	



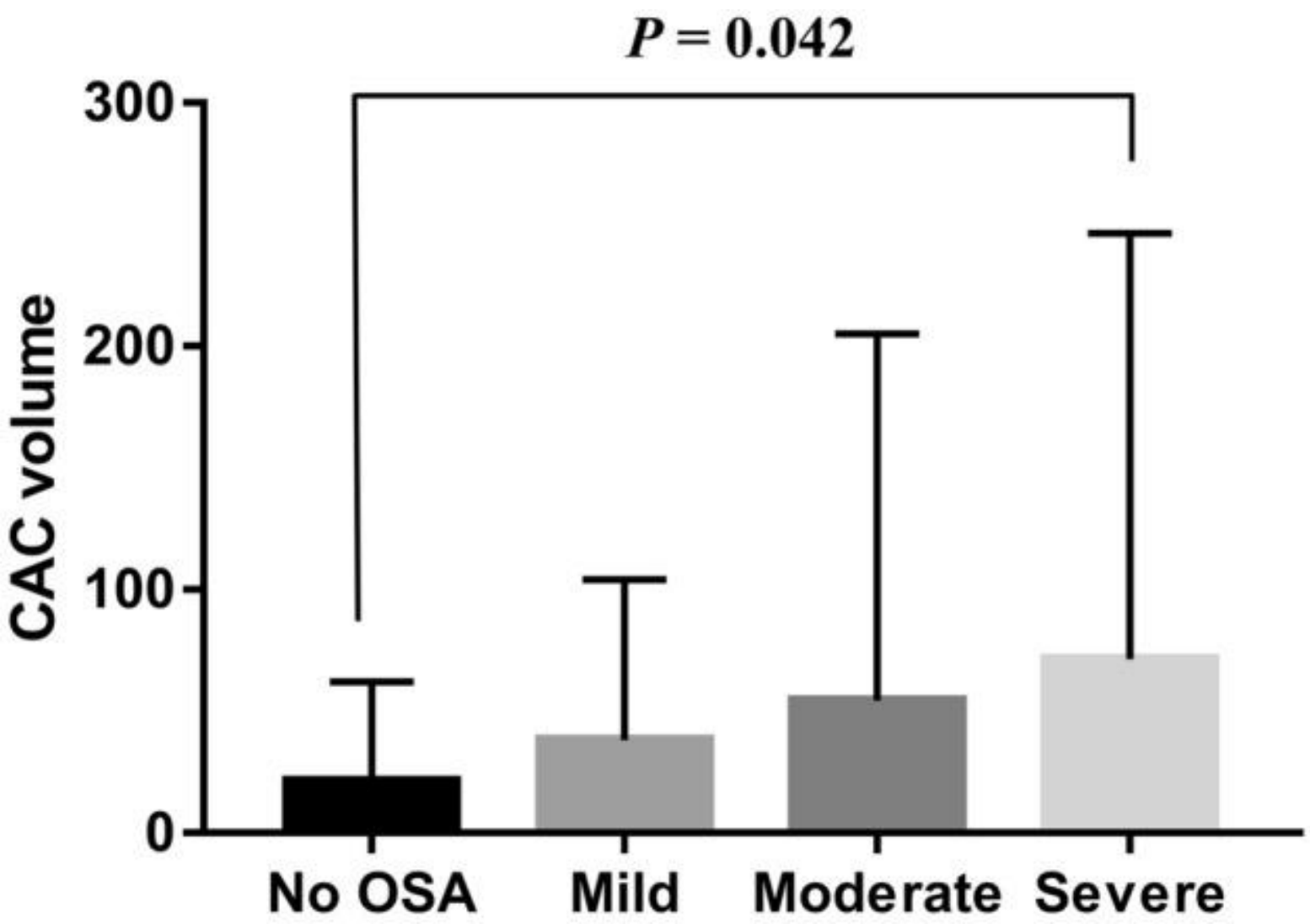
Heterogeneity test:

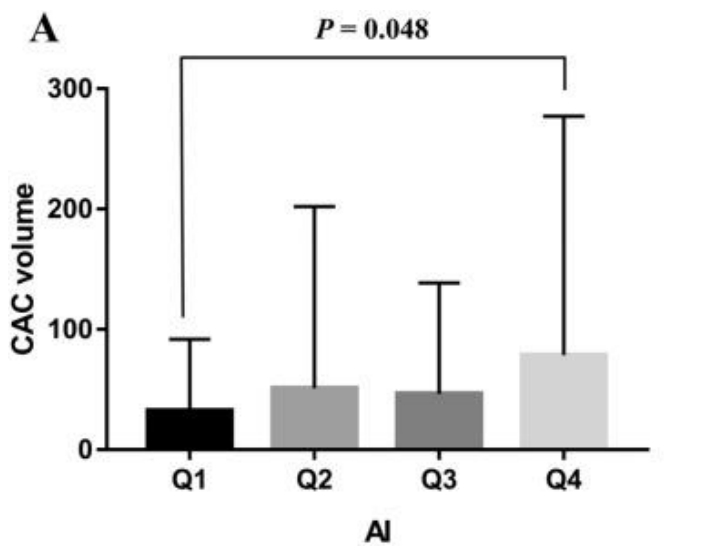
Q-value = 10.644, df=4, p-value= 0.031, I-square=62.42%

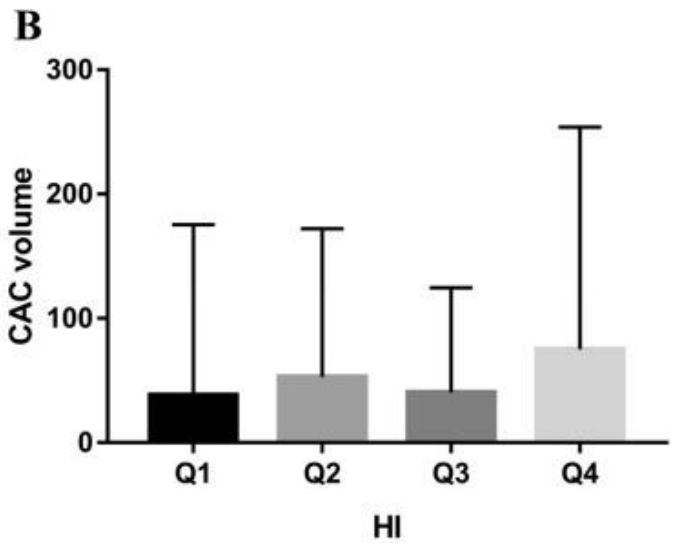
OSA as a predictor of CAC>400



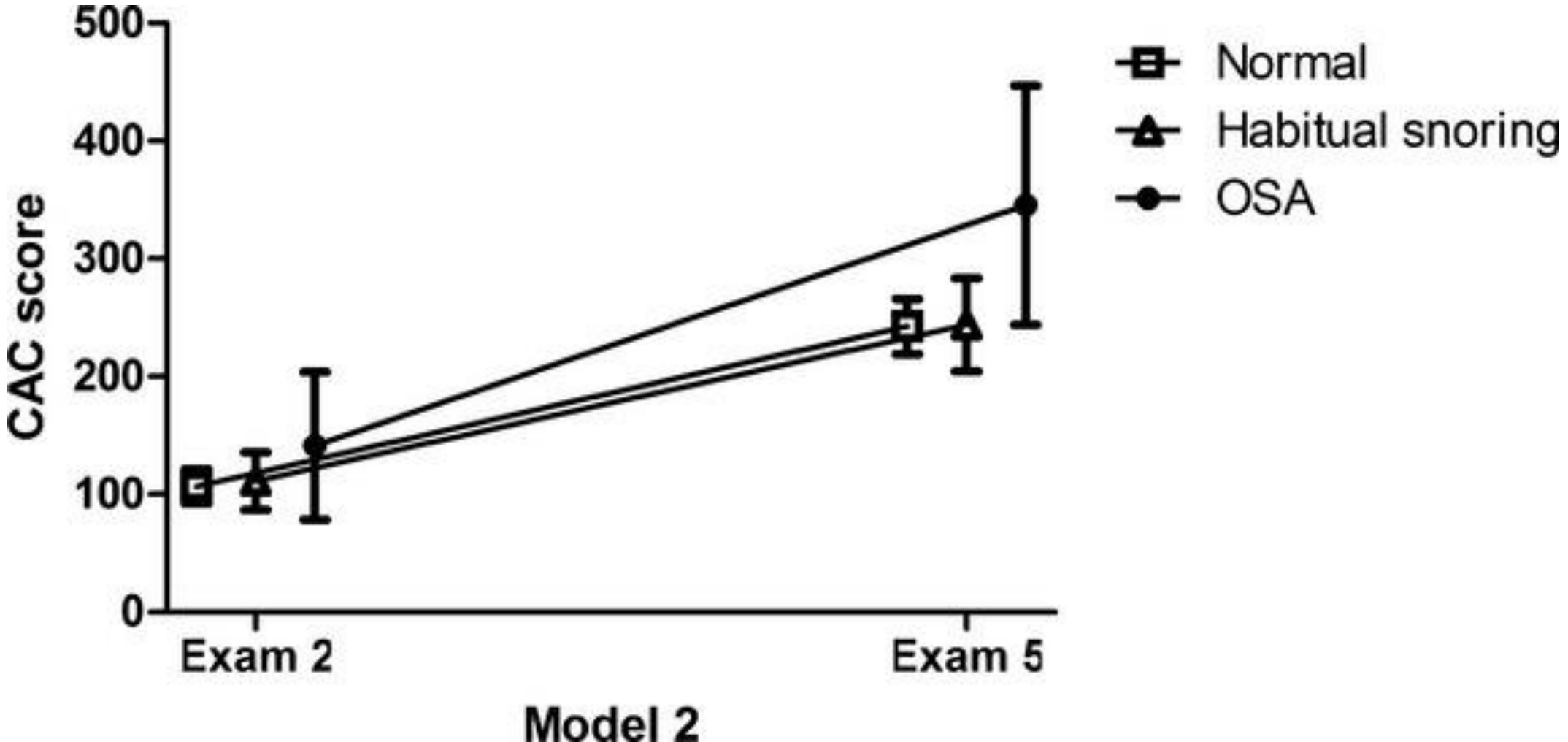
OSA and CAC volume among males



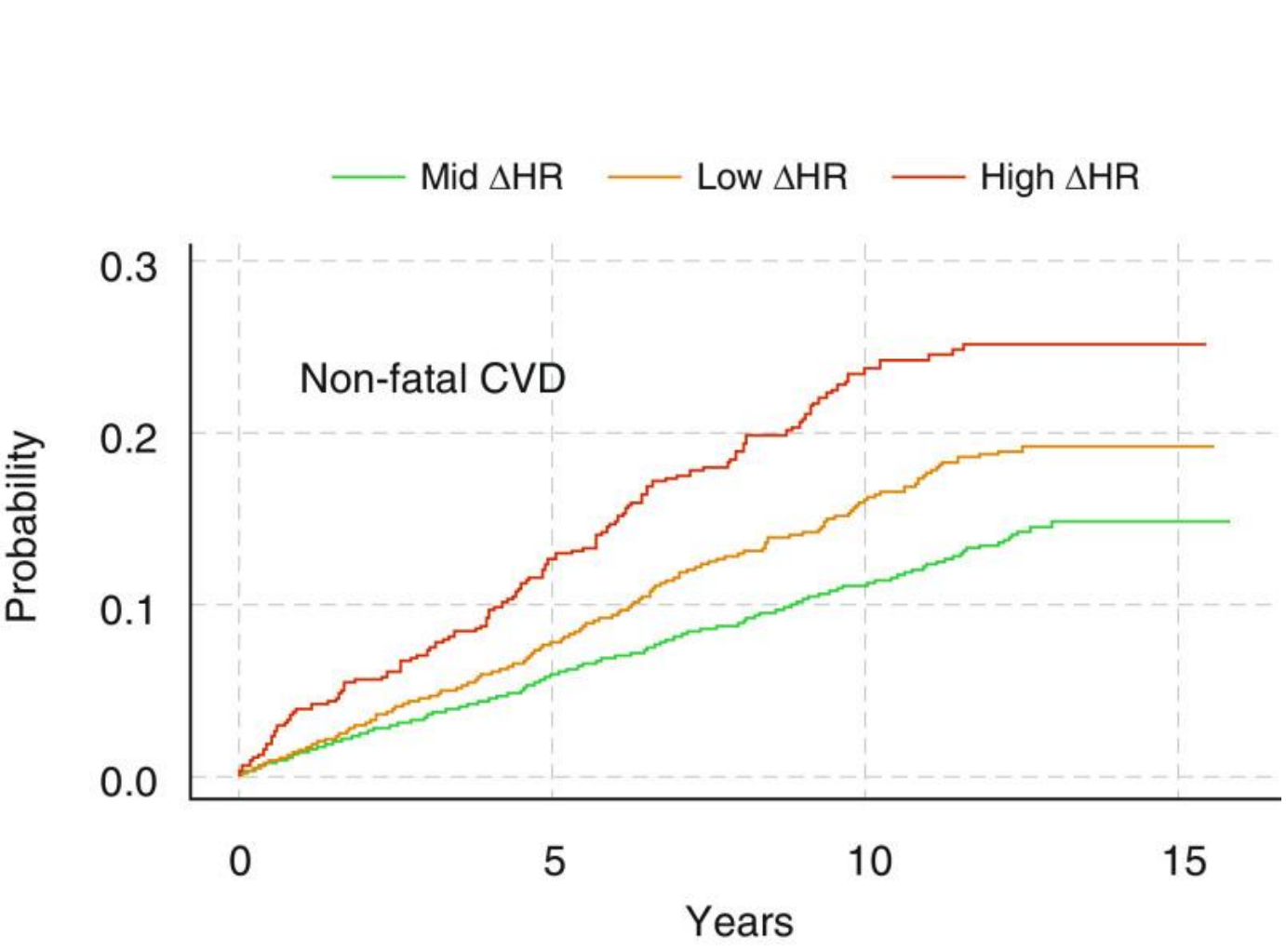


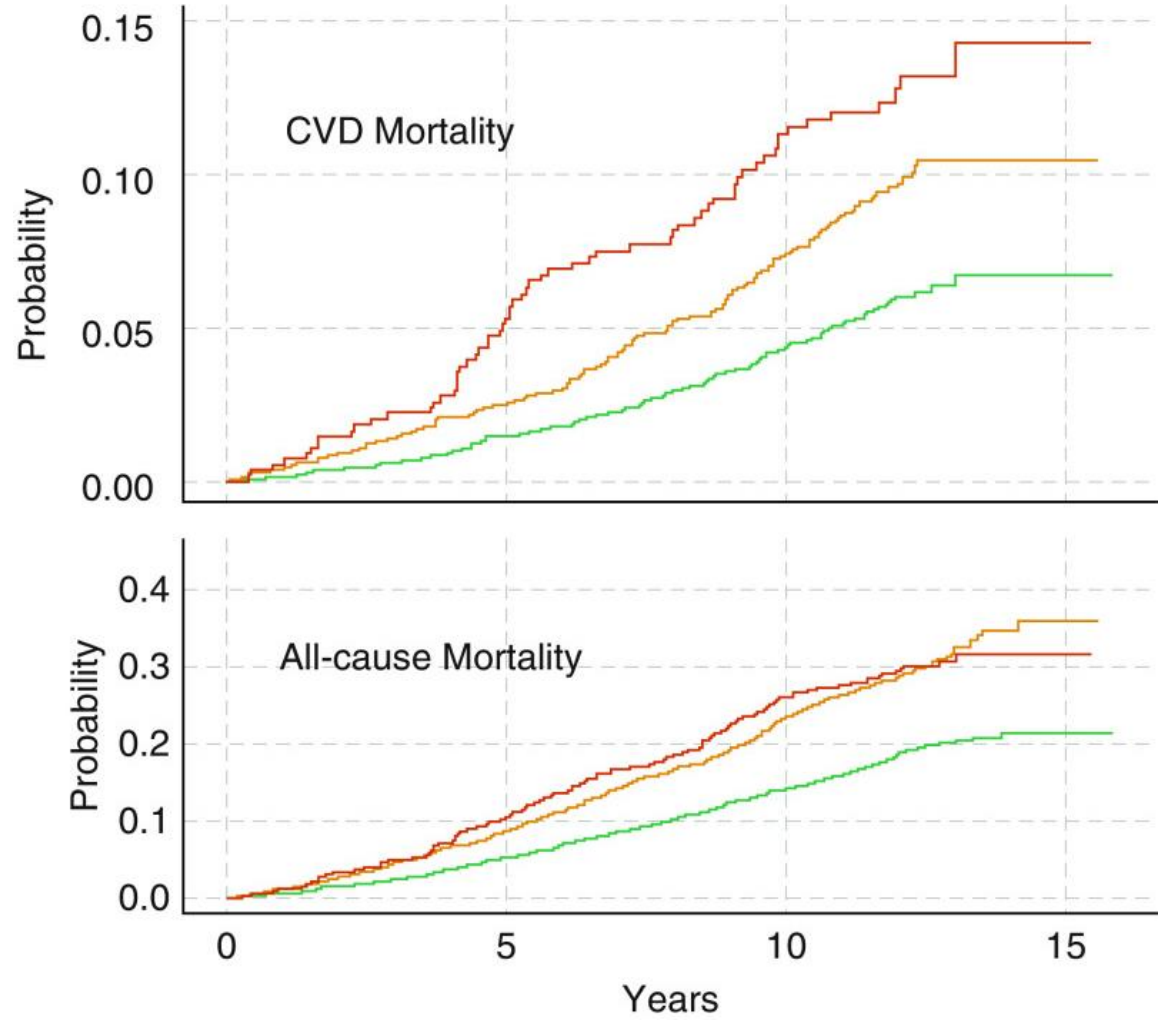


MESA: OSA and CAC progression

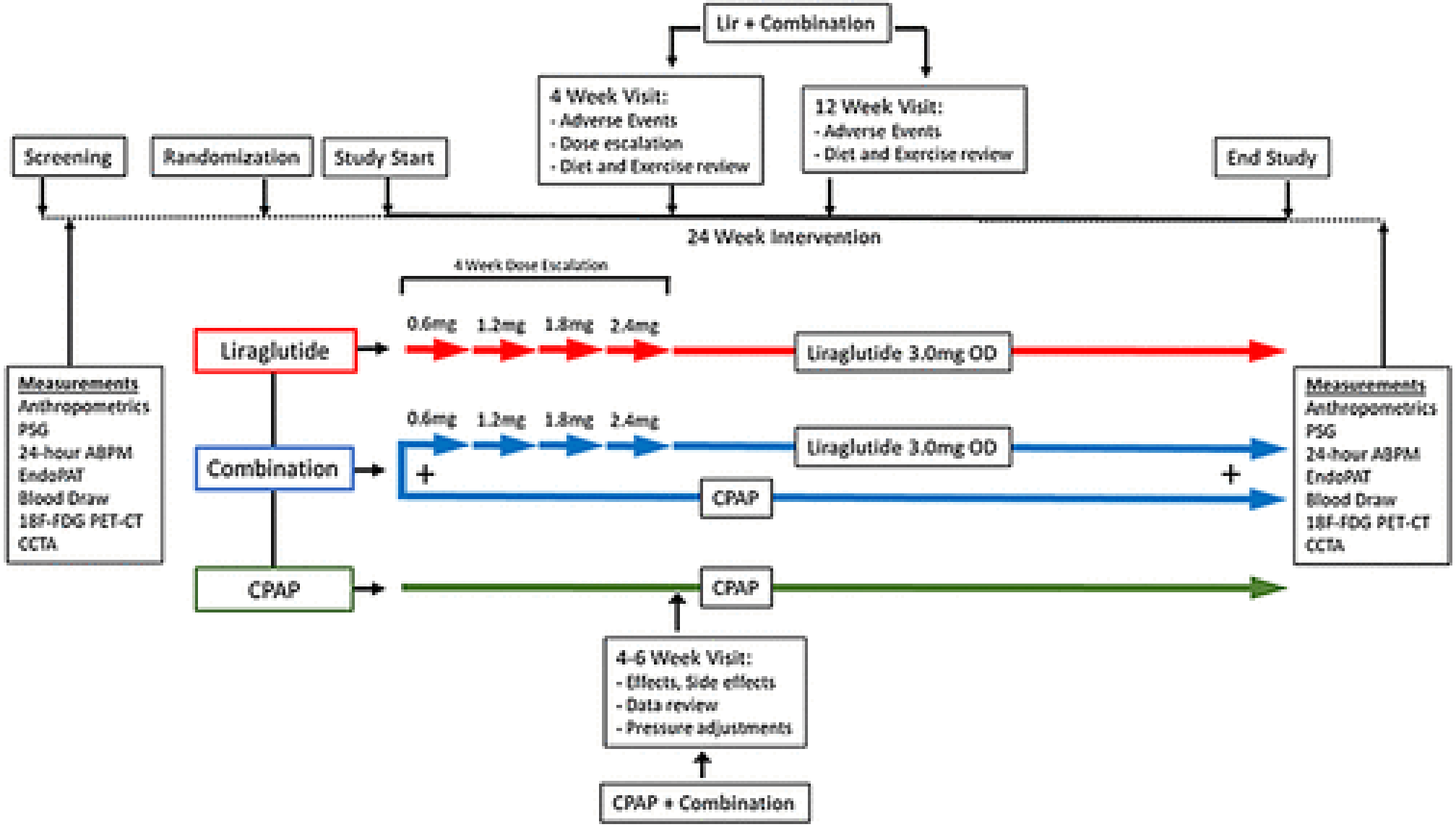


OSA-specific pulse-rate response and CVD

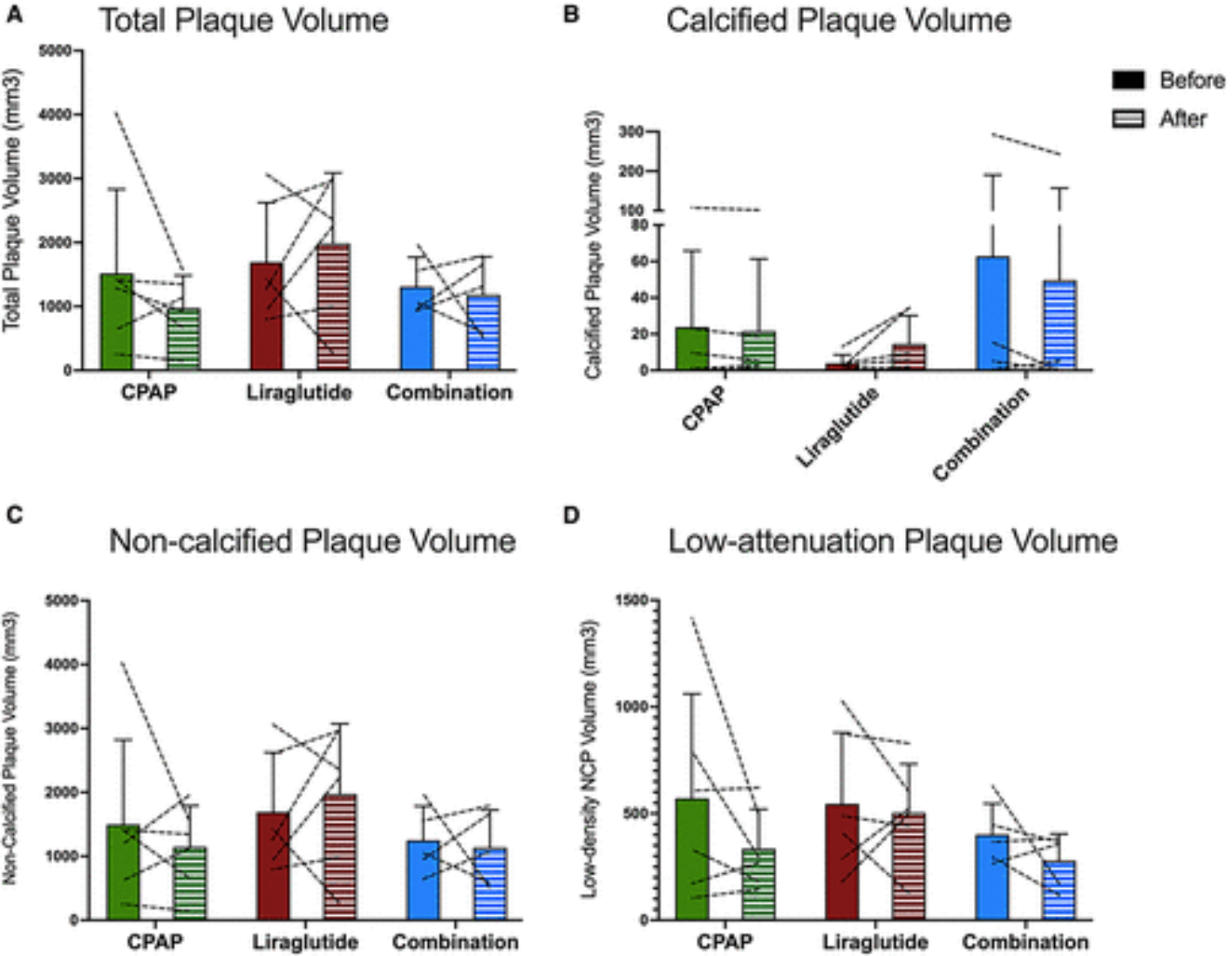


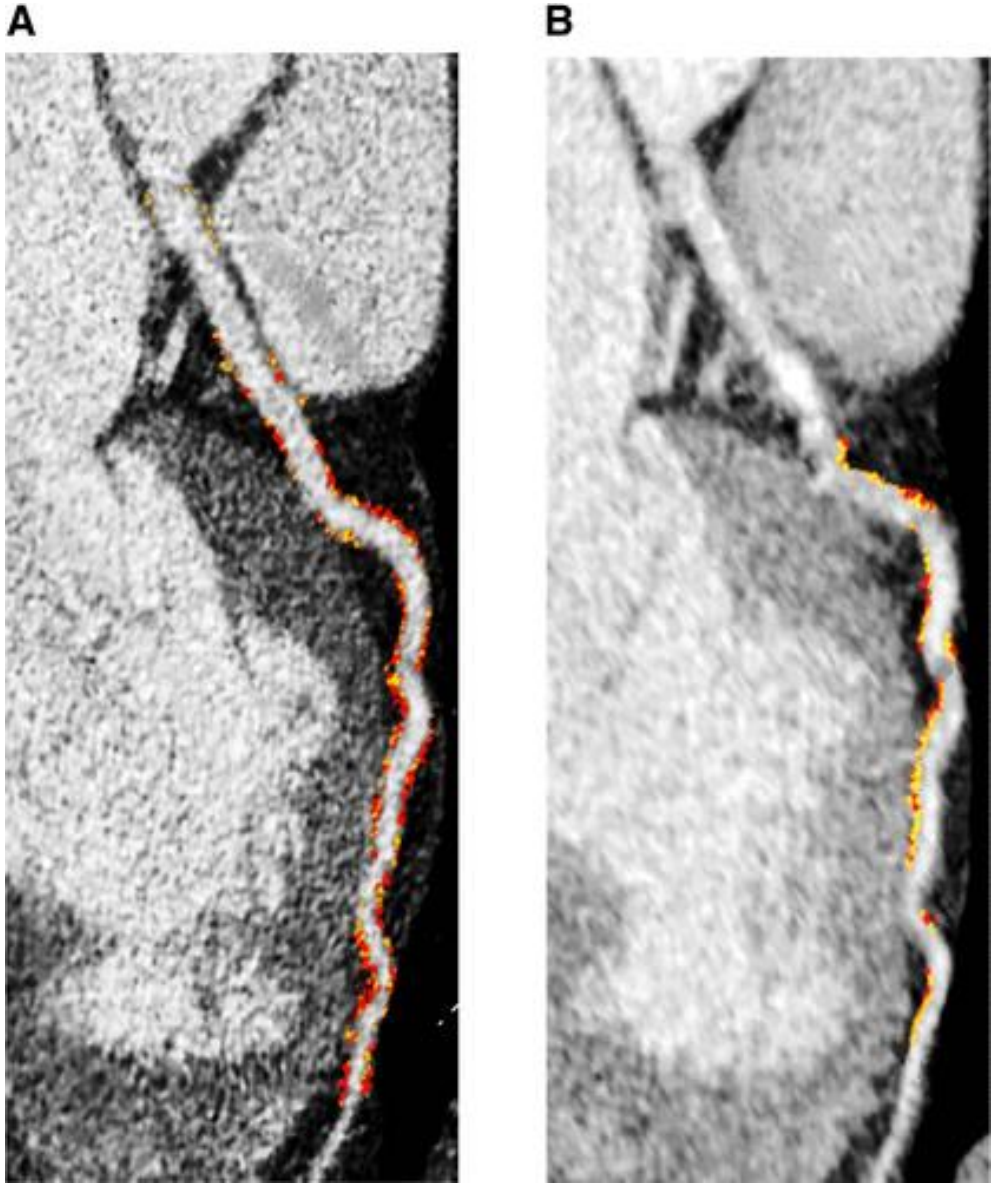


CPAP and GLP-1s for CVD in OSA



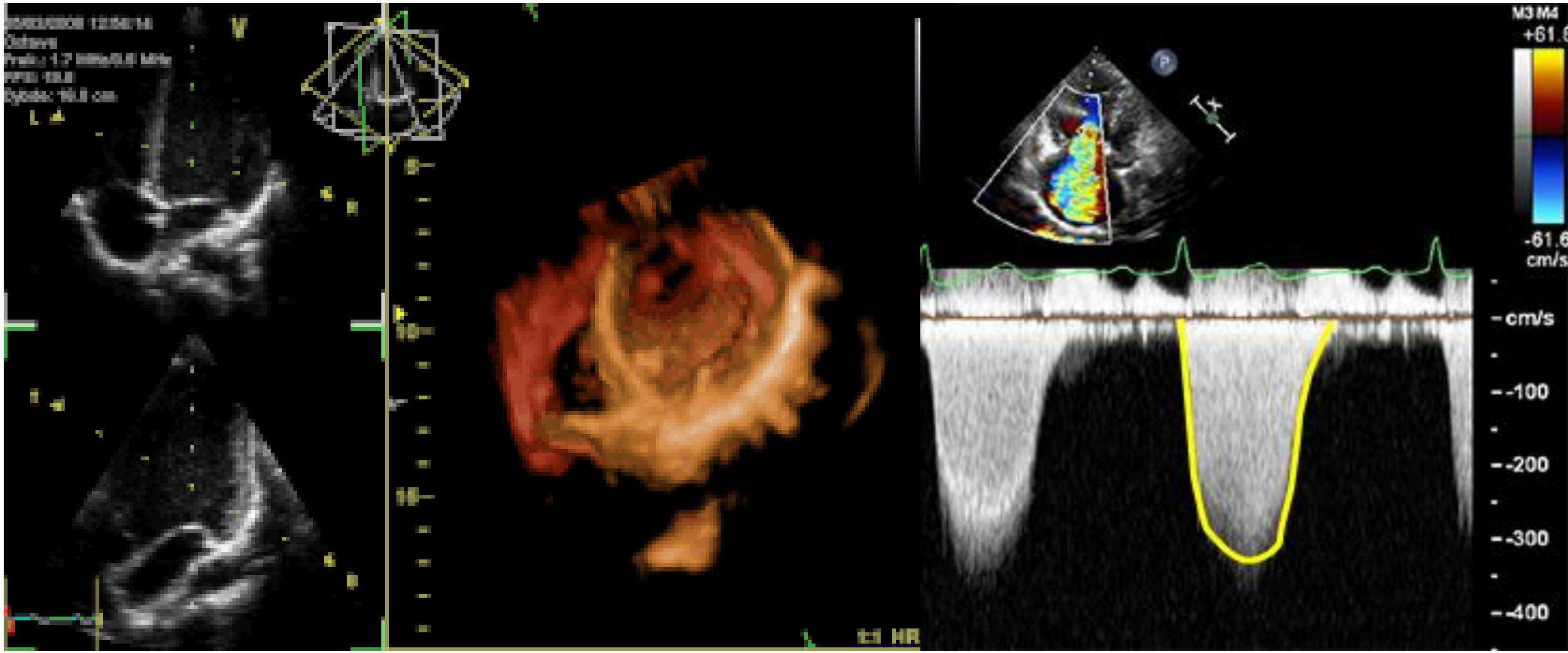
CPAP but not GLP-1s improve CVD in OSA



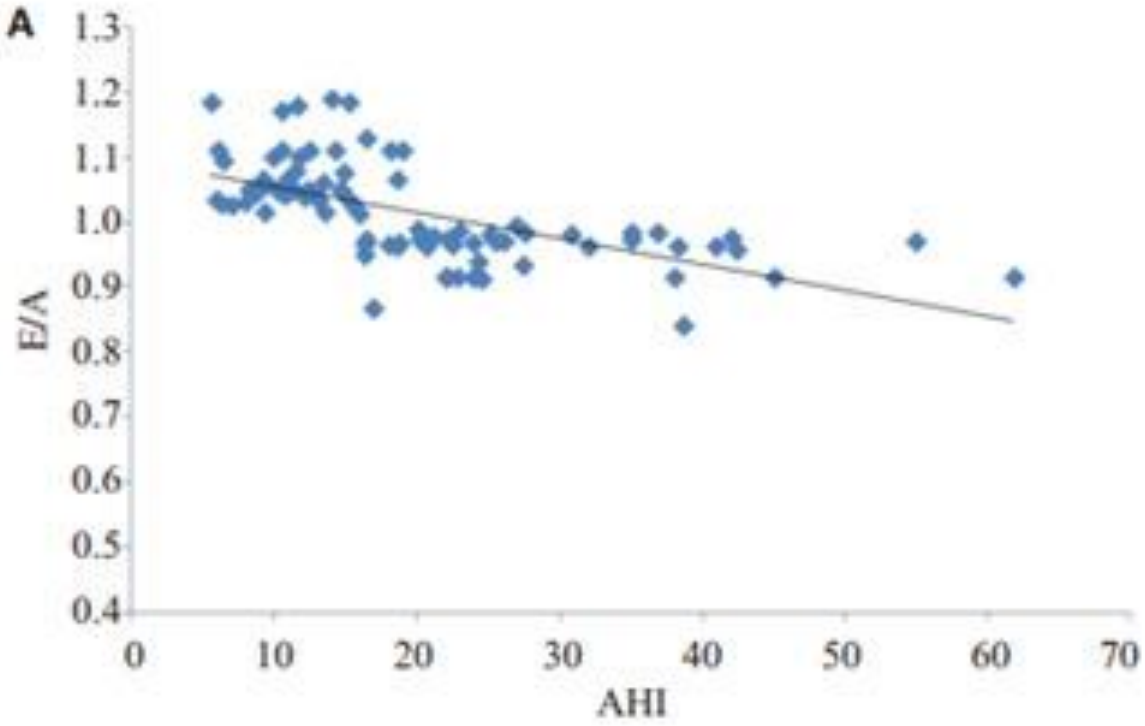


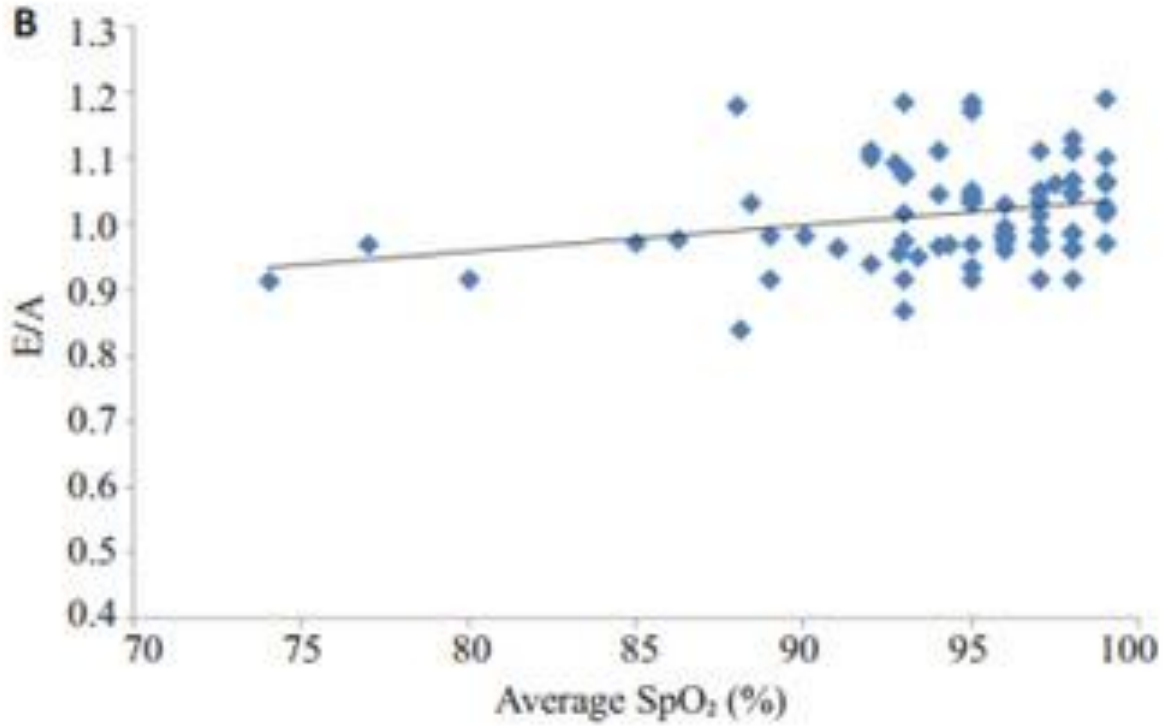
Echo

What’s an Echo?

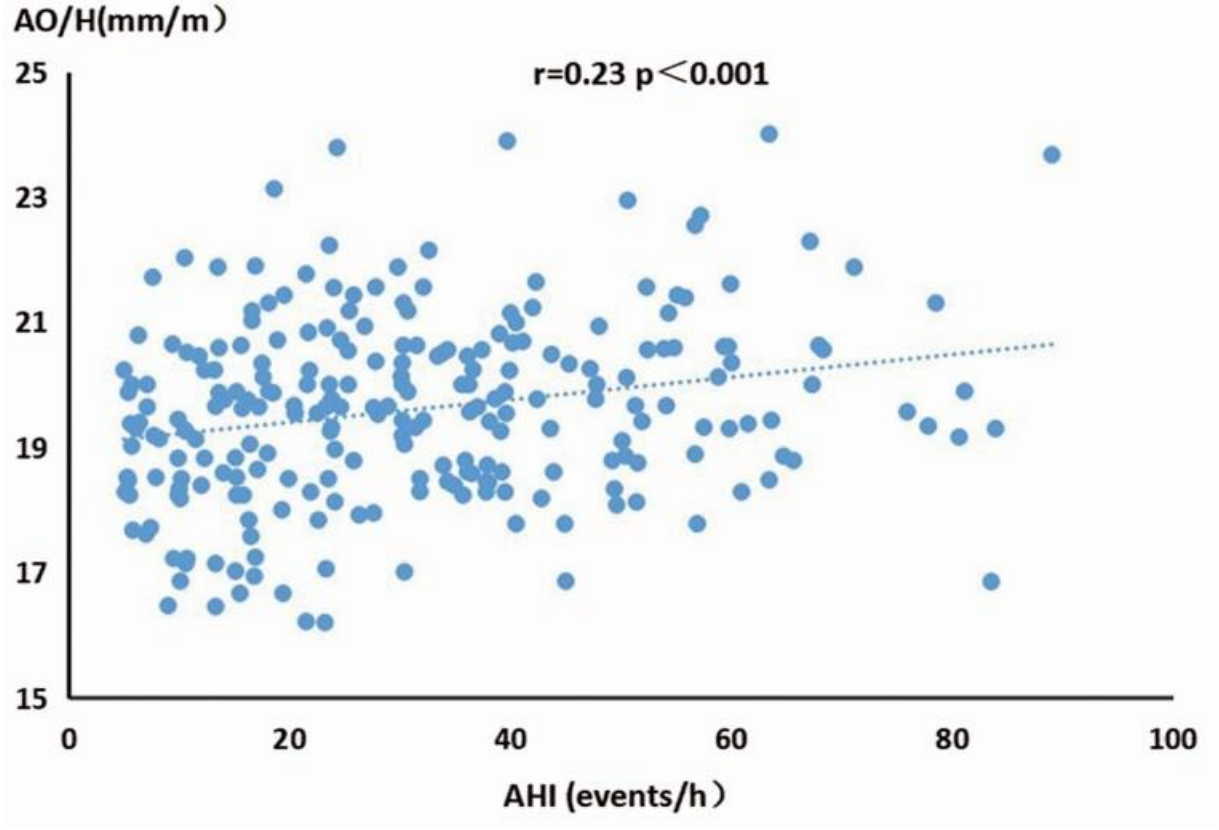


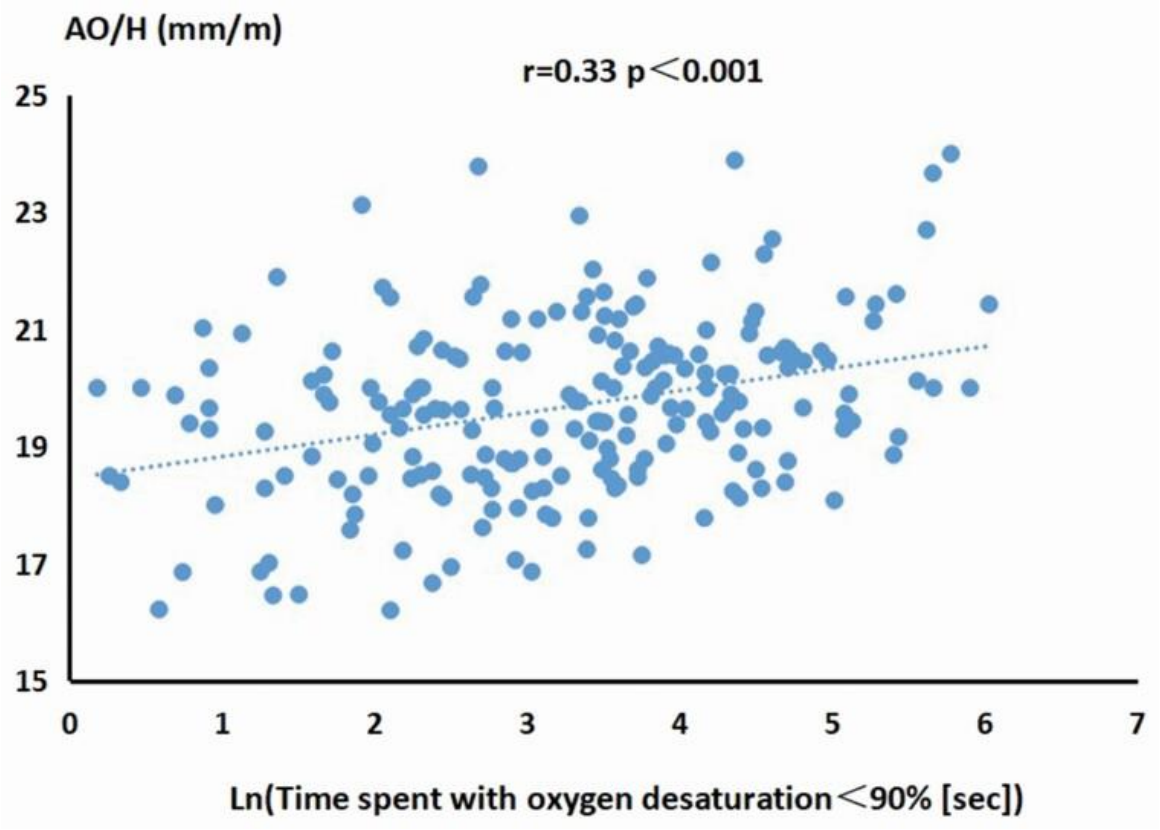
Diastolic Dysfunction and Osa severity



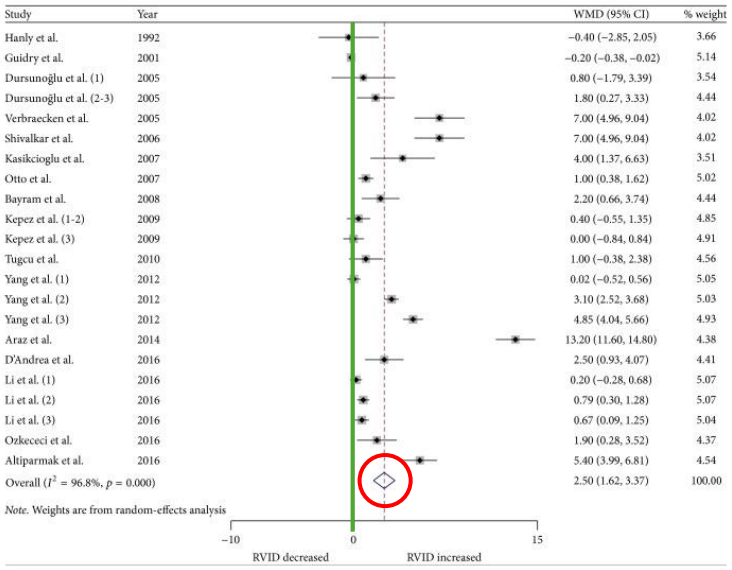


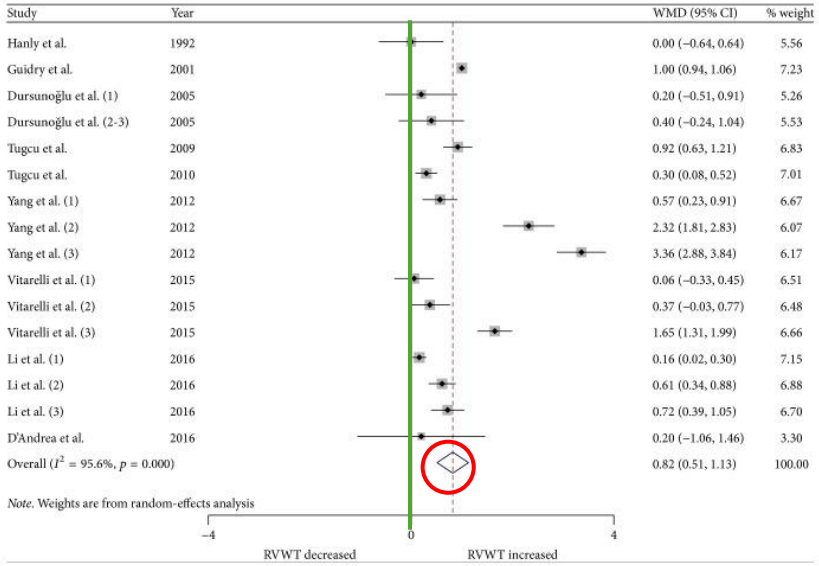
Aortic Root Diameter among HTN and OSA

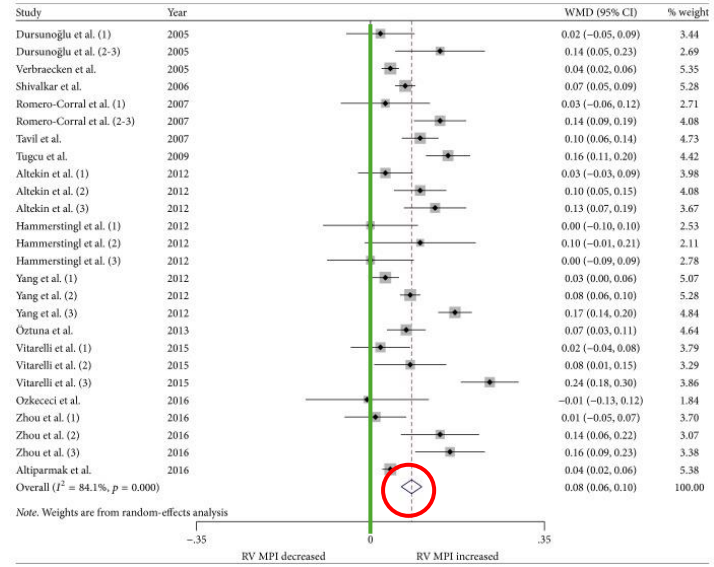


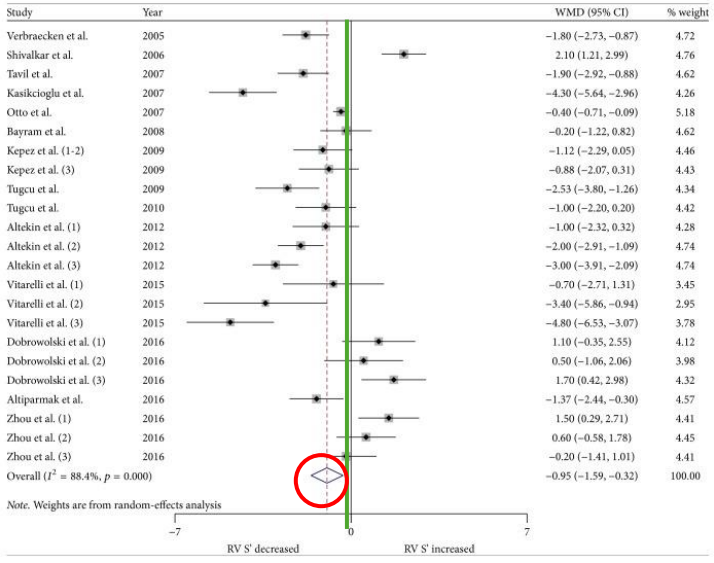


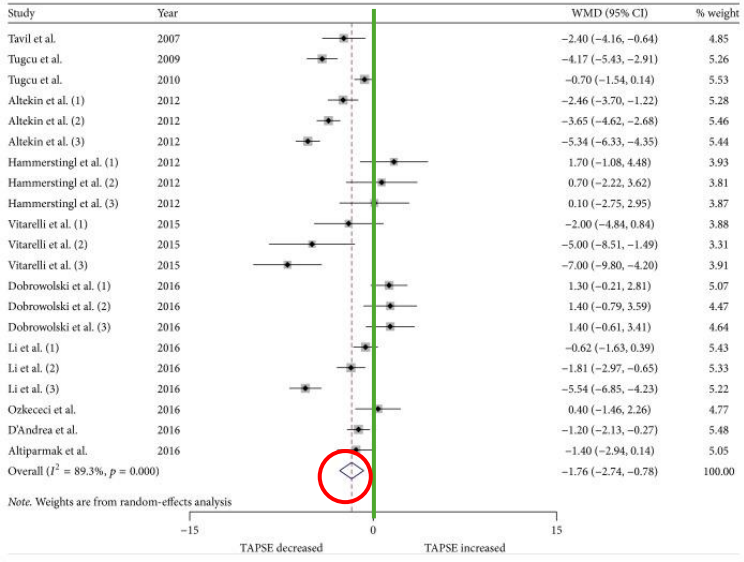
RV remodeling in OSA: Meta-analysis

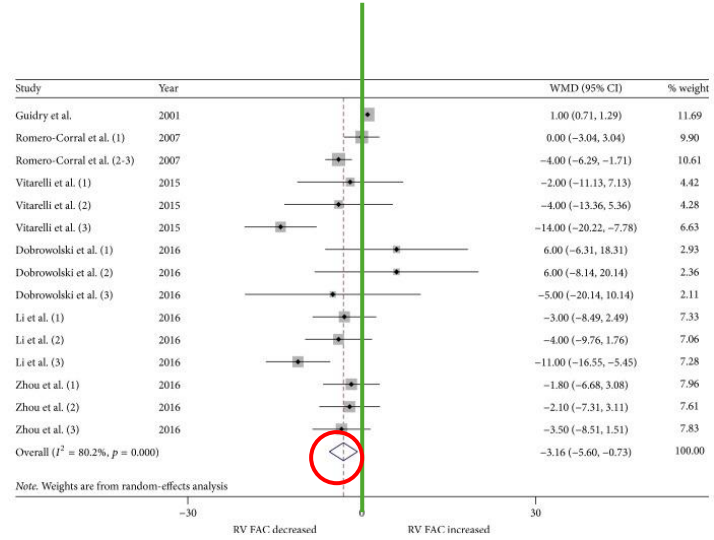




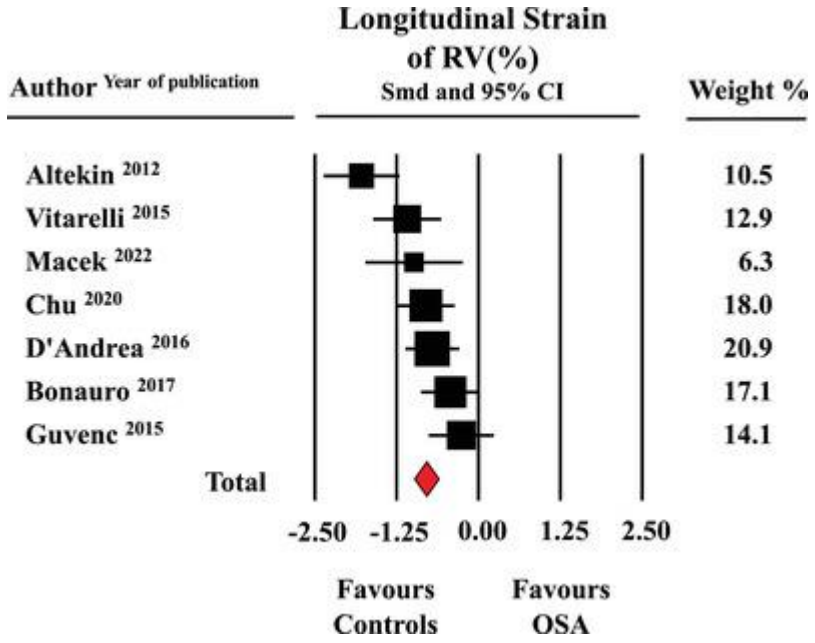


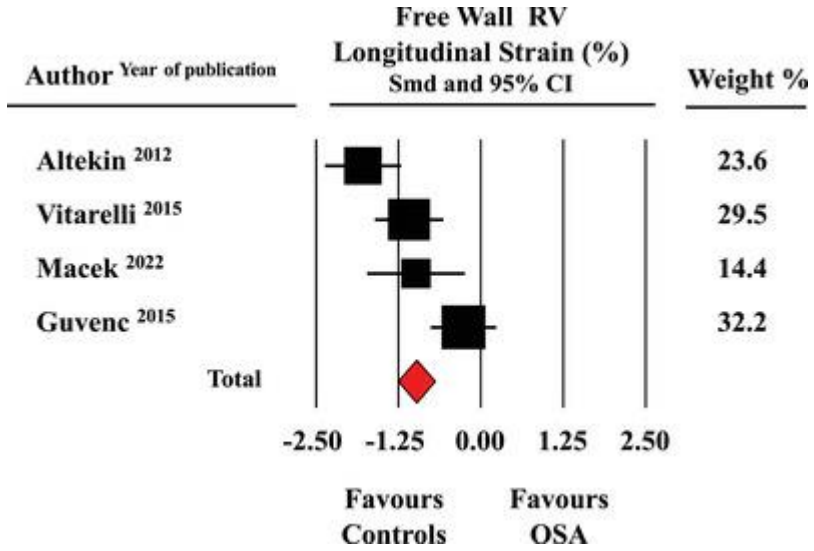


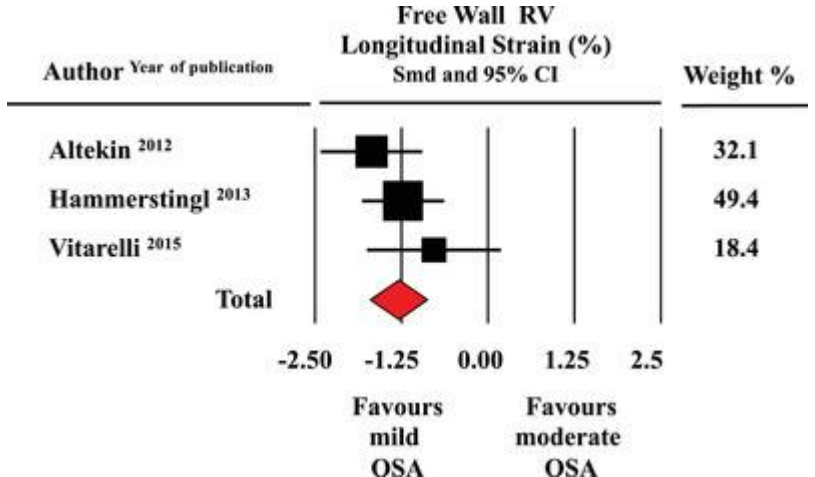


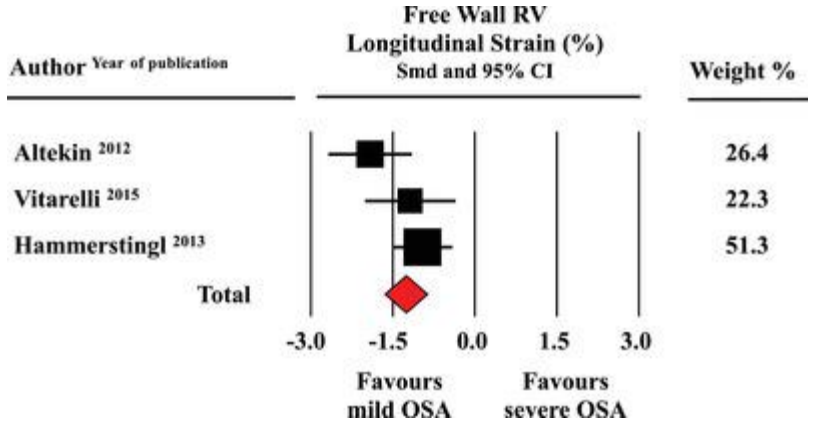


RV strain and function: Meta-analysis

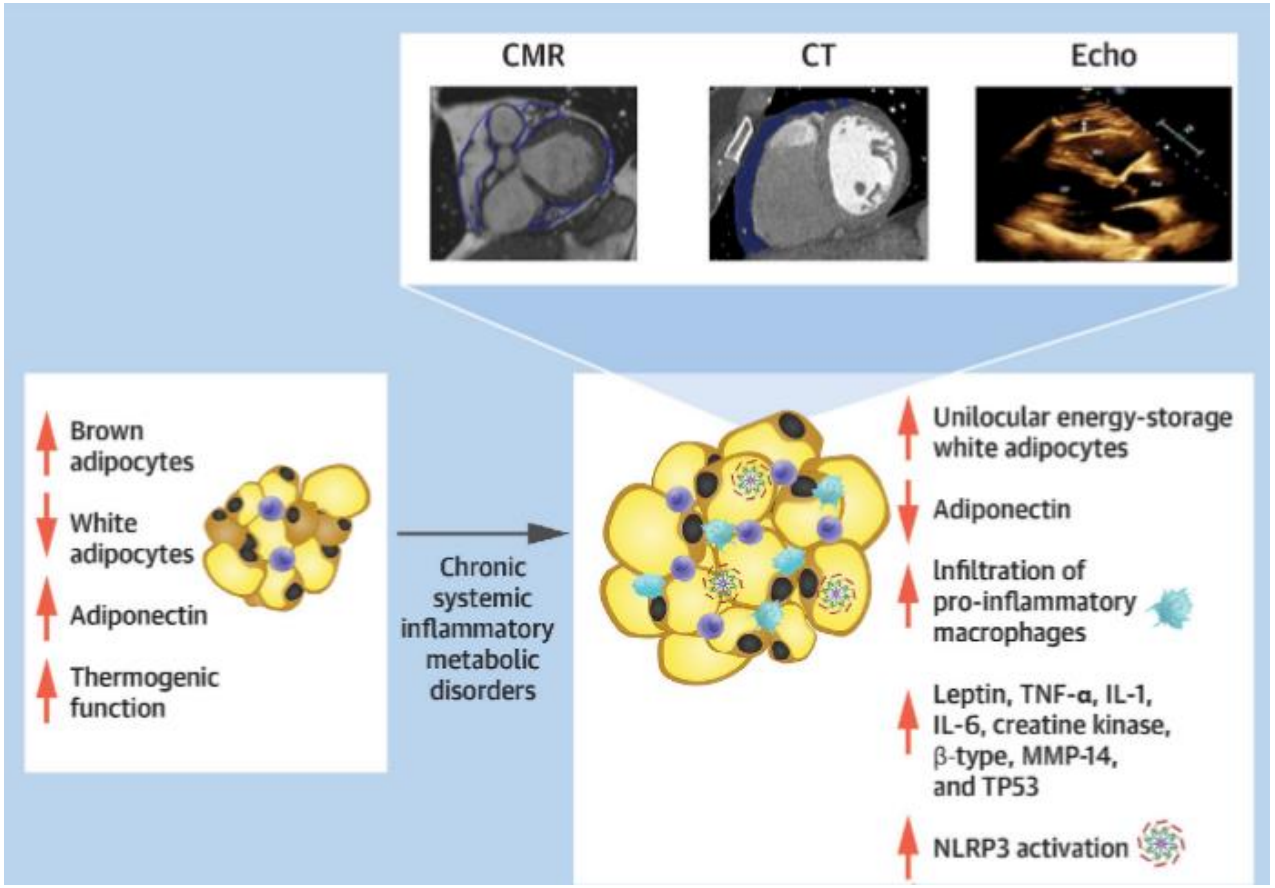




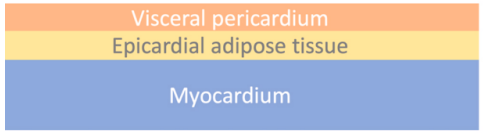




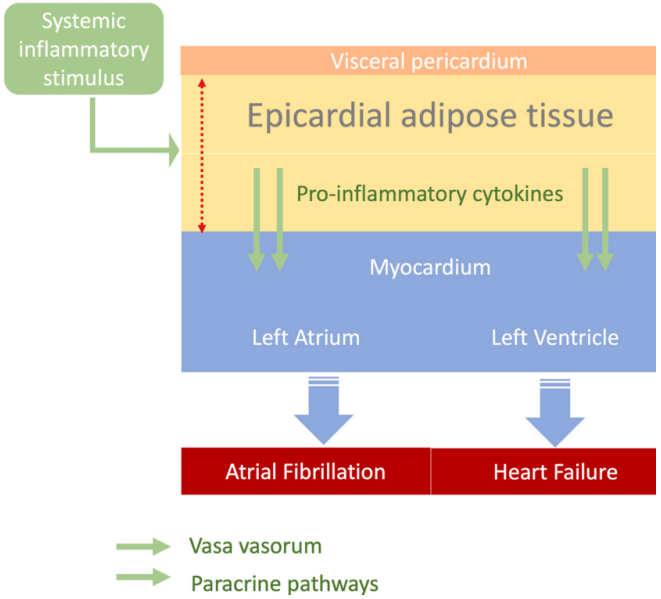
Epicardial Adipose Tissue and OSA



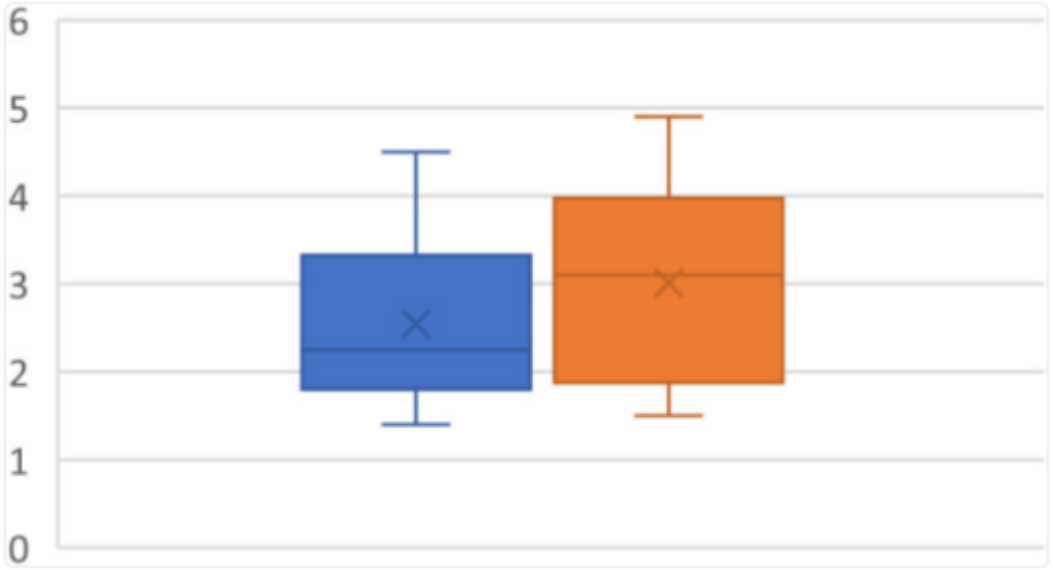
Normal anatomy and function

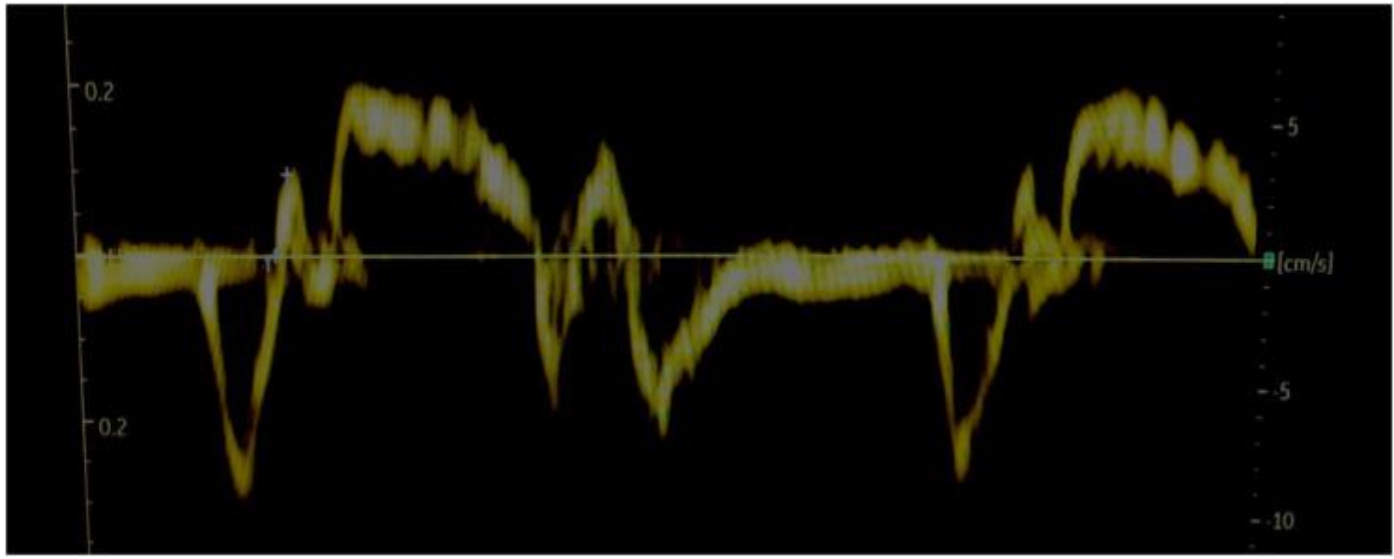


Inflammatory states



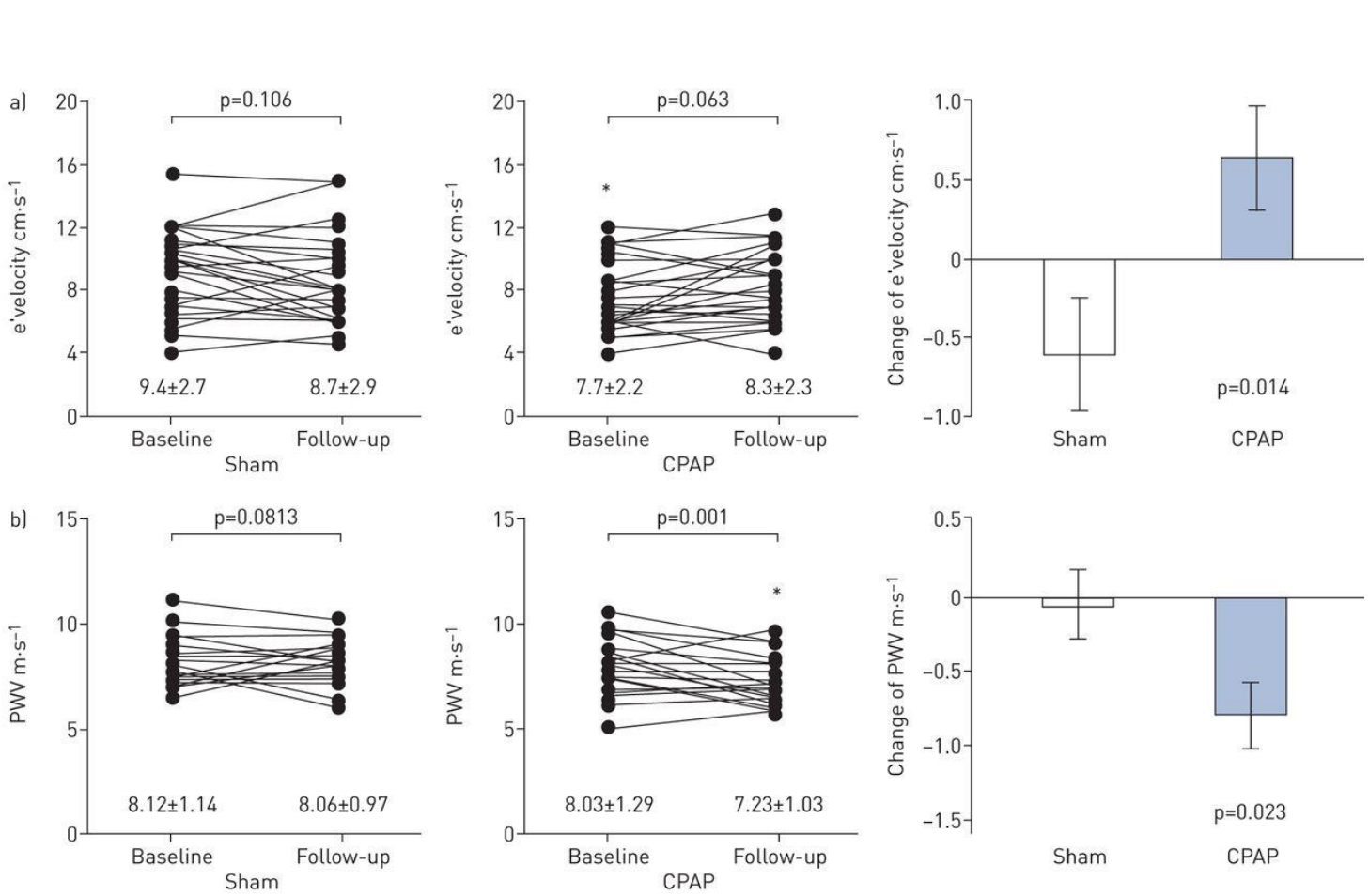
Diastolic Dysfunction and CPAP

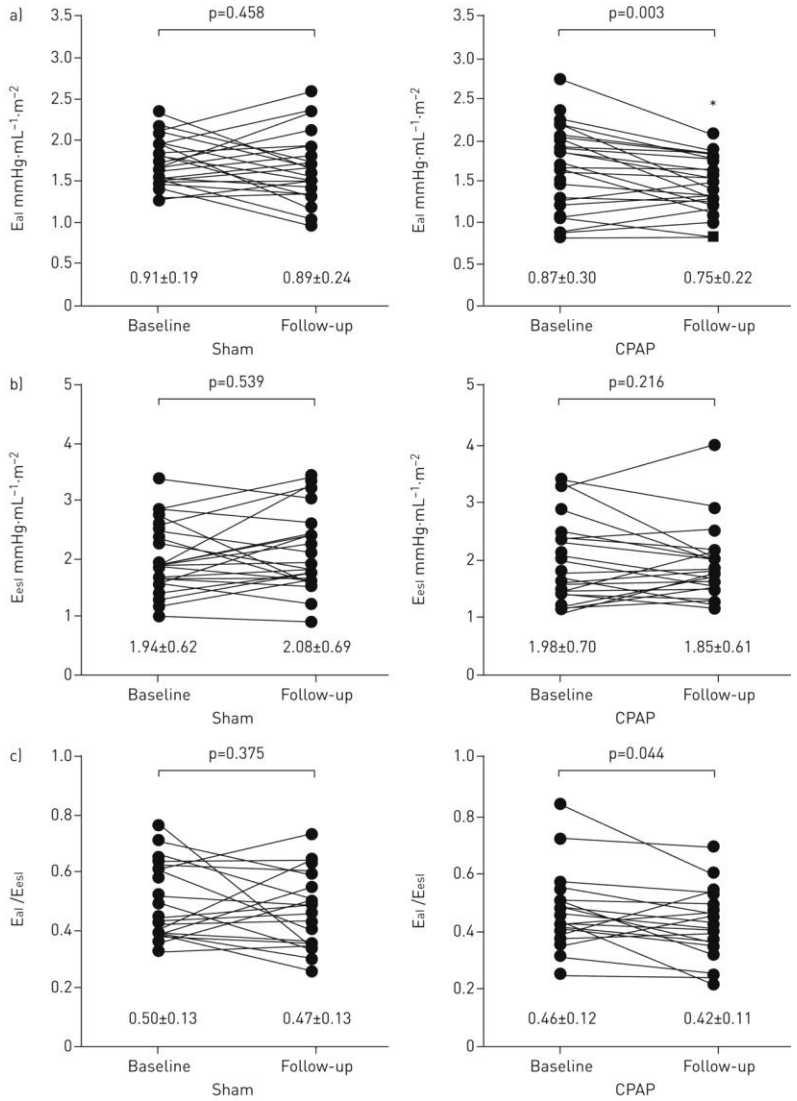




Isovolumetric acceleration (MS^2) with pre-CPAP (blue) and post CPAP (Orange)

CPAP on LV diastolic dysfunction





Acute CPAP effects on TTE

Parameter (unit)	Paired differences (baseline minus CPAP)				*P*
	Mean±SD	SEM	95% CI		
			Lower	Upper	
LVEDD (cm)	0.065±0.646	0.062	−0.059	0.189	0.299
LVESD (cm)	−0.065±0.606	0.059	−0.181	0.051	0.271
LVEDV (mL)	4.15±12.508	1.209	1.757	6.551	0.001
LVESV (mL)	0.57±6.423	0.621	−0.666	1.796	0.365
LA_{vol} (mL)	3.76±13.878	1.342	1.098	6.419	0.006
RA_{area} (cm^2)	1.05±2.812	0.272	0.516	1.594	<0.001
IVC_{max} (cm)	−0.44±0.388	0.038	−0.519	−0.371	<0.001
IVC_{min} (cm)	−0.42±0.334	0.0322	−0.485	−0.358	<0.001
IVC variability (%)	8.44±17.662	1.707	5.053	11.823	<0.001
TRV_{max} (cm/s)	38.36±39.059	3.776	30.873	45.846	<0.001
TAPSE (cm)	0.17±0.476	0.047	0.075	0.260	0.001
RVOTVTI (cm)	1.92±3.304	0.321	1.287	2.560	<0.001
SV (mL)	5.44±13.218	1.284	2.890	7.981	<0.001
EF (%)	1.94±7.475	0.723	0.507	3.372	0.008
LVOTVTI (cm)	1.64±4.141	0.402	0.840	2.435	<0.001

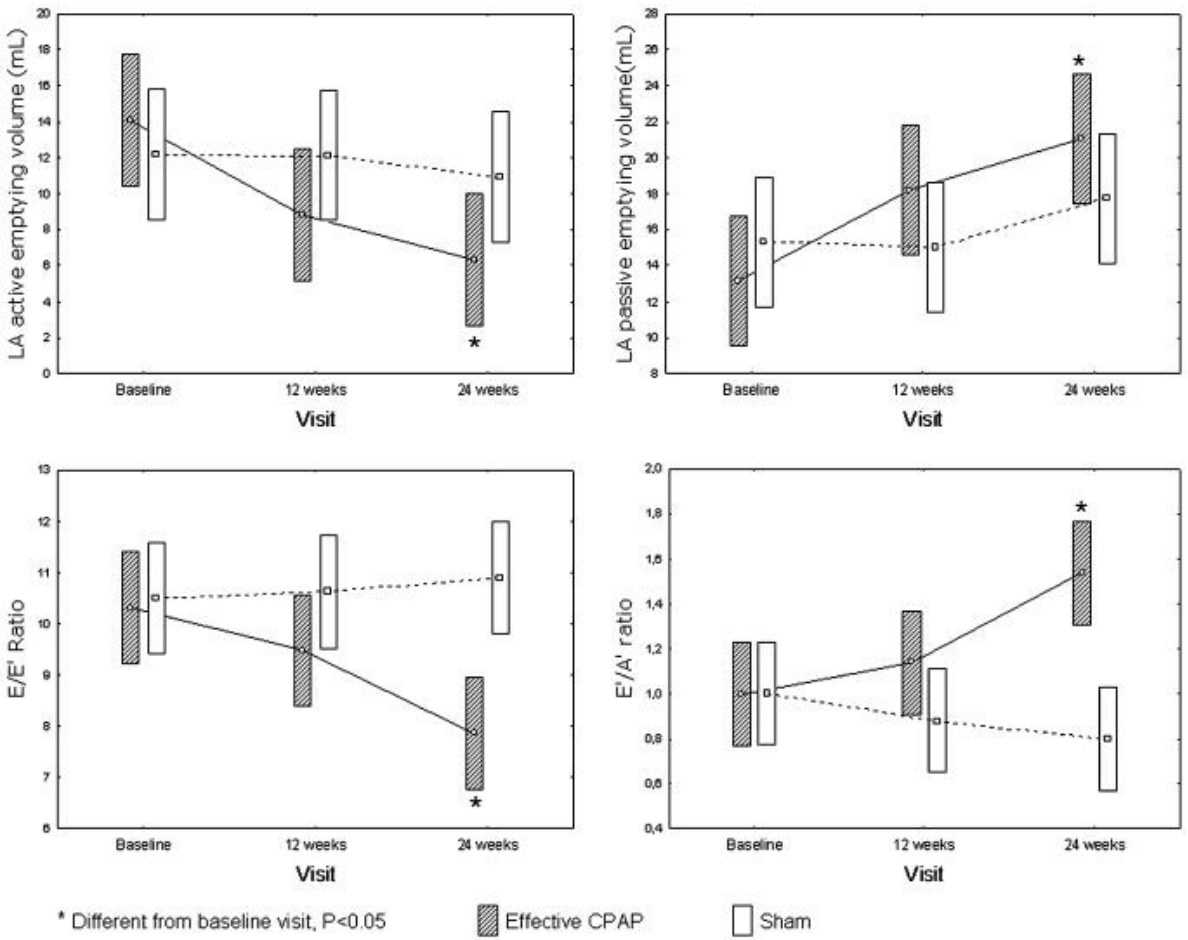
Differences in the left heart diastolic function measurements of the patients before and after continuous positive airway pressure therapy

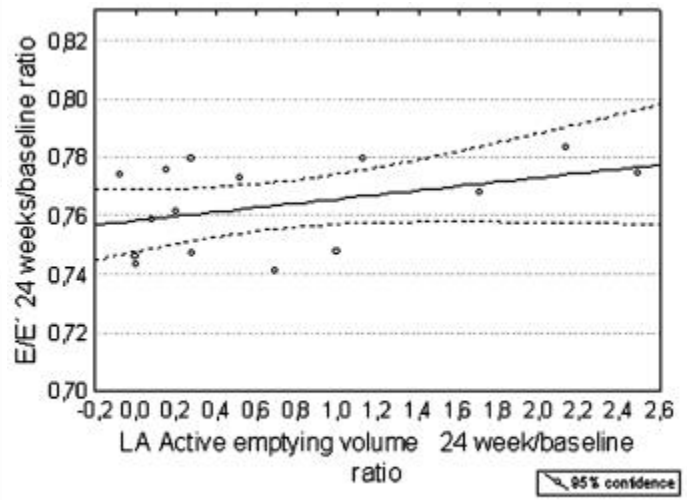
Parameter	Paired differences(baseline minus CPAP)				*P*
	Mean±SD	SEM	95% CI		
			Lower	Upper	
E/A ratio	0.26±0.296	0.030	−0.033	0.085	0.391
E/E' ratio	0.44±3.976	0.388	−0.333	1.206	0.263

Differences in the measurements reflecting right heart pressure effect before and after continuous positive airway pressure in patients with continuous positive airway pressure therapy <10 cmH_2O (n=73) and ≥10 cmH_2O (n=34)

CPAP (cmH_2O)	Parameter (unit)	Paired differences(baseline minus CPAP)				*P*
		Mean±SD	SEM	95% CI		
				Lower	Upper	
<10	TRV_{max}(cm/s)	34.84±36.846	4.313	26.245	43.439	<0.001
≥10	TRV_{max}(cm/s)	45.91±43.037	7.381	30.896	60.928	<0.001
<8	TRV_{max}(cm/s)	35.24±38.277	4.983	25.262	45.212	<0.001
≥8	TRV_{max}(cm/s)	42.20±40.068	5.783	30.564	53.832	<0.001

3D Echo and CPAP effects on LV parameters





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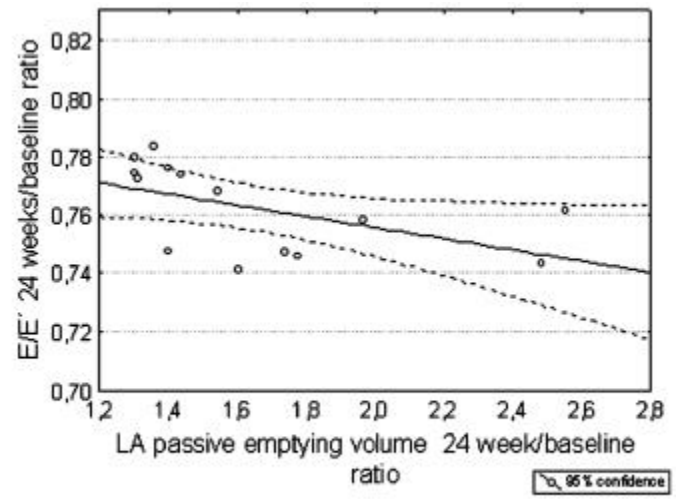


B

Subclinical LV GLS and OSA by severity

Patient's echocardiographic data before and after treatment with CPAP

	Baseline	Follow-up	*p* value
LVEF (%)	70.0 ± 5.3	69.0 ± 4.6	0.084
Mitral E/A	1.0 ± 0.4	1.0 ± 0.3	0.405
$e^{'}$ (m/sec)	0.068 ± 0.018	0.069 ± 0.018	0.267
$E/e^{'}$	11.2 ± 3.3	11.3 ± 2.8	0.929
LAVI (mL)	19.9 ± 6.0	20.9 ± 8.8	0.288
GLS (%)	−18.1 ± 2.7	−19.0 ± 2.8	0.004
GLS > −18%, *n* (%)	47 (51)	33 (36)	0.053
GLS > −16%, *n* (%)	22 (24)	10 (11)	0.031

Logistic regression analysis for the predictors of cardiac dysfunction (GLS > −18%)

	Univariate		Multivariate	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Age per 1 year	1.01 (0.99–1.03)	0.315		
Sex (male)	2.13 (1.14–3.97)	0.017	2.01 (1.07–3.78)	0.03
BMI per 1.0	1.00 (0.94–1.06)	0.998		
Hypertension	1.03 (0.64–1.65)	0.905		
Diabetes	1.05 (0.48–2.26)	0.909		
Moderate-to-severe OSA	3.02 (1.31–7.00)	0.01	2.83 (1.22–6.61)	0.016

CPAP treatment impact on RV by 2D Echo

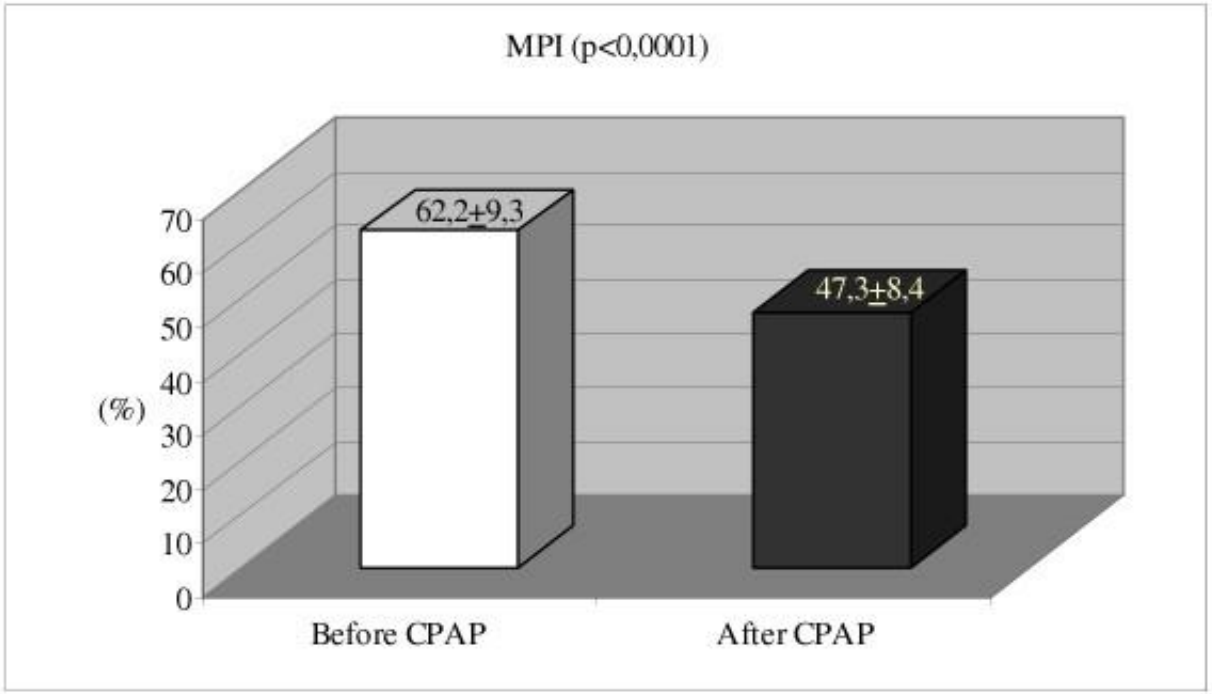
Echocardiographic parameters describing right heart structure and function

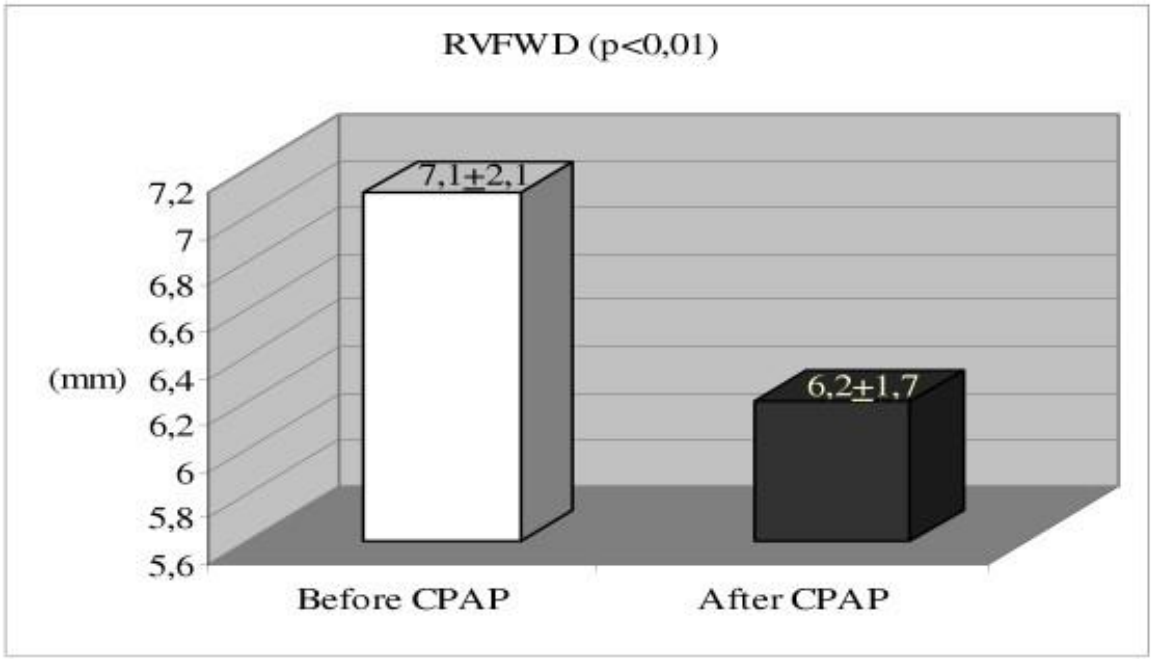
		Patients treated with CPAP $n = 16$			Patients without CPAP $n = 22$		
		Baseline	Follow-up	*p* value	Baseline	Follow-up	*p* value
RVOT	(mm)	31.8 ± 4.3	32.9 ± 2.9	ns	32.0 ± 2.5	30.0 ± 7.2	ns
TAPSE	(mm)	22.1 ± 4.3	25.5 ± 4.6	0.005	23.7 ± 5.3	25.8 ± 4.6	ns
RV S'	(cm/s)	14.1 ± 3.2	17.2 ± 5.2	0.005	15.0 ± 3.9	15.1 ± 2.8	ns
RV E'	(cm/s)	11.4 ± 2.8	11.5 ± 2.5	ns	11.6 ± 3.1	10.8 ± 2.9	ns
RAd1	(mm)	46.2 ± 12.1	50.6 ± 7.9	ns	42.9 ± 7.2	43.4 ± 7.1	ns
RAd2	(mm)	49.4 ± 5.4	50.1 ± 9.2	ns	48.6 ± 6.2	49.4 ± 6.6	ns
RAA	(cm2)	20.7 ± 2.7	22.9 ± 6.7	ns	18.6 ± 4.4	19.0 ± 4.7	ns
RVD1	(mm)	44.1 ± 5.2	45.9 ± 6.4	ns	41.4 ± 5.6	42.2 ± 5.5	ns
RVD2	(mm)	36.6 ± 5.7	37.3 ± 6.8	ns	33.6 ± 6.6	32.6 ± 5.2	ns
RVD3	(mm)	74.9 ± 28.0	75.1 ± 22.6	ns	71.3 ± 22.2	73.4 ± 20.8	ns
TRPG	(mmHg)	22.9 ± 6.2	23.6 ± 6.7	ns	24.2 ± 4.6	25.3 ± 6.8	ns

Subgroup analysis of RV systolic parameters according to age and weight

		Baseline	TAPSE Follow-up	*p* value	Baseline	RV S' Follow-up	*p* value
Age (years)	< 60	23.2 ± 3.7	27.6 ± 4.6	0.01	15.1 ± 3.1	19.8 ± 5.1	0.01
	≥ 60	20.6 ± 4.8	22.8 ± 3.1	ns	12.7 ± 2.9	14.9 ± 3.3	ns
BMI (kg/m^2)	< 35	20.7 ± 3.8	23.8 ± 4.8	0.04	13.1 ± 2.5	15.1 ± 4.4	0.04
	≥ 35	24.3 ± 4.6	27.0 ± 4.2	0.02	15.3 ± 3.7	20.1 ± 5.0	0.01

CPAP effect on RV and global function





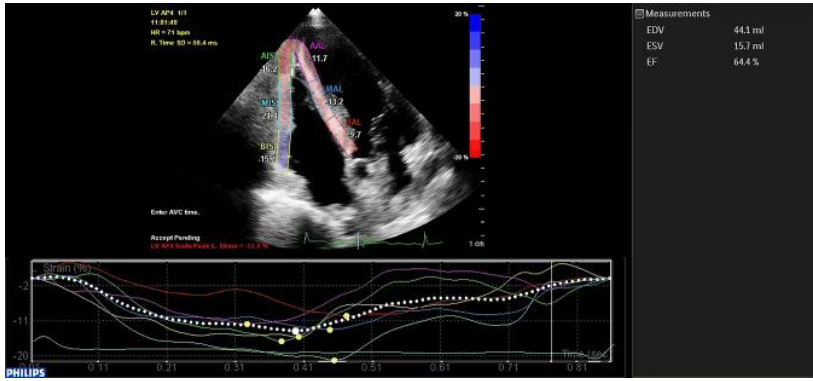
CPAP on RV performance among High Altitude

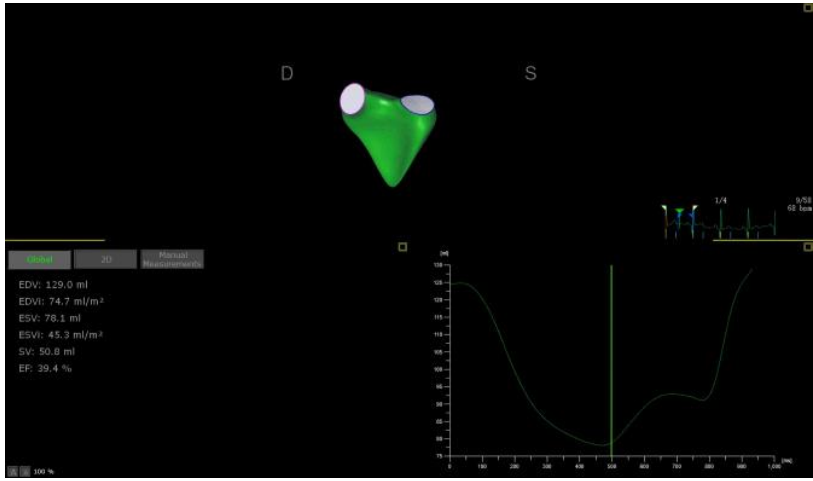
RV echocardiographic variables in control and OSAS groups.

	Control group (n = 31)	OSAS group (n = 71)	*P* value
RAVI (ml/m^2)	29.3 ± 4.7	37.7 ± 4.4	< 0.001
LVEF (%)	63.5.3 ± 4.9	63.0 ± 4.6	0.67
RV apicobasal diameter (mm)	35.1 ± 4.0	36.3 ± 4.2	0.21
RV mediolateral diameter (mm)	30.0 ± 3.9	30.5 ± 3.3	0.55
RV long-axis diameter (mm)	67.2 ± 5.0	68.2 ± 4.4	0.38
RVWT (mm)	4.0 ± 0.7	4.4 ± 0.6	0.01
TAPSE (mm)	20.7 ± 2.3	20.0 ± 2.2	0.18
Outlet RV VTI (cm)	13.2 ± 2.5	13.5 ± 2.5	0.49
TV E/A	1.2 ± 0.3	1.2 ± 0.2	0.89
TV S_a (cm/s)	13.2 ± 2.5	13.6 ± 2.4	0.49
TV E/e'	3.9 ± 0.7	4.3 ± 1.0	0.12
Systolic PAP (mmHg)	29.6 ± 4.7	42.7 ± 8.4	< 0.001
High sPAP	9 (30%)	34 (48%)	0.001
PVR (Woods)	1.7 ± 0.3	1.9 ± 0.3	< 0.001
RV GLS (%)	23.1 ± 3.8	18.8 ± 5.9	0.002
RV LLS (%)	25.7 ± 2.9	22.8 ± 3.4	< 0.001
RV SLS (%)	18.6 ± 5.2	18.8 ± 4.4	0.86
RVEDV (ml)	82.2 ± 11.1	99.7 ± 13.3	< 0.001
RVESV (ml)	41.2 ± 6.7	58.5 ± 11.1	< 0.001
RVEDVI (ml/m^2)	45.9 ± 6.7	45.9 ± 6.7	< 0.001
RVESVI (ml/m^2)	23.0 ± 4.0	31.8 ± 6.2	< 0.001
RVEF (%)	49.4 ± 3.4	41.5 ± 4.8	< 0.001

Changes in RV parameters, cardiac biomarkers and polysomnographic data before and after CPAP therapy.

	Before CPAP therapy (n = 45)	After CPAP therapy (n = 45)	*P* value
RAVI (ml/m^2)	37.2 ± 4.9	32.9 ± 3.9	< 0.001
RV apicobasal diameter (mm)	34.4 ± 3.4	35.3 ± 4.3	0.37
RV mediolateral diameter (mm)	30.8 ± 3.4	29.7 ± 2.9	0.14
RV long-axis diameter (mm)	67.9 ± 4.6	66.8 ± 3.9	0.28
RVWT (mm)	4.3 ± 0.5	4.2 ± 0.5	0.29
TAPSE (mm)	19.9 ± 2.1	19.8 ± 2.4	0.95
RV VTI (cm)	13.5 ± 2.4	13.9 ± 2.0	0.46
TV E/A	1.3 ± 0.2	1.2 ± 0.2	0.95
TV S_a (cm/s)	15.1 ± 2.7	14.5 ± 2.0	0.41
TV E/e'	5.4 ± 1.5	4.9 ± 1.0	0.54
Systolic PAP (mmHg)	44.6 ± 8.0	37.9 ± 6.9	0.001
High sPAP	22 (49%)	14 (31%)	0.001
PVR (Woods)	2.0 ± 0.4	1.7 ± 0.3	< 0.001
RV GLS (– %)	20.3 ± 3.3	23.1 ± 3.1	0.001
RV LLS (– %)	20.7 ± 3.5	23.0 ± 3.0	0.006
RV SLS (– %)	18.3 ± 4.2	18.8 ± 4.1	0.63
RVEDV (ml)	101.7 ± 13.2	92.6 ± 11.9	0.005
RVESV (ml)	57.3 ± 10.4	47.0 ± 9.4	< 0.001
RVEDVI (ml/m^2)	55.5 ± 7.0	50.6 ± 7.0	0.007
RVESVI (ml/m^2)	31.2 ± 5.6	25.7 ± 5.4	< 0.001
RVEF (%)	43.9 ± 5.2	49.5 ± 4.9	< 0.001
AHI (events/hour)	49.7 ± 16.4	4.9 ± 1.7	< 0.001
Awake S_aO_2 (%)	93.0 ± 1.9	93.6 ± 1.8	0.27
Minimal S_aO_2 (%)	72.5 ± 8.4	84.3 ± 5.8	< 0.001
Time S_aO_2 < 90% (min)	24.3 ± 12.0	3.4 ± 2.0	< 0.001

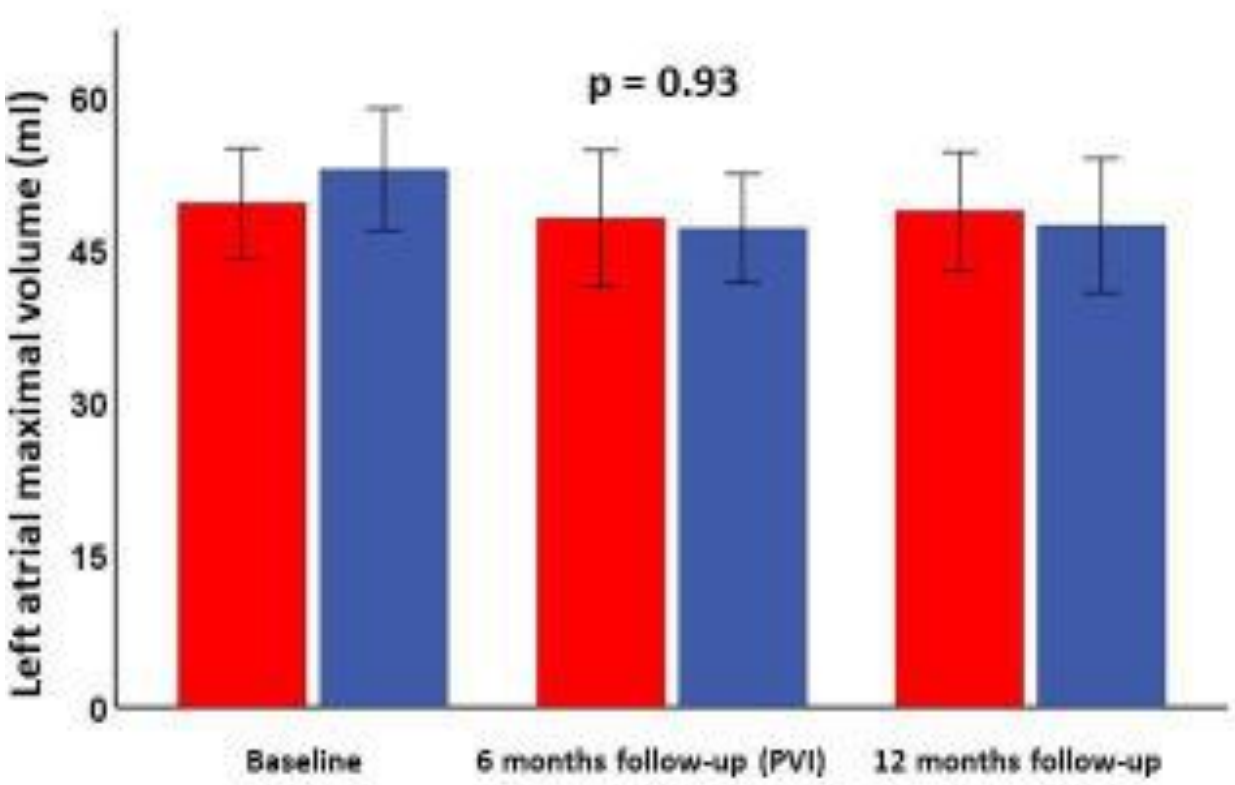


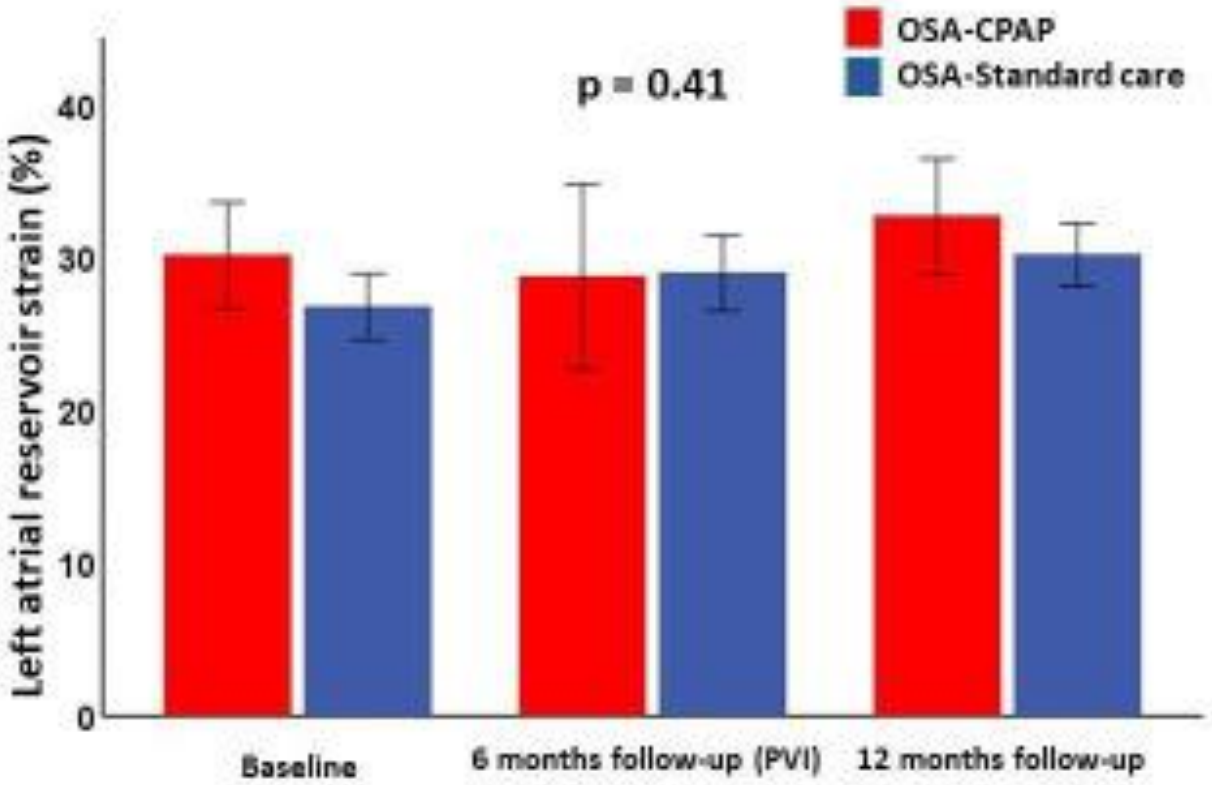


OSA, Weight flux, CPAP and Chamber size

	↓ weight (n.20)		=/↑ weight (n.21)		ANOVA	
Metabolic parameters	**Basal**	**CPAP**	**Basal**	**CPAP**	**CPAP**	**Weight change**
LV parameters						
LV diastolic diameter, mm	50 ± 5	50 ± 6	49 ± 5	49 ± 4	ns	ns
Septal thickness, mm	11.6 ± 1.2	10.5 ± 0.9[b]	11.5 ± 1.4	11.6 ± 1.5	ns	0.006
Posterior wall thickness, mm	11.4 ± 1.3	10.4 ± 1.1[b]	11.1 ± 1.2	11 ± 1.3	ns	0.008
Relative wall thickness	0.46 ± 0.06	0.42 ± 0.05[b]	0.46 ± 0.08	0.46 ± 0.07	ns	0.003
LV mass index, g/m$^{2.7}$	64.7 ± 13.2[a]	55.3 ± 11.4[b]	54.9 ± 12.7	56.1 ± 12.4	ns	0.008
LV Ejection fraction, %	63 ± 4	64 ± 5	64 ± 4	65 ± 4	ns	ns
E/A	0.91 ± 0.24	1.07 ± 0.29[b]	0.95 ± 0.21	0.97 ± 0.28	ns	0.018

CPAP among OSA and Afib by TTE

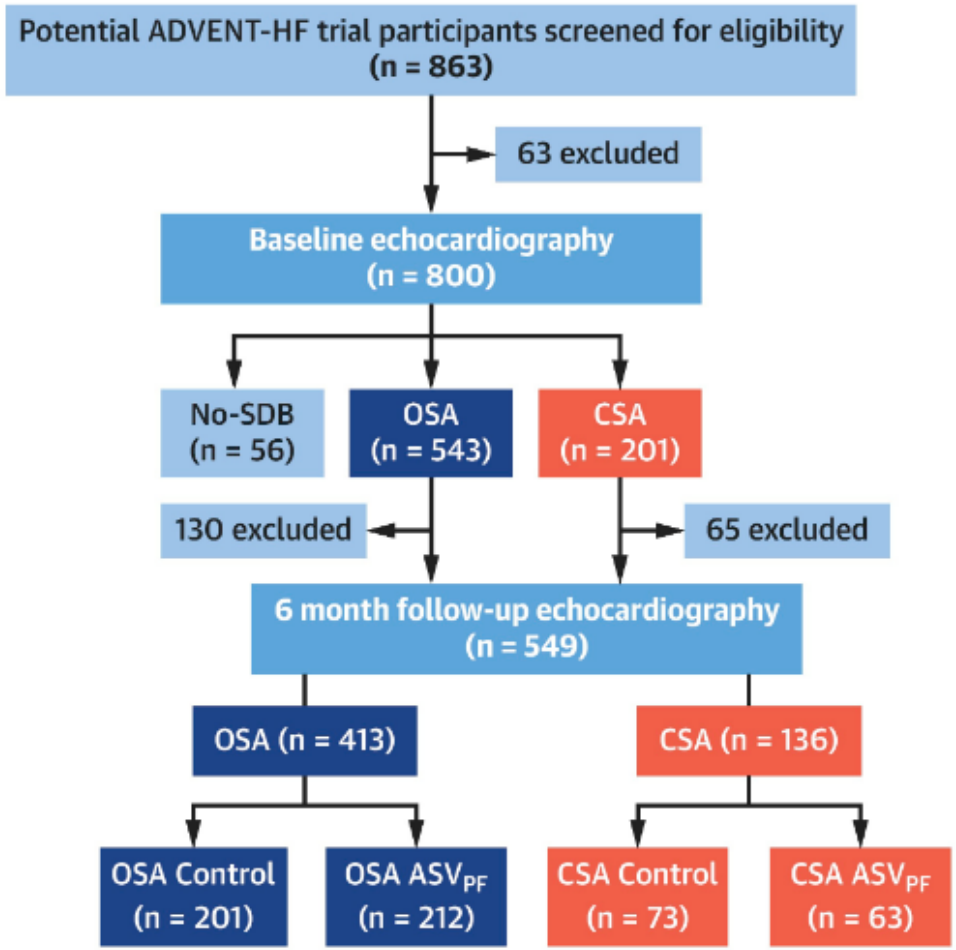




CENTRAL ILLUSTRATION: Echocardiographic Characteristics in ADVENT-HF at Baseline and Follow-Up

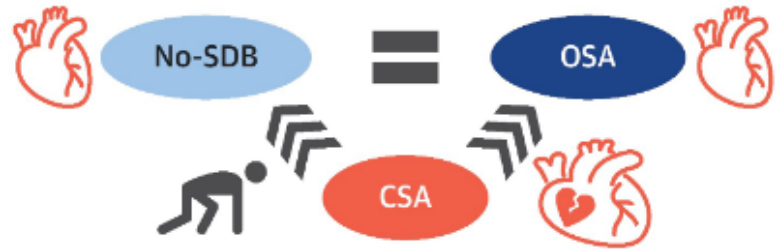
Echocardiographic Component of ADVENT-HF Trial

Left ventricular structure and function before and after treatment with ASV_{PF}



At Baseline

Subjects with OSA had similar LV structure and function as those without SDB.



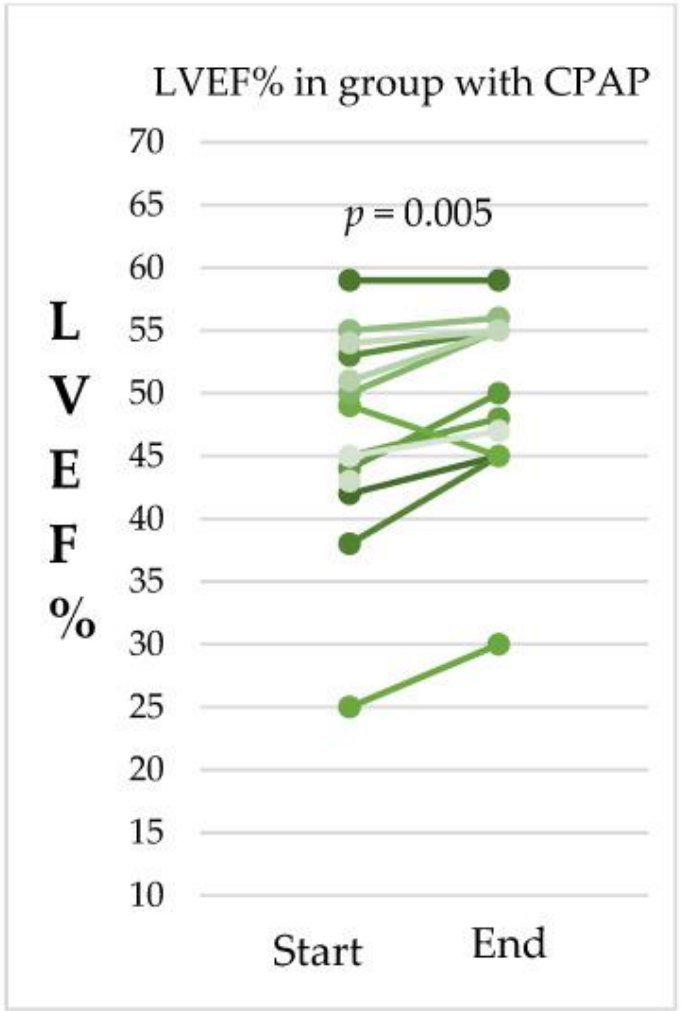
Those with CSA were older and had more severe sleep apnea.
Those with CSA had more LVH and lower LVEF than the other groups.
LV volumes were larger in those with CSA than those with OSA.

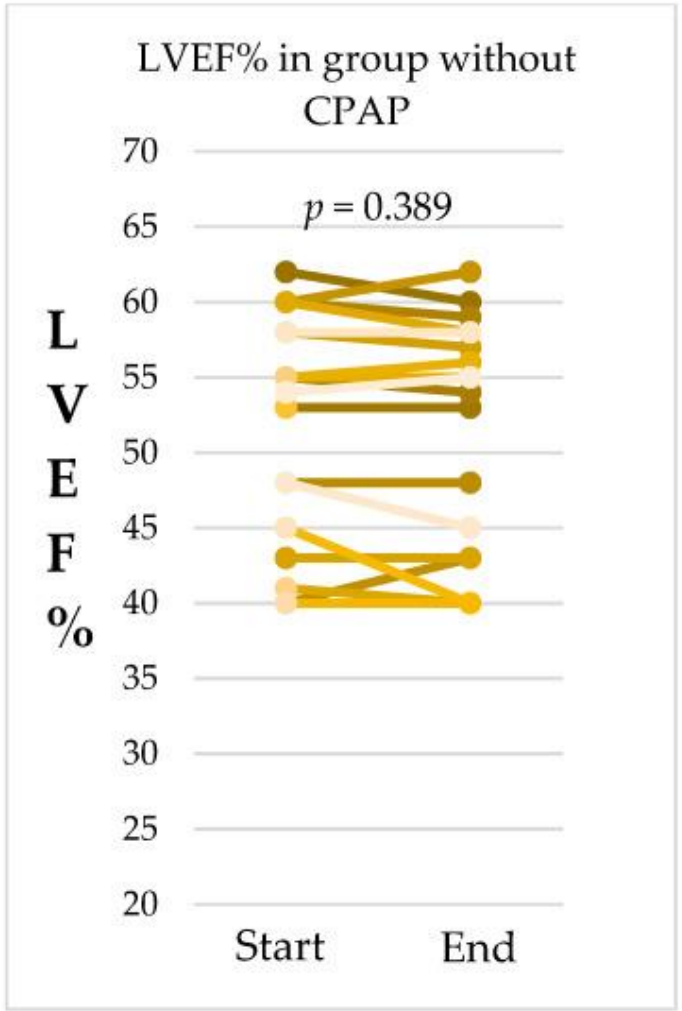
6 Months After the ASV_{PF} Treatment

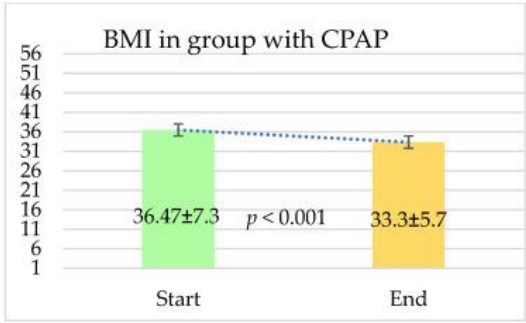
SDB suppression by ASV_{PF} had no significant effect on LV structure or function

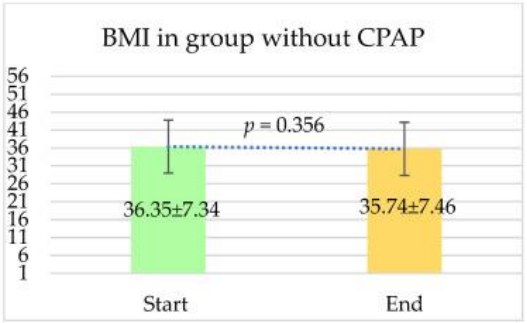
Yatsu S, et al. JACC Heart Fail. 2025;10.1016/j.jchf.2025.02.016

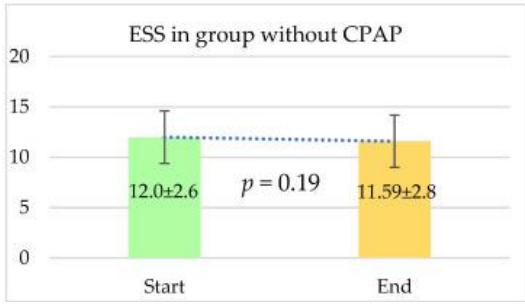
CPAP effect on Acute Decompensated HF

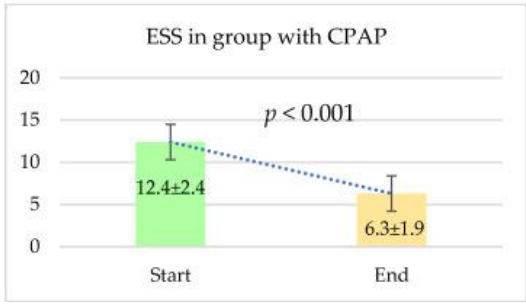


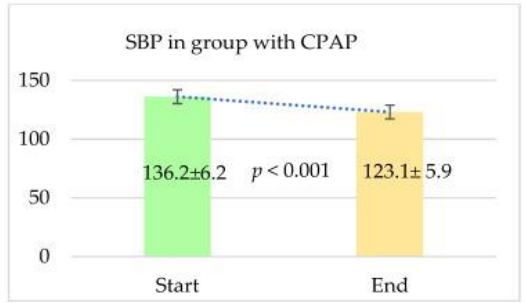


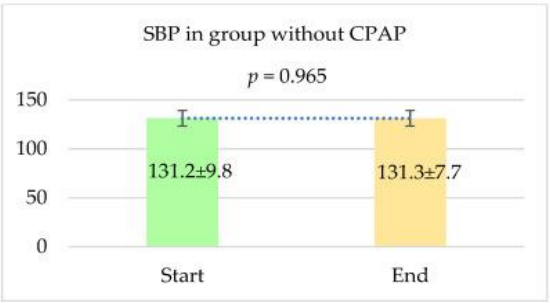




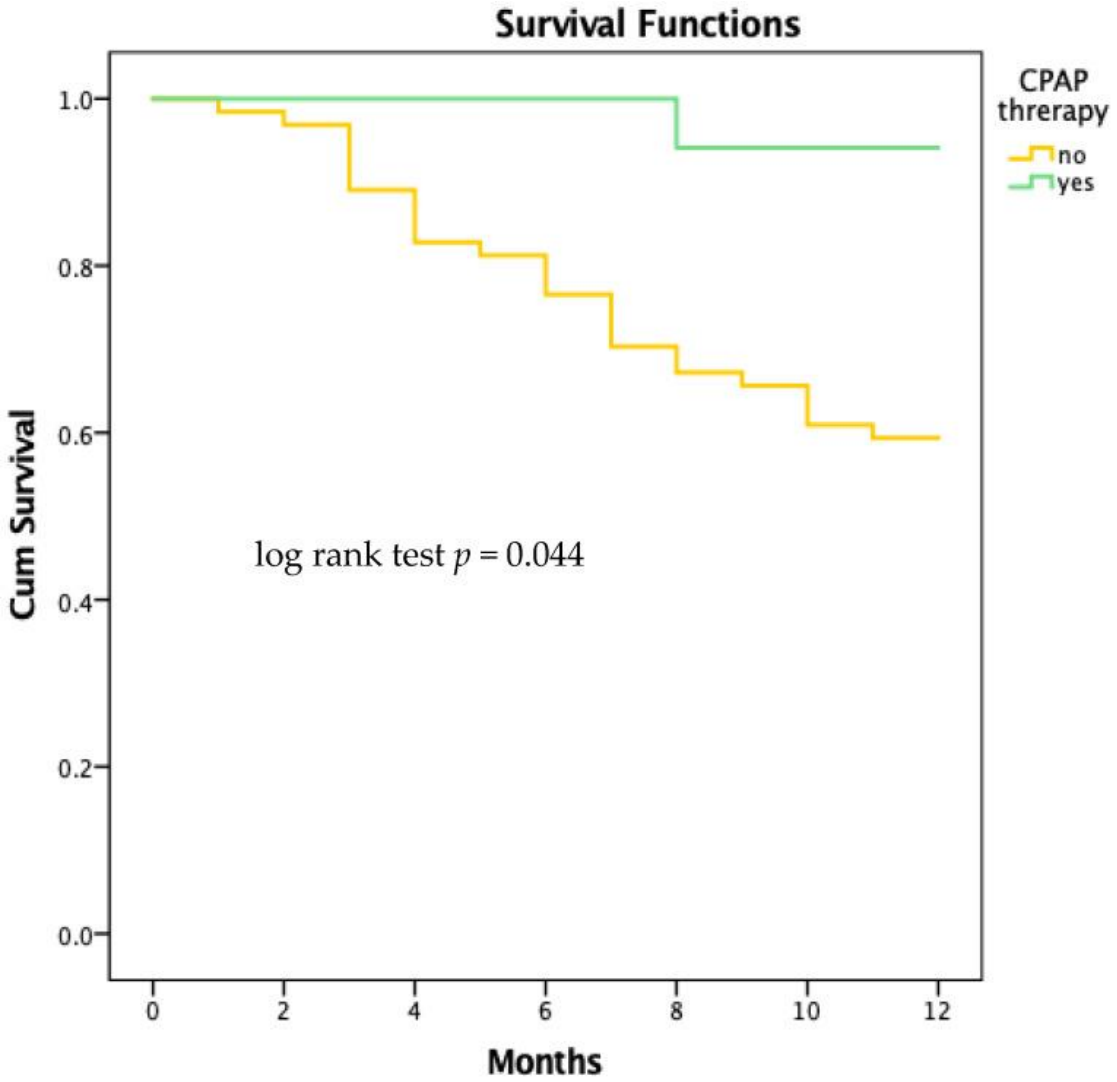


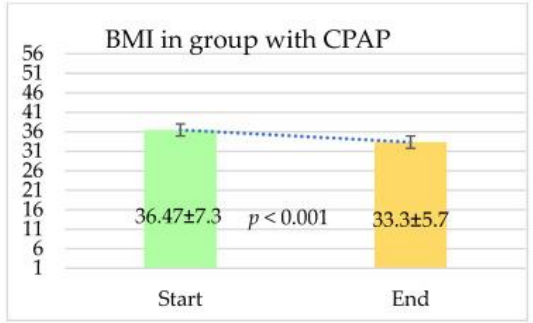


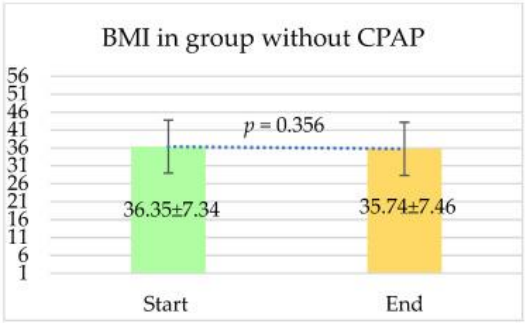


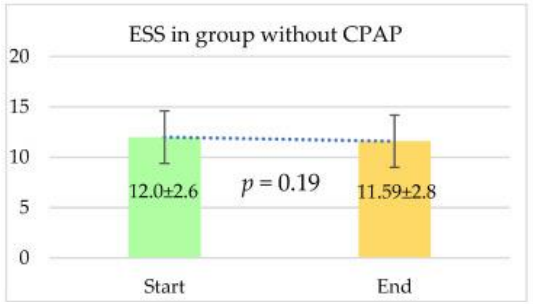


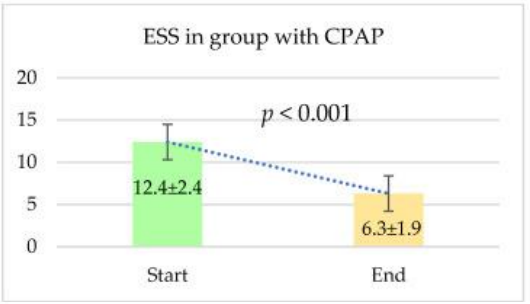
CPAP effect on Acute Decompensated HF

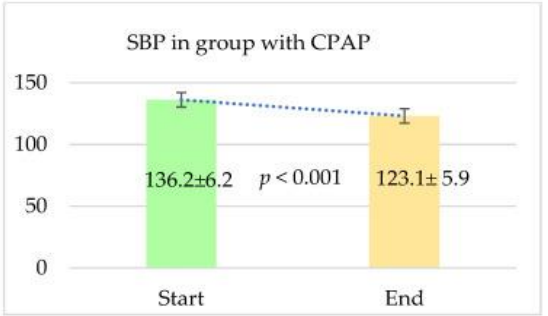


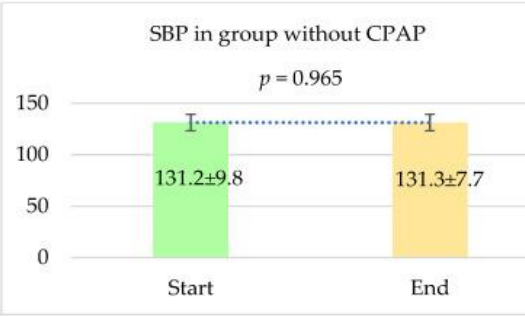




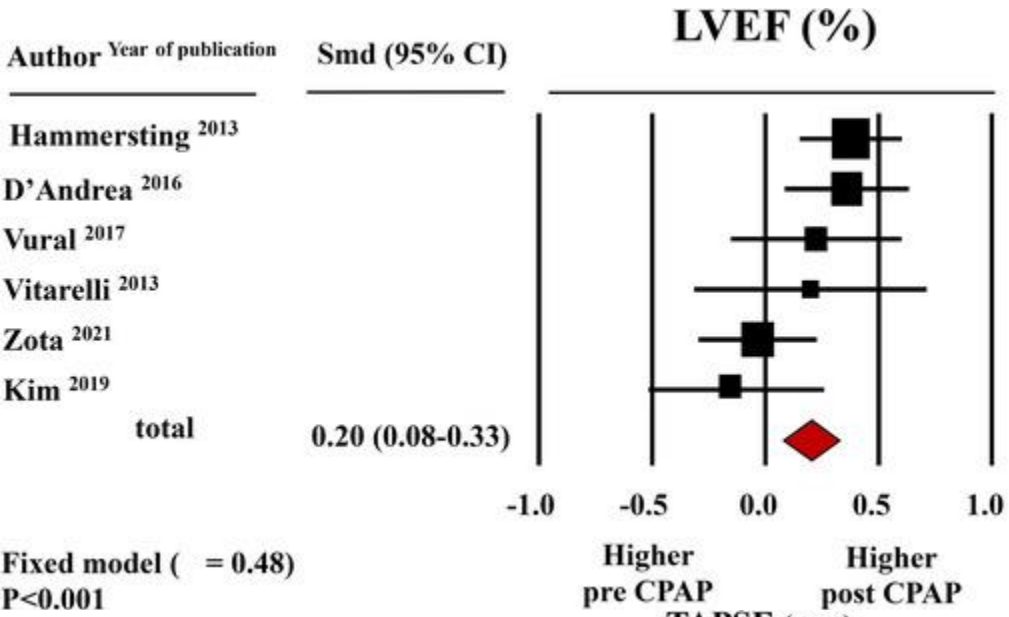


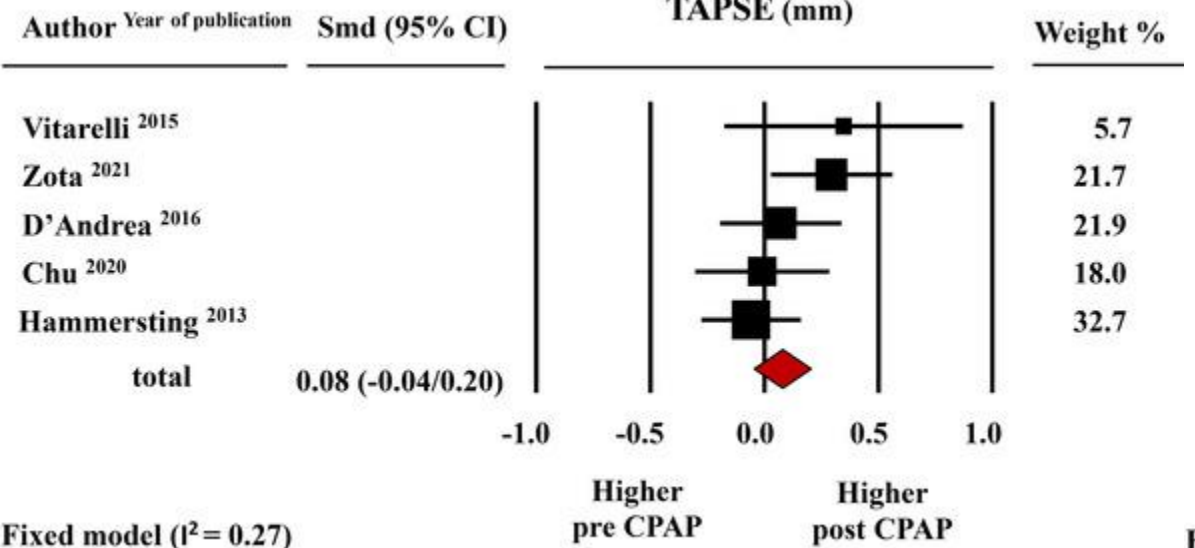


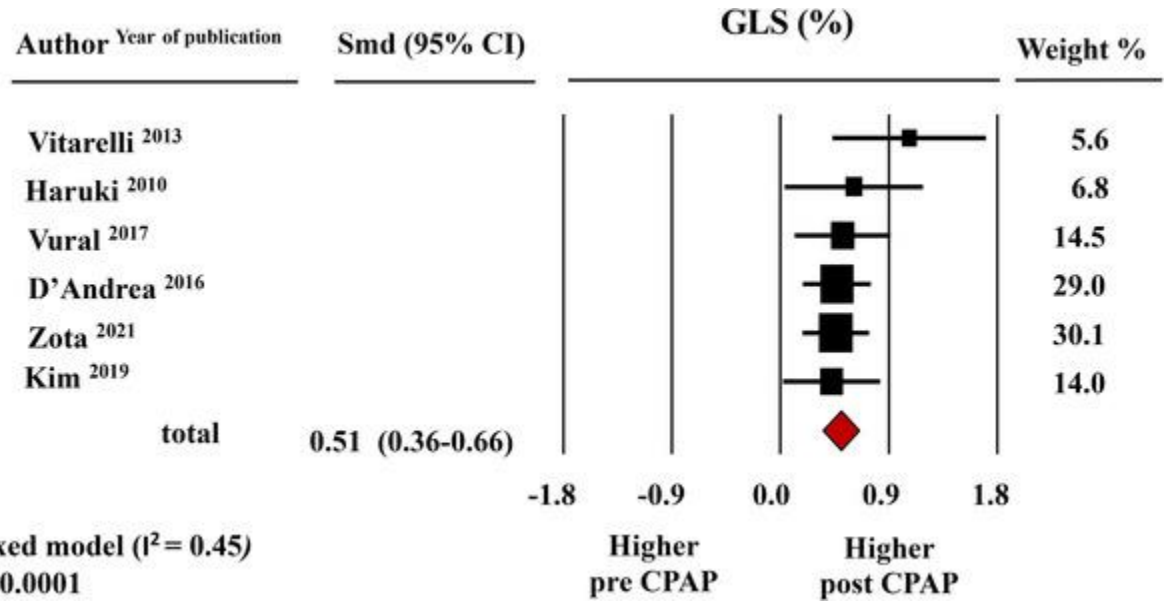


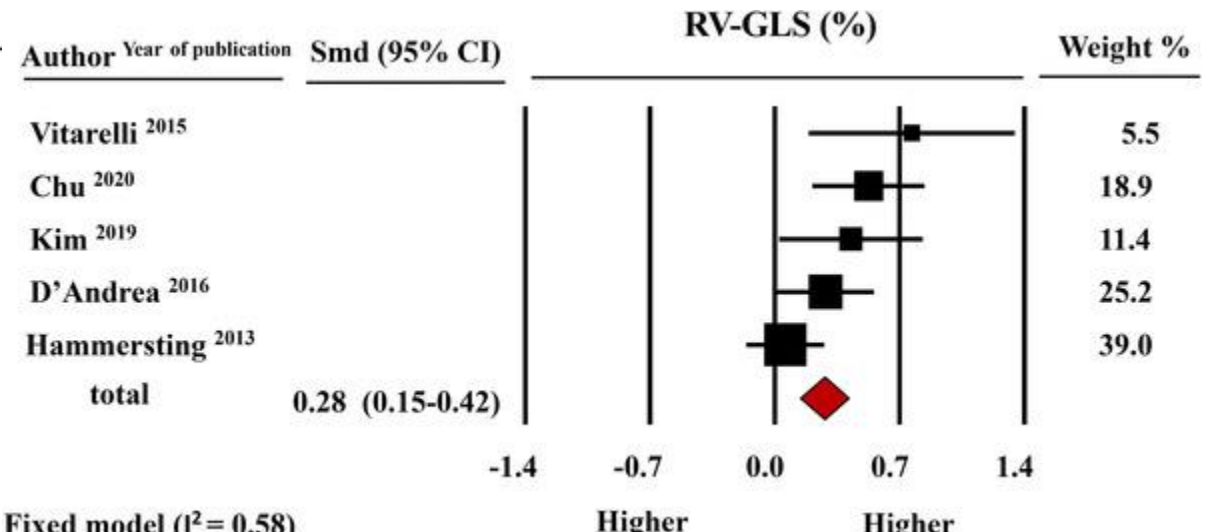


CPAP on LV/RV Function: Meta-Analysis





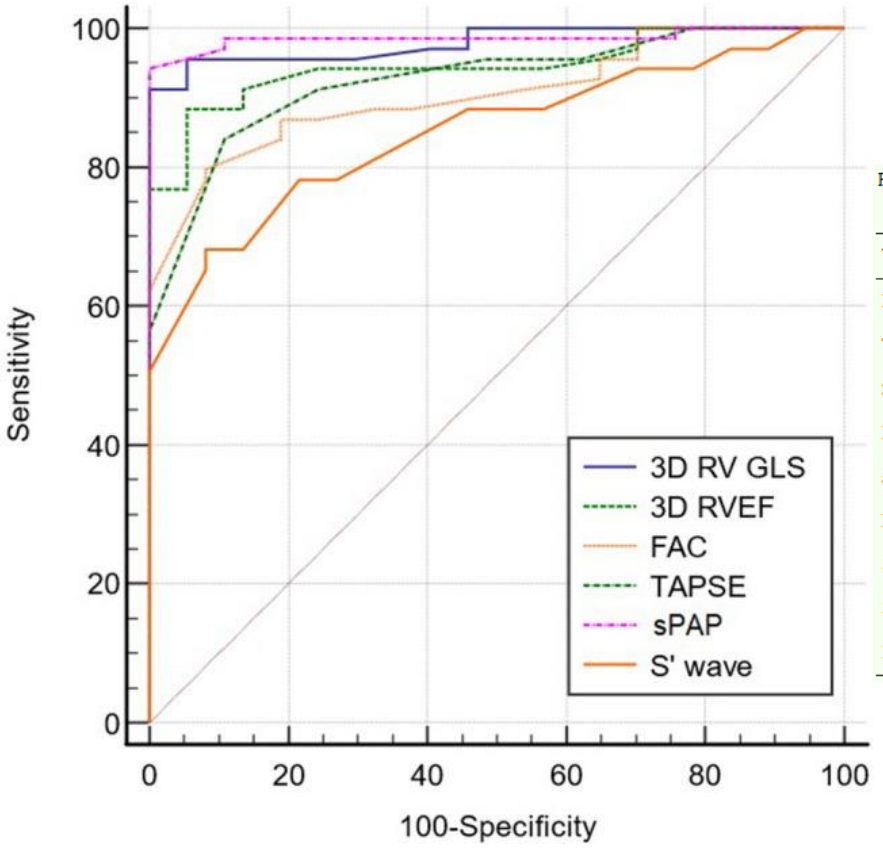




Predicting OSA Severity by Echo

Baseline characteristics of randomized patients.

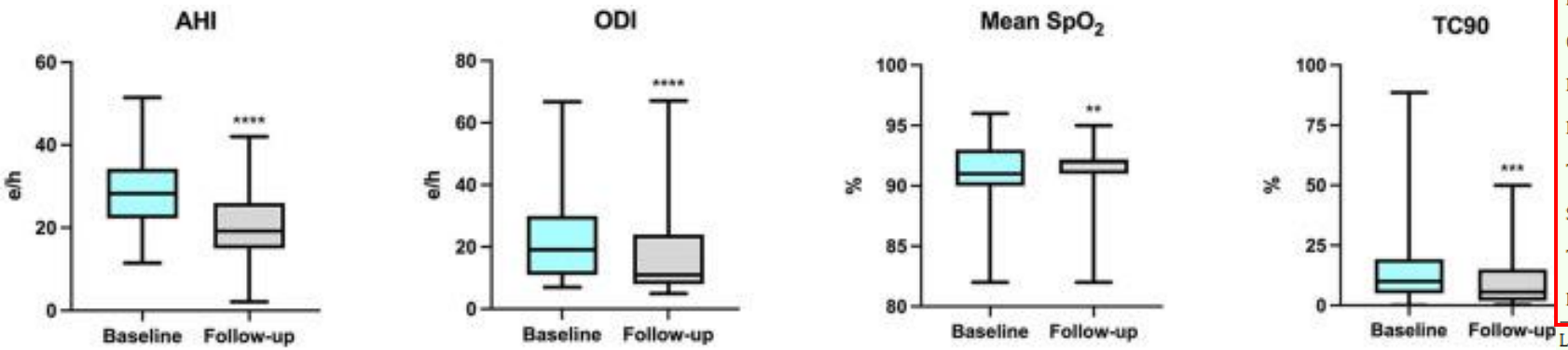
Variable	Patients (n = 69)	Controls (n = 37)	*P* value
Age years	59 (51; 67)	55 (47; 62)	0.394
Female sex, n, %	31 (44.9%)	18 (48.6%)	0.871
BMI kg -m^2	34 (30.8; 40.2)	29 (28.5; 30.1)	< 0.001
Additional cardiovascular risk factors			
DM type 2, n , %	11 (15.9%)	5 (13.5%)	0.96
Arterial hypertension, n, %	63 (91.3%)	22 (59.5%)	< 0.001
Smoking , n , %	20 (29%)	17 (45.9%)	0.125
Results of the sleep study			
AHI h^{-1}	28.6 (18.3;42.1)	2.8 (1.2;3.3)	< 0.001
ODI h^{-1}	29.2 (21.9; 42.9)	3.5 (2; 6.2)	< 0.001
Echocardiographic parameters			
RV, mm	40 (37;43)	36 (33;37)	< 0.001
TAPSE, mm	19 (17;20)	23 (21.5;24)	< 0.001
S' wave, cm/s	9.6 (9;11)	12 (11.9; 13.5)	< 0.001
RA-RV Gradient, mmHg	35 (30; 40)	19 (16.5; 22.5)	< 0.001
sPAP, mmHg	42 (37; 47)	22 (16.5; 24.5)	< 0.001
RV FAC, %	28 (25; 30)	35 (33.4;45)	< 0.001
3D RVEF, %	31.9 (27.8;39)	50 (45.4; 59)	< 0.001
3D RV GLS, %	– 13.5 (– 17.2; – 10.6)	– 22.3 (– 28.3;– 20.9)	< 0.001
LVEF, %	55 (50;55)	55 (55;63)	0.09
Serum parameters			
NT-pro BNP, pg/ml	290 (201;620)	35 (15;127.5)	< 0.001

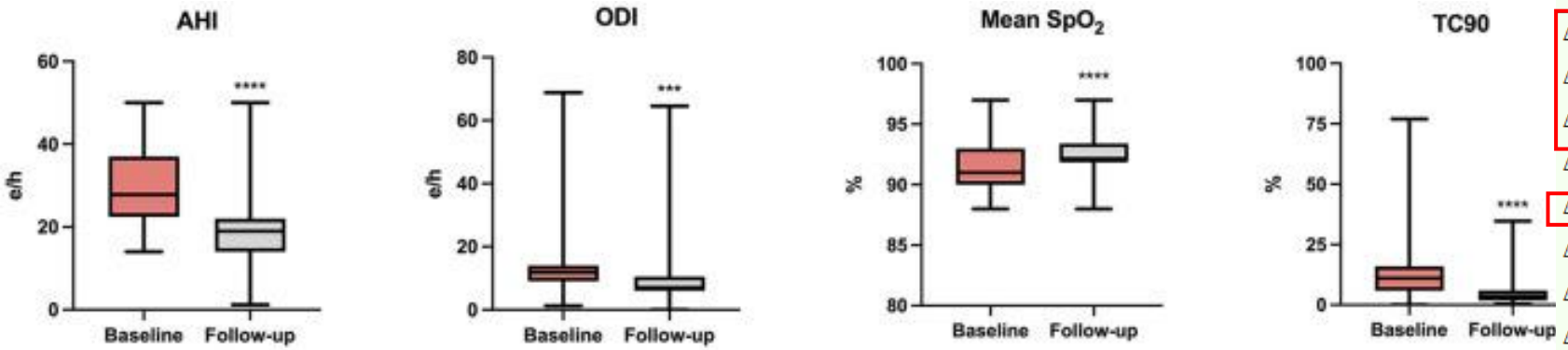


ROC cutoff values predicting OSA.

Variable	Cutoff	Sensitivity (%)	Specificity (%)	AUC (95% CI)
RV, mm	> 39	59.4	100	0.835 (0.75–0.9)
TAPSE, mm	≤ 20	84	89	0.92 (0.85–0.96)
S' wave, cm/s	≤ 10.2	68.1	91.8	0.85 (0.76–0.91)
RA-RV Gradient, mmHg	> 29	82.6	94.5	0.94 (0.87–0.97)
sPAP, mmHg	> 30	94.2	100	0.98 (0.94–0.99)
RV FAC, %	≤ 31	79.7	91.8	0.9 (0.83–0.95)
3D RVEF, %	≤ 41	88.4	94.5	0.94(0.88–0.98)
3D RV GLS, %	> – 19.2	91.3	100	0.979 (0.93–0.99)
NT-proBNP, pg/ml	> 136	91.3	91.8	0.96 (0.9–0.99)

Entresto and Sleep Apnea





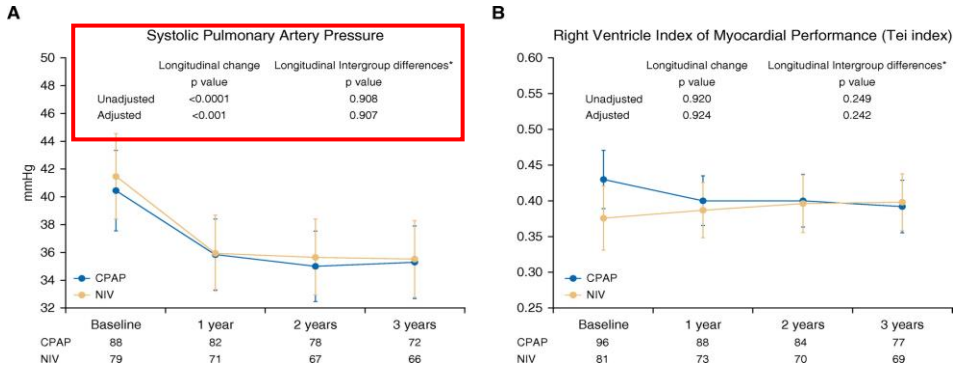
Echocardiographic parameters at baseline and after 6 months of therapy with sac/val.

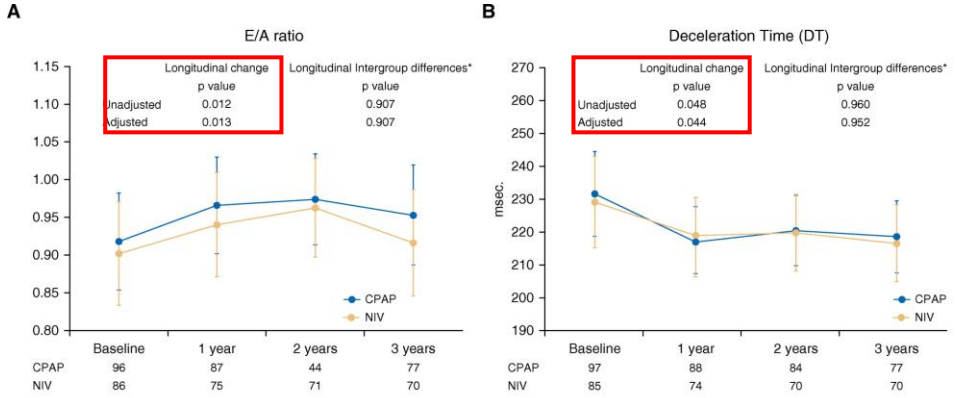
Variables	Baseline (n = 132)	Follow-up (n = 132)	Standardized mean difference (Hedges'g)	p
LAVI, mL/m^2	49.8 ± 13.7	46.1 ± 12.0	0.27	0.001
LVEDV/BSA, mL/m^2	89.6 ± 9.8	87.8 ± 8.4	0.20	<0.0001
LVESV/BSA, mL/m^2	61.0 ± 7.1	57.3 ± 5.9	0.57	<0.0001
LVEF, %	31.9 ± 1.4	34.7 ± 1.6	1.86	<0.0001
CI, mL/min/m^2	1,675.6 ± 199.9	1,856.6 ± 212.9	0.87	<0.0001
E/e' ratio	17.4 ± 3.5	15.9 ± 2.8	0.47	<0.0001
GLS, %	−7.9 ± 1.7	−9.0 ± 1.4	0.71	<0.0001
RVOT, cm	2.6 ± 0.4	2.1 ± 0.4	1.25	<0.0001
RAA, cm^2	20.5 ± 2.8	19.3 ± 2.3	0.4	<0.0001
TAPSE, mm	16.3 ± 1.1	17.1 ± 1.7	0.56	<0.0001
S-PAP, mmHg	44.5 ± 6.6	41.5 ± 6.6	0.45	<0.0001
TAPSE/S-PAP, mm/mmHg	0.37 ± 0.06	0.42 ± 0.08	0.70	<0.0001
IVC, mm	20.2 ± 1.3	19.1 ± 3.3	0.44	<0.0001

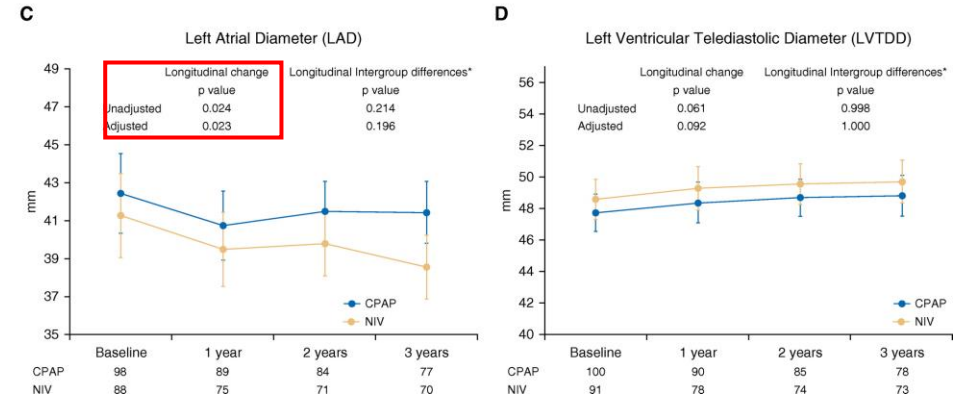
Linear regression analysis focused on AHI variation (Δ AHI) as dependent variable.

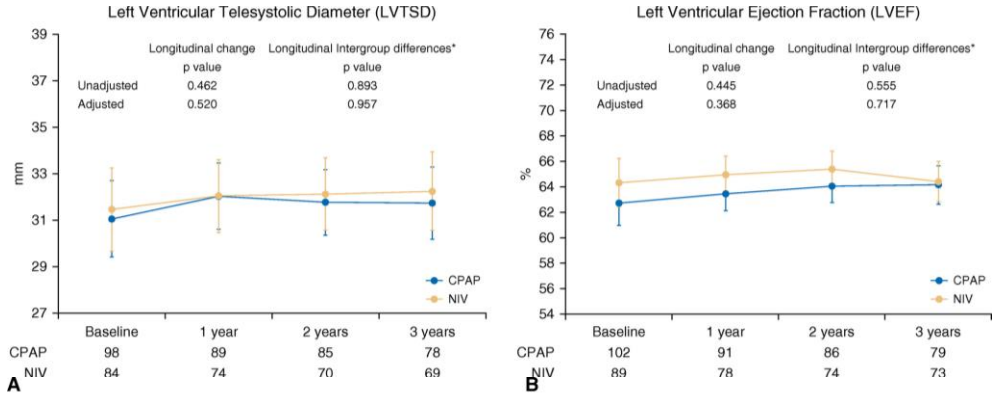
Variables	R	p
Δ CI, mL/min/m^2	0.486	<0.0001
Δ TAPSE, mm	0.325	<0.0001
Δ NT-proBNP, pg/mL	−0.404	<0.0001
Δ IVC, mm	−0.048	0.293
Δ eGFR, mL/min/1.73m^2	0.405	<0.0001
Δ RAA, cm^2	−0.086	0.164
Δ MLHFQ, score	−0.066	0.228
Δ RVOT, cm	−0.104	0.218
Δ TAPSE/S-PAP, mm/mmHg	0.036	0.340
Δ E/e' ratio	−0.008	0.463
Δ LVEDV/BSA, mL/m^2	−0.022	0.401
Δ S-PAP, mmHg	−0.055	0.264

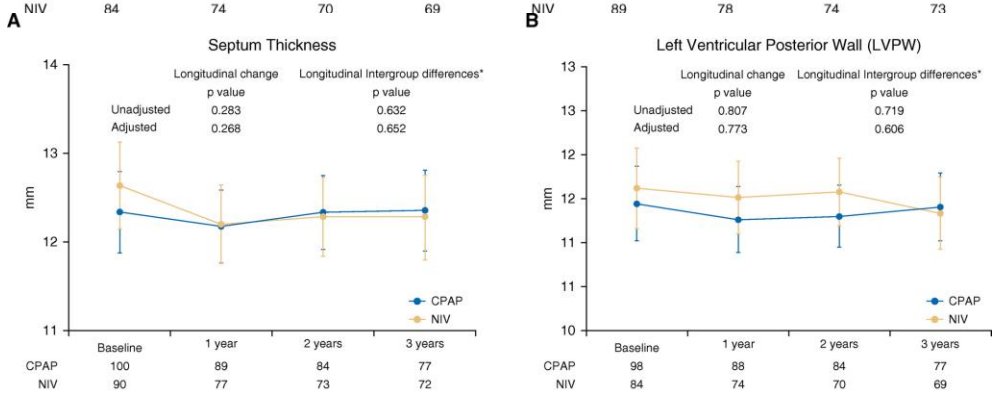
OHS induced PH and NIV on Echo

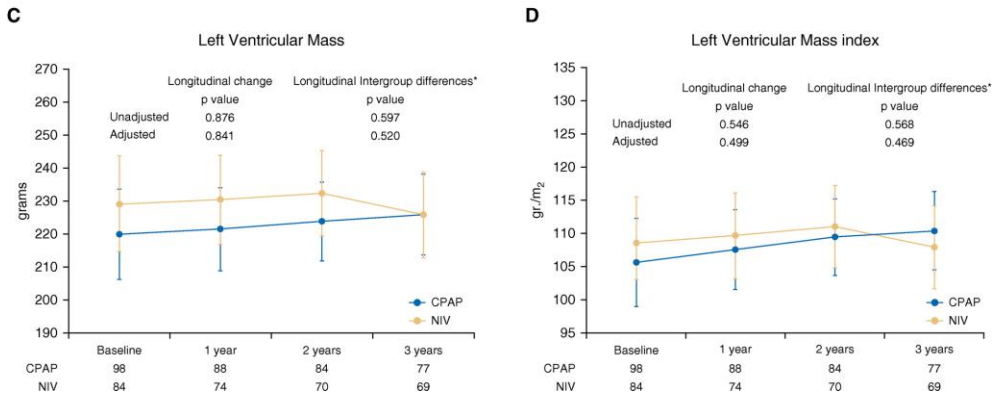




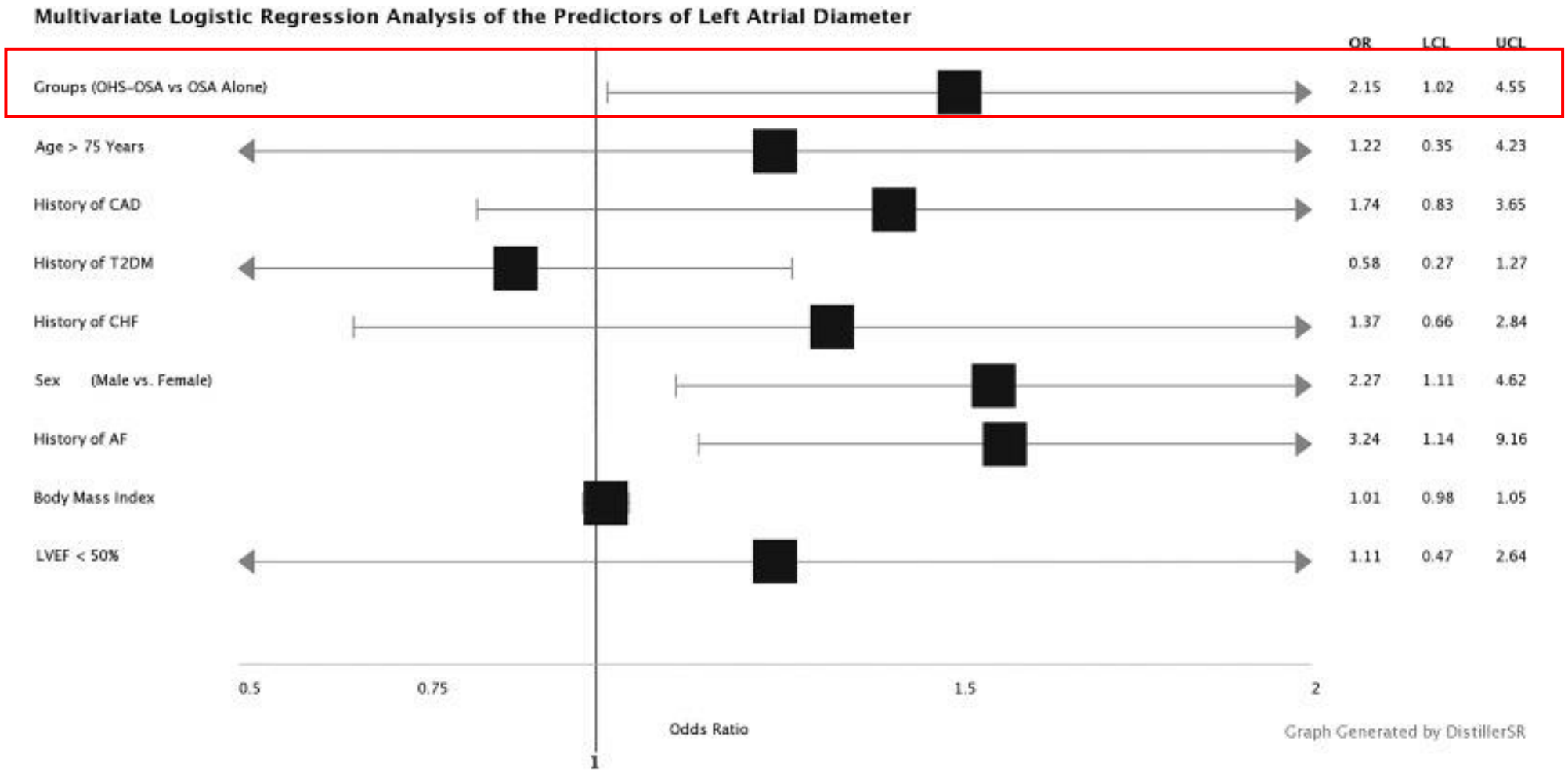








OHS Overlap with OSA on LA Diameter



Summary of Imaging of OSA:

	CMR	Echo	CCT
OSA increases	LVMI*, ECV* , LVEDV**, LVESV, RVEDV**, RVESV**, aortic diameter, and sphericity, LAVI^, Atrial LGE^, %LGE^ and RVEF^	RVD, RVWT, RV performance index, RV free wall diameter, LVMI, diastolic A wave*, EAT thickness	EAT attenuation*, CAC score*, CAC density*, chances of CAC>400, CAC progression
OSA decreases	FMD**, RVEF	RV s', TAPSE, RV FAC, RV GLS, LV GLS*, E/A ratio*, RV diastolic function	-
OSA no effect	Aortic PW and distensibility	LVEF*, LV wall thickness, LV cavity size, atria size	-
CPAP improves	RVEDD, RVEDV, RVEF, RVOT outflow acceleration time^, PASP, LVMI, atrial GLS, LAVI, RAVI	LVEF, LV GLS, RV GLS, isovolumetric acceleration, TAPSE (2/3 studies), RV s', E/e' (by 3D echo), LAEF, LA active emptying volume, tricuspid gradient, LVMI^, Aortic root diameter and distensibility^	Total, low density, and noncalcified plaque volumes
CPAP no effect	FMD, LAVI^, LVEF, arterial stiffness	TAPSE, E/e' (by 2D echo), FAC, RVD, RV %strain, diastolic dysfunction^, LAVI, E/A^, LVEF, e'	-

*=linearly with AHI/severity; **=additional interactive effect with other risk factors, ^=in select subsets

Utility of Advanced Imaging for Sleep Physician

- There aren't clearly established guidelines regarding the utilization of cardiac imaging for OSA alone
  - There are for screening for OSA among other disease states or concomitant disorders such as hypertension
- My personal practice:
  - Any patient with an AHI >15 or moderate-severe OSA would get an echo with strain measurements with repeat echo no less than 3 months but likely yearly to assure no other pathologies
  - AHI>15 and asymptomatic should get a Calcium score
  - If cardiorespiratory fitness subjectively is not improving or other symptoms, consider echo or stress testing or coronary CTA as per chest pain guidelines
  - If ventricular arrhythmia or concerning echo findings, consider CMR to confirm or determine additional pathologies

Questions Not Answered

- How do GLP1 agonist studies account for statins in assessment of effect?
- Does Entresto have a role in treatment of OSA beyond HF?
- How much does coronary microvascular dysfunction mediate the effects of OSA? Is this further mediated by EAT?
- How do oral devices and INSPIRE affect echocardiography parameters?
- To what degree did CPAP adherence affect many of these cardiac studies?
- How does CPAP affect ECV on CMR?

What Do We Make of This Study?

Key Question

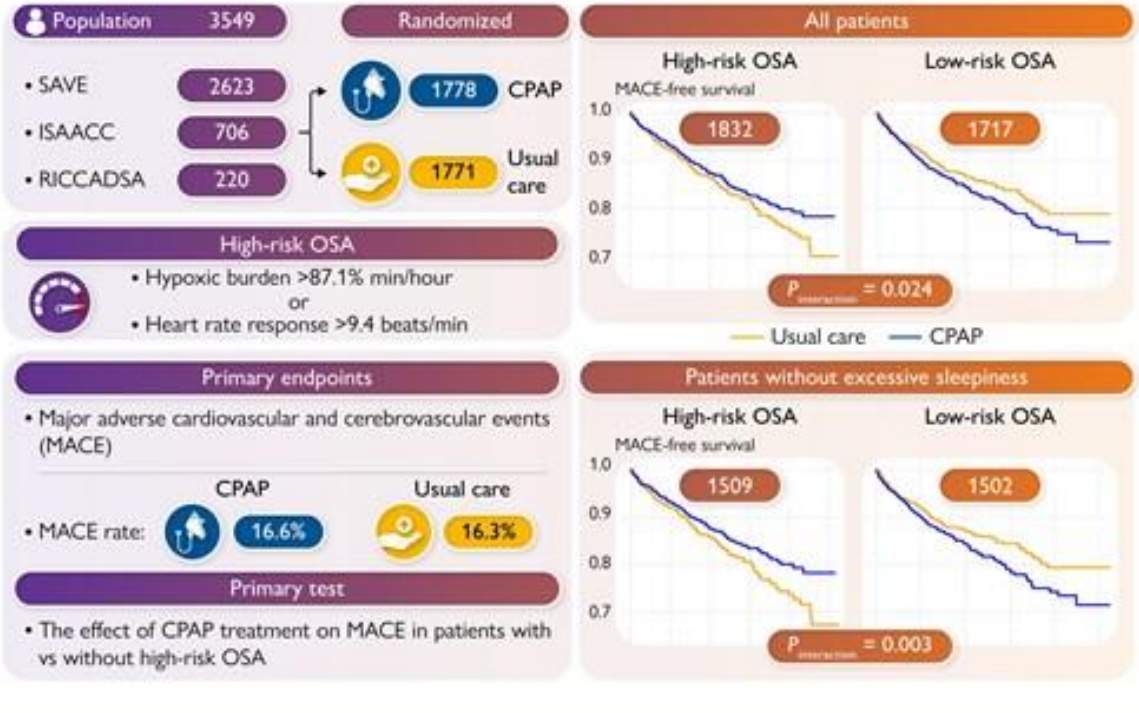
Randomized trials of continuous positive airway pressure (CPAP) have failed to show benefit for secondary cardiovascular outcome prevention. Does CPAP confer cardiovascular benefit preferentially in patients with high-risk obstructive sleep apnoea (OSA)?

Key Finding

In this pooled analysis of three trials, the benefit of CPAP in reducing major adverse cardiovascular and cerebrovascular events was greater in patients with high-risk OSA compared to those with low-risk OSA. In low-risk OSA, CPAP may increase cardiovascular risk.

Take Home Message

Assessment of OSA phenotypes may guide the indication for CPAP to reduce cardiovascular risk.



Additional Key Features of the meta-analysis

- Post-hoc analysis different from the initial design of evaluation for testing CPAP in high and low risk OSA
- Potential for selection bias and cherry picking these studies
- Utilization of delta HR and hypoxic burden as opposed to AHI or other more well established markers in the cardiology literature
  - Granted, I'm not the most familiar so perhaps this is better in some ways I don't understand
- The nature of each of the cardiovascular treatments in each of these studies is also variable as treatment for CAD has rapidly evolved over the past 20 years

Key features

- SAVE trial average 3.3 hours per night CPAP and based CPAP on Epworth sleepiness scale scores of 11 as a cutoff without mention of AHI or severity of OSA or pressure settings; presumed 100% adherence to be included in the study
- ISAACC trial mean adherence time on CPAP was 2.78 hours and patients were nonsleepy; no mention of adherence rate
- RICCADSA did have 60% adherence rate but average time not mentioned; however, their cox regression models for CPAP usage >3h/night and >5h/night did not achieve significance while their >4h/night did so it may have been underpowered to achieve these ends overall

SLEEP ■

What Are the Effects of CPAP Therapy Termination on All-Cause Mortality in OSA From the Nationwide Claims Data Lake for Sleep Apnea Study (ALASKA)?

CHEST

STUDY DESIGN

Analysis of **French national health insurance reimbursement** system database for **all CPAP users**

Propensity match comparing those who stopped CPAP in 1st year to those who continued to use CPAP

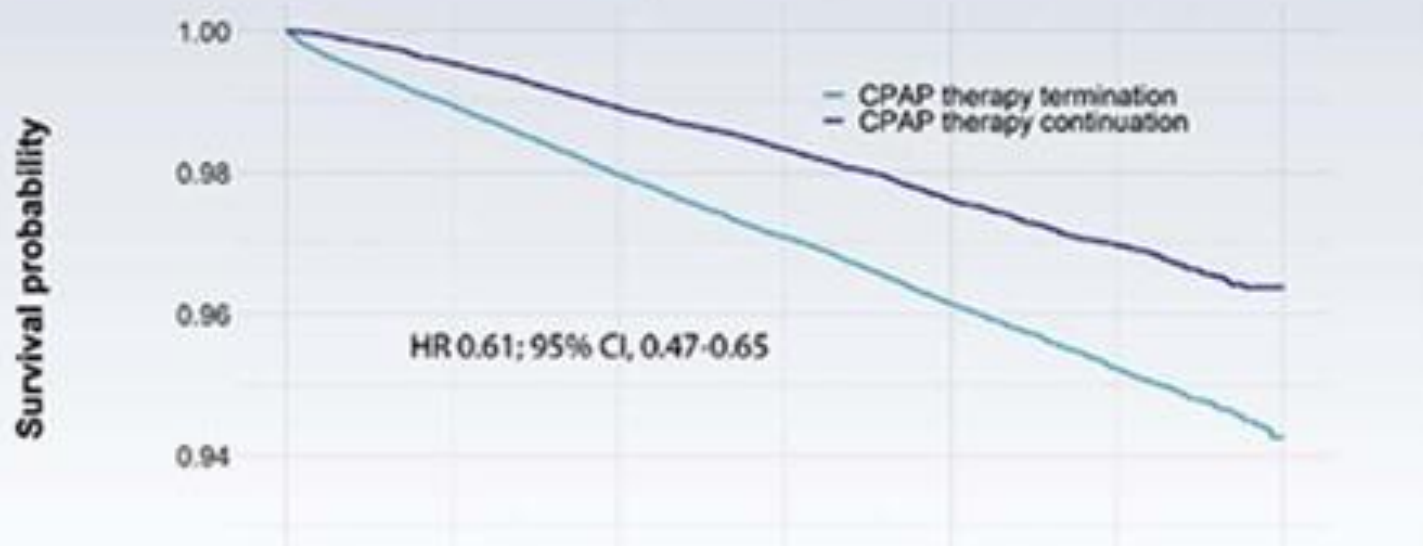
CPAP Continuation (n = 88,007)

VS

CPAP Termination (n = 88,007)

RESULTS

CPAP continuation associated with a **significantly lower risk of all-cause death**



Heart failure also **less common** in those continuing with CPAP (HR 0.77; 95% CI, 0.71-0.81)

Continuation of CPAP therapy has the potential to reduce all-cause mortality in patients with OSA.

Pépin J, et al. *CHEST*. June 2022 | @Journal_CHEST | https://doi.org/10.1016/j.chest.2022.02.013

Question for MOC

- A 60 year old male with PMH of diabetes, hypertension, BMI of 28 and hyperlipidemia all of which are currently diet controlled presents for evaluation of OSA. The patient's family attests to daytime somnolence, witnessed apneic episodes and some memory problems for which the patient has insight into. Patient otherwise has been able to continue exercising including daily 30 minute moderate intensity runs. Home study demonstrates an AHI of 4.5. A recent echocardiogram demonstrates normal LVEF, moderate diastolic dysfunction and decreased right ventricular systolic function with a PASP of 45mmHg. Physical exam demonstrates no edema, JVD or rales. What is the next best management choice?
  - A. Cardiac MRI to evaluate for presumed heart failure and Holter monitor for arrhythmia
  - B. Coronary CTA to evaluate for obstructive coronary disease and initiating GLP1 agonist for presumed OSA
  - C. Repeat in-lab PSG and Coronary artery calcium score
  - D. Refer to Cardiology

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Questions