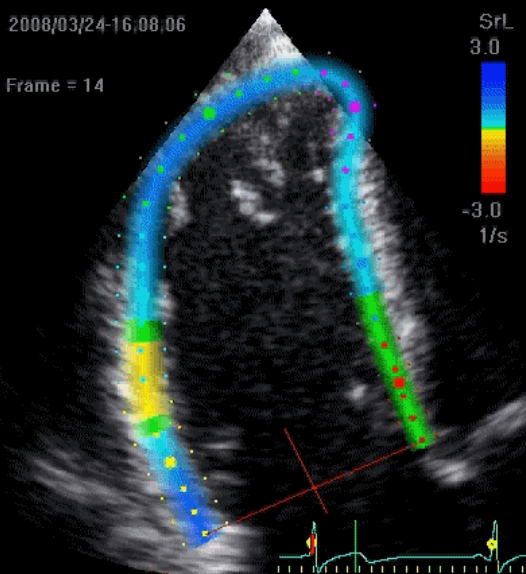




2008/03/24-16.08.06

Frame = 14



# RV and pulmonary circulation during exercise

*EAE Teaching Course*

*Sofia, Bulgaria 2012*



THE UNIVERSITY OF  
MELBOURNE

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**University Hospitals Leuven, Belgium**

**St Vincent's Hospital, University of Melbourne, Australia**

**[andre.lagerche@uz.kuleuven.be](mailto:andre.lagerche@uz.kuleuven.be)**

Disclosures: none



# RV function is most relevant when the RV has to work

## 1. Pathological load

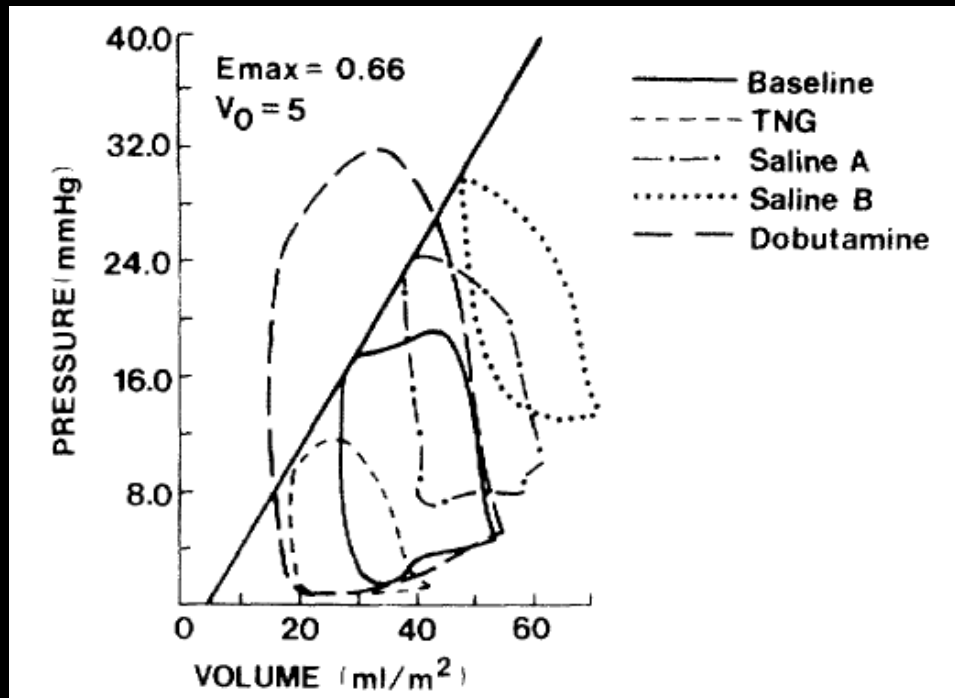
- Increased afterload due to LV dysfunction
- Pulmonary vascular disorders
- Chronically increased preload

## 2. Activity, exercise, exertion

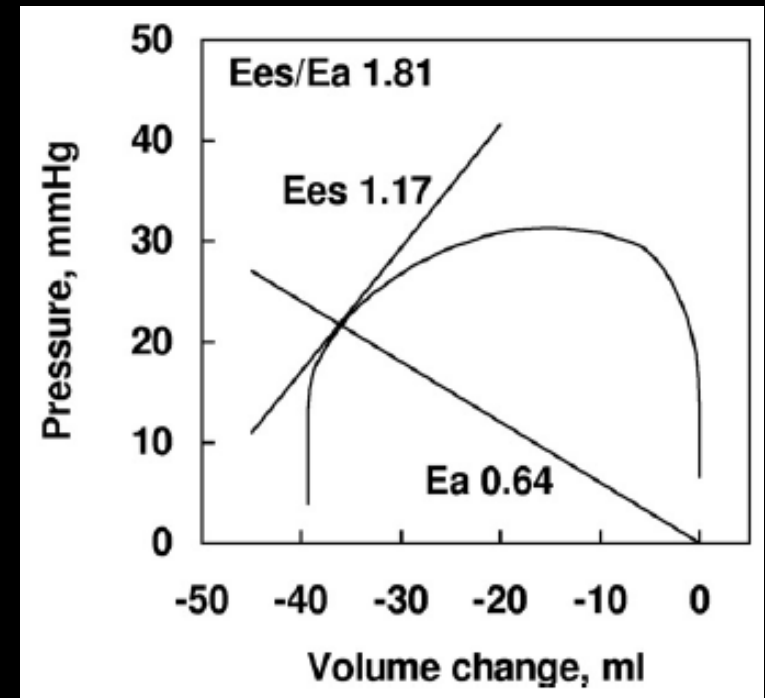
- Increased RV wall stress
- Increased RV work
- Increased O<sub>2</sub> demand

# The healthy RV at rest

- it doesn't need to do anything



Brown and Ditchey *Circulation* 1988



Pagnamenta, Naeije et al.  
*J Appl Physiol* 2010

# The LV does not do all of the work

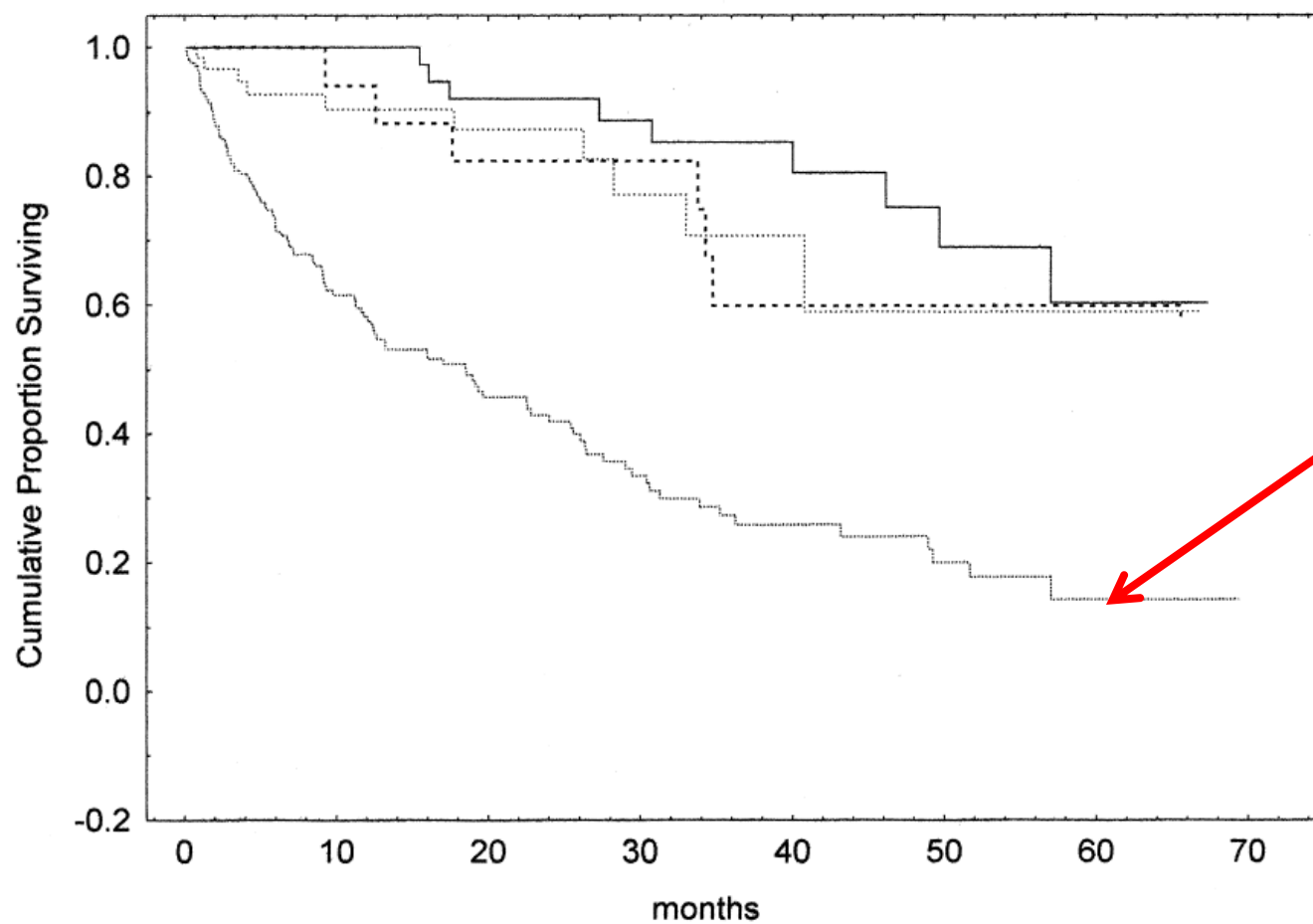
- Potentially misleading conclusions:
  - LV contributes substantially to RV pressure generation (Seki 1975, Yamaguchi 1991, Feneley 1995)

BUT

- When RV afterload is increased then, in the absence of a functional RV, CO rapidly falls (J Hoffman Thorac Cardiovasc Surg 1994)

# Independent and Additive Prognostic Value of Right Ventricular Systolic Function and Pulmonary Artery Pressure in Patients With Chronic Heart Failure

Stefano Ghio, MD, FESC,\* Antonello Gavazzi, MD, FESC,\* Carlo Campana, MD,\*  
Corinna Inserra, MD,\* Catherine Klersy, MD,† Roberta Sebastiani, MD,\* Eloisa Arbustini, MD,‡  
Franco Recusani, MD,\* Luigi Tavazzi, MD, FESC, FACC\*

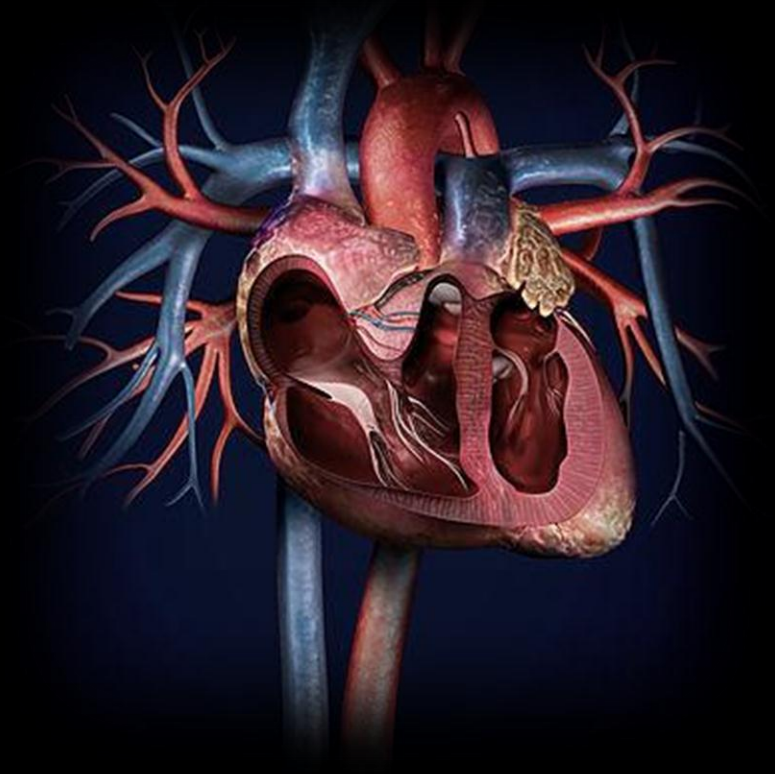


JACC 2001

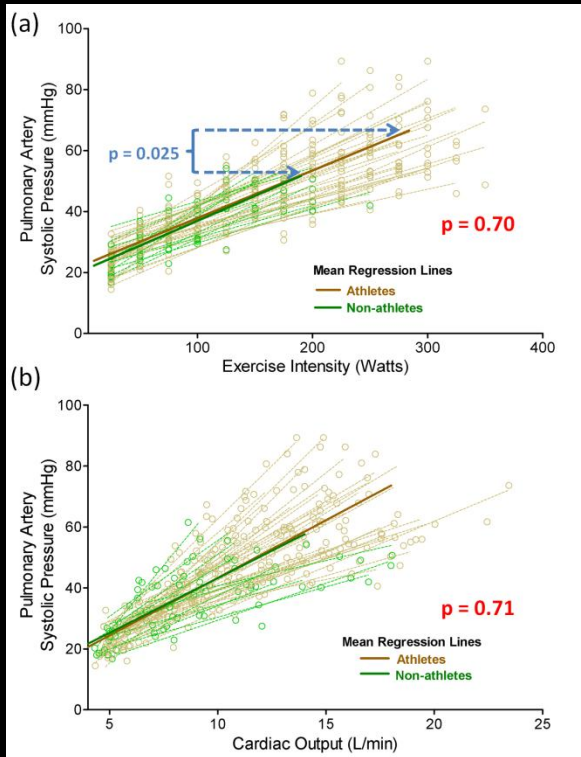
↑PASP  
↓RVEF



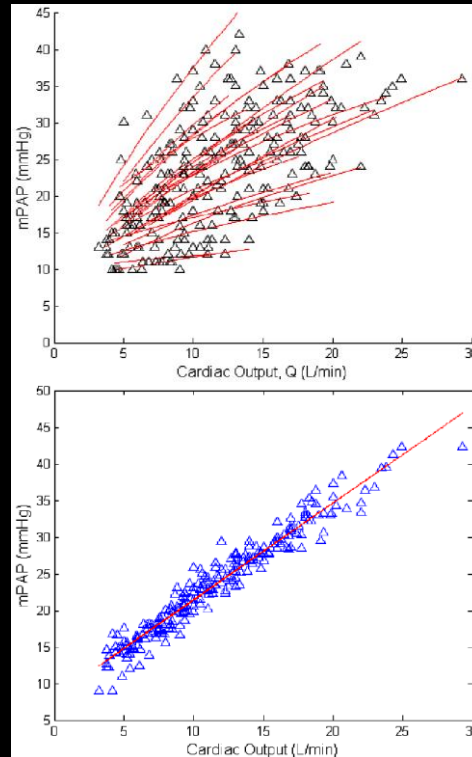
*Why do we have  
a right ventricle?*



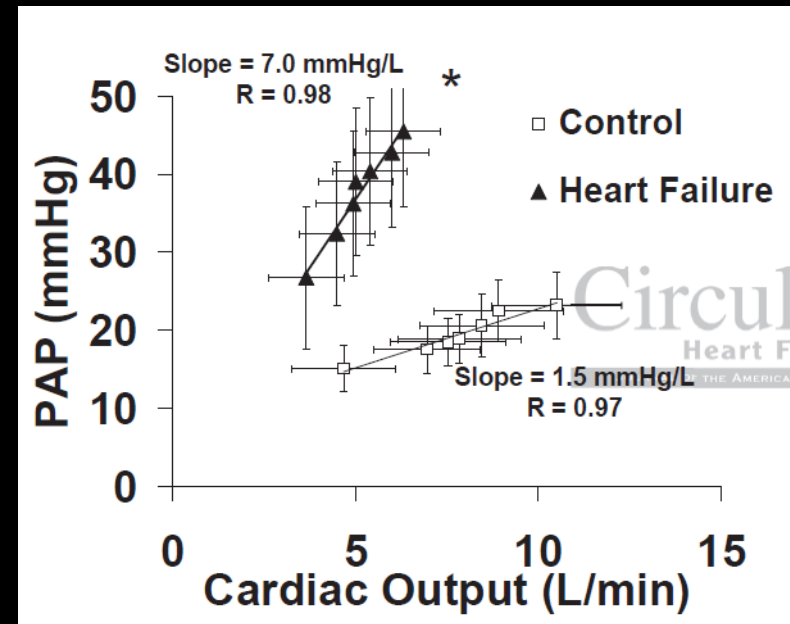
# Exercise = RV work



La Gerche, Prior et al.  
*J App Physiol* 2010



Argiento, Naeije et al.  
*Eur Resp J* 2010



Lewis, Semigran et al.  
*Circ Heart Failure* 2011



# Investigating ventricular hemodynamics during exercise

- ***Echocardiography***
  - Volumes
  - Cardiac Output
  - Pulmonary artery pressures
- ***Cardiac MRI***
- ***Radial arterial catheter***

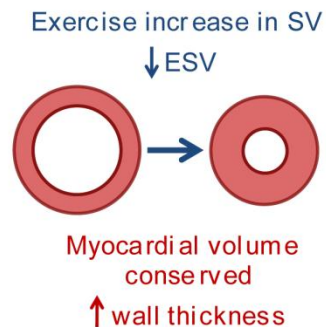
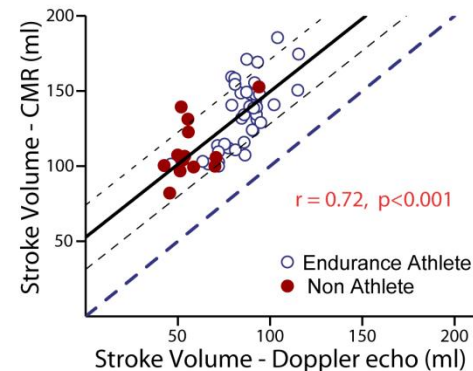
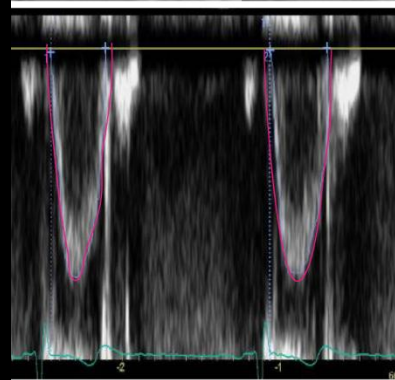
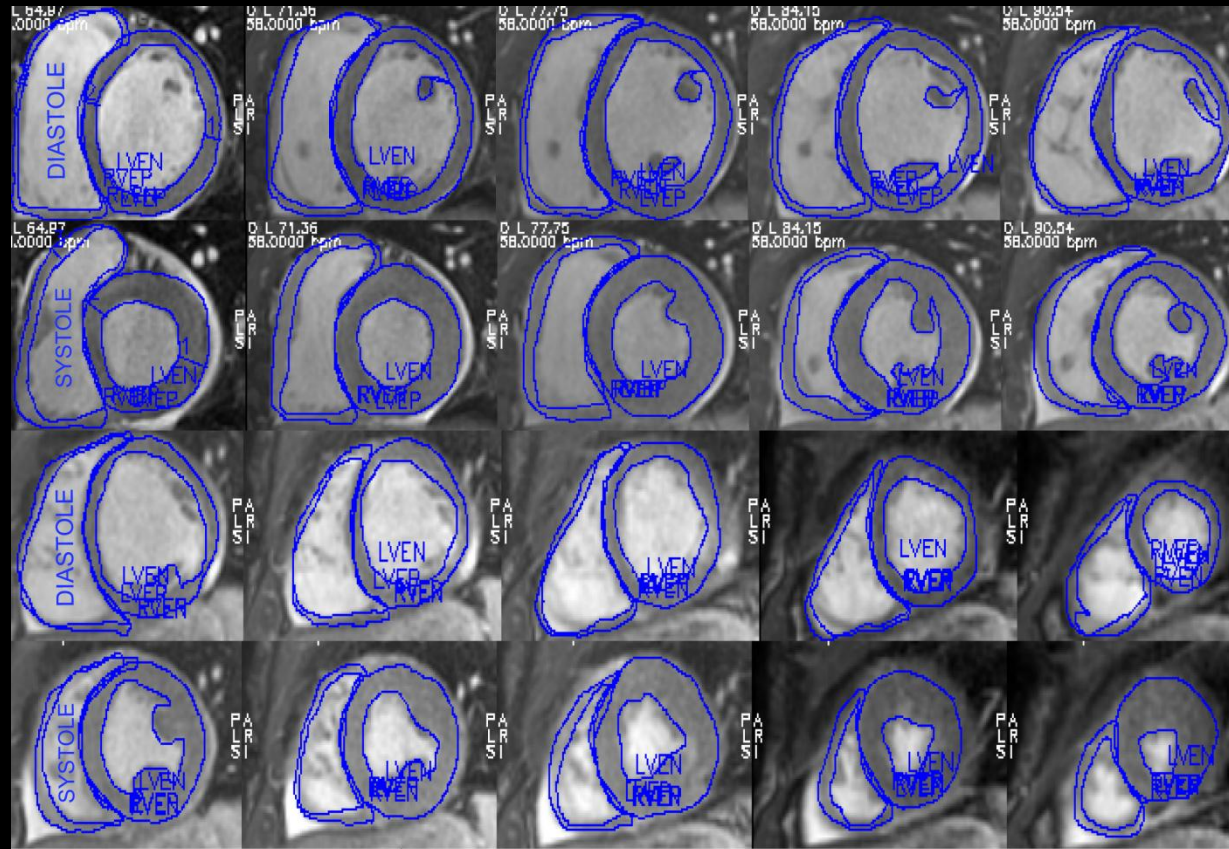
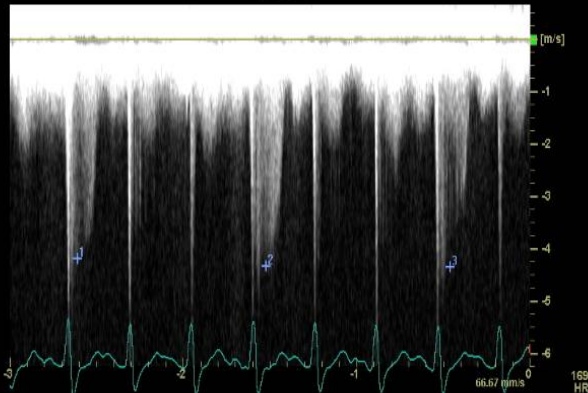
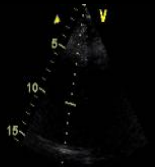


# Measuring wall stress

## Hybrid technique

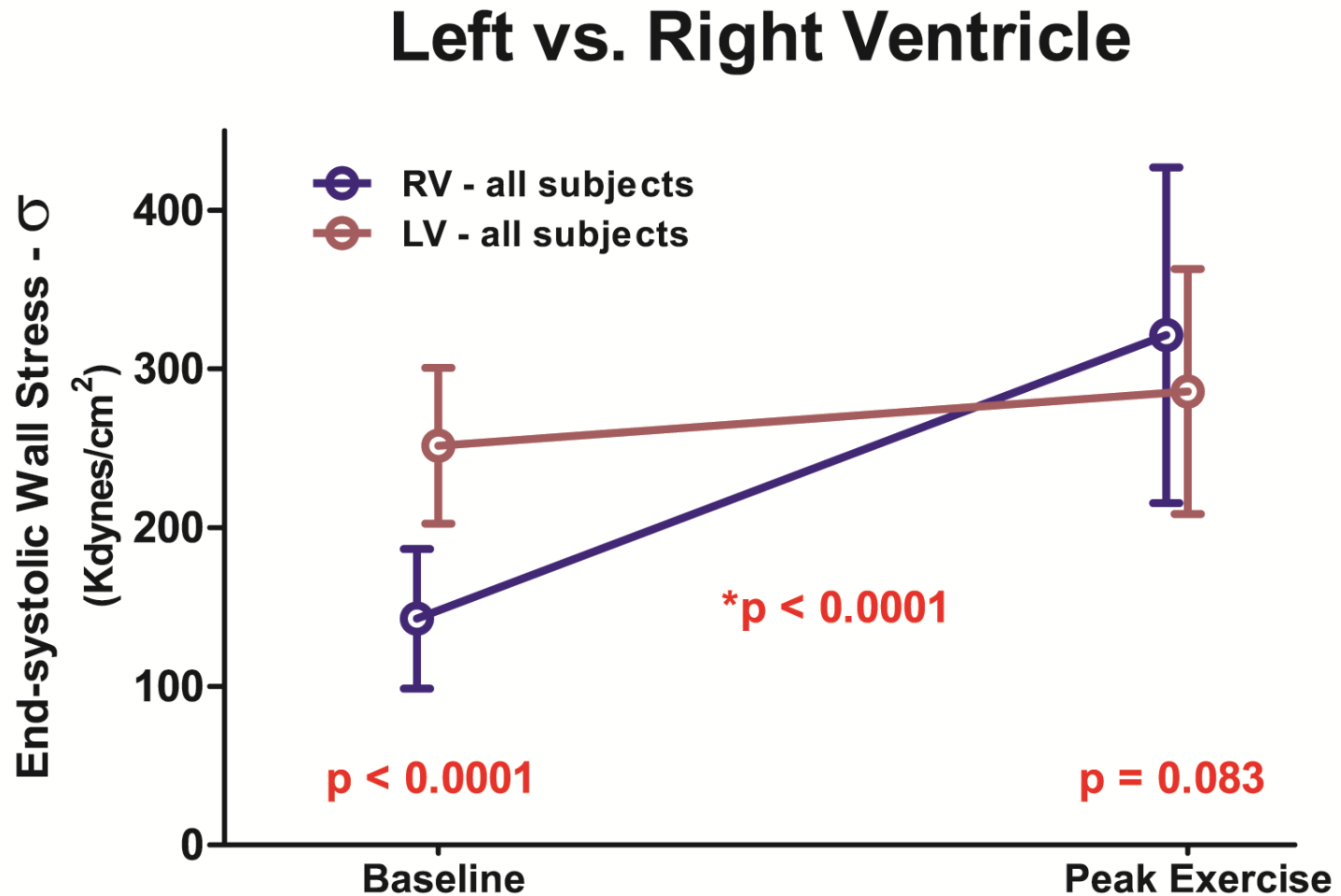
- Volumes and mass from CMR
- Change in volumes from echo VTI
- Pressures
  - SBP: arterial line
  - PASP: echo

3	TR Vmax, 9	4.360 m/s
	TR maxPG, 9	75.68 mmHg
2	TR Vmax, 9	4.332 m/s
	TR maxPG, 9	75.06 mmHg
1	TR Vmax, 9	4.189 m/s
	TR maxPG, 9	70.18 mmHg





# Change in wall stress with exercise

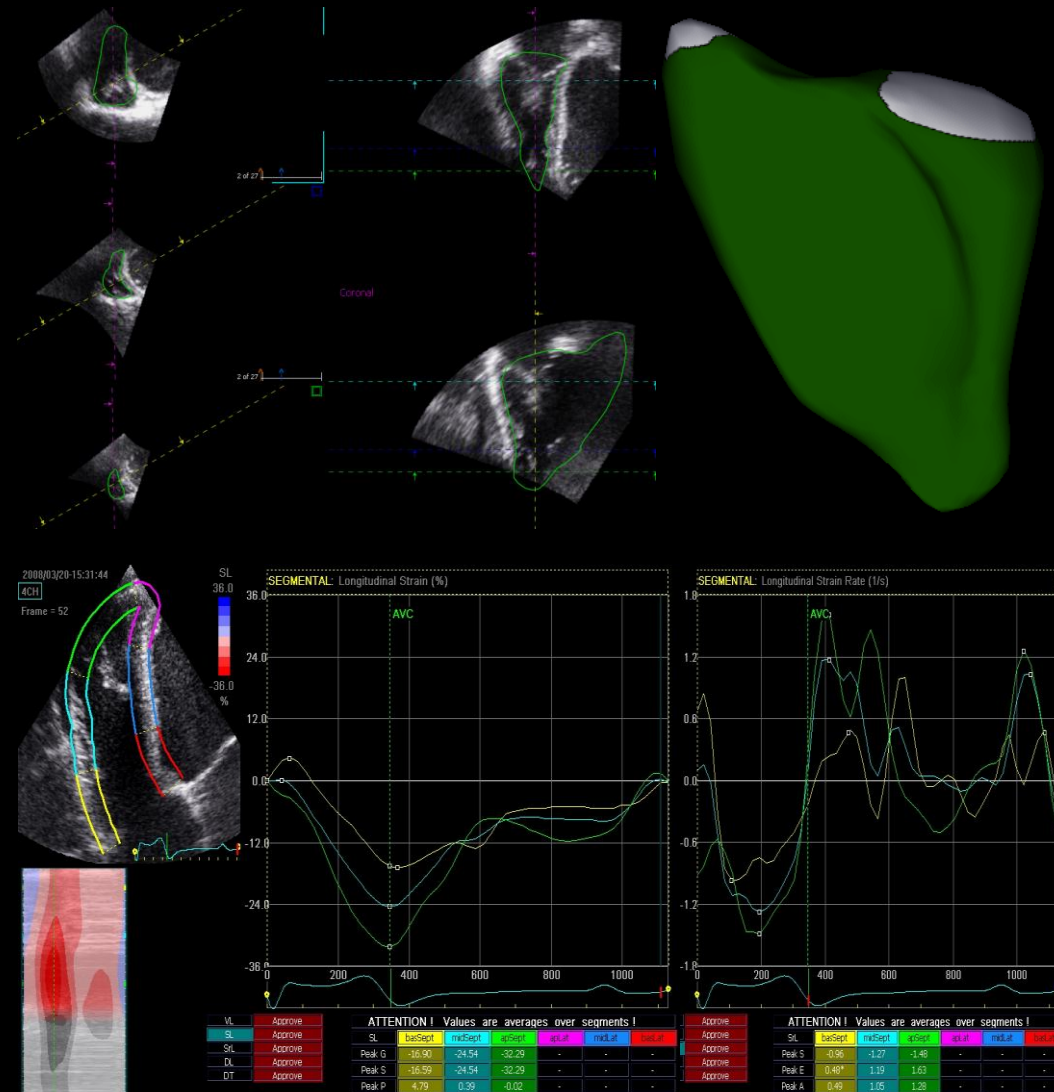


*...and after 8  
hours*



# Methods - echocardiography

- 3D Echo (GE Vivid 7)  
LV and RV full volume acquisitions  
TomTec software  
Validated against CMR
- Strain and SR imaging  
2D speckle tracking  
RV and LV separately  
60 -90 frames per second
- Traditional  
RV Fractional Area Change  
Tricuspid Annular Plane  
Systolic Excursion



# Results: RV but not LV dysfunction

	Baseline	Post-race	Follow-up	p-value
Right Ventricular Measures				
RVEF (%)	51.0 ± 3.6	46.4 ± 6.5	50.0 ± 3.8	<0.0001
RVFAC (%)	51.5 ± 6.0	44.3 ± 11.2	49.8 ± 6.6	<0.0001
TAPSE	24.9 ± 3.9	24.0 ± 4.5	26.5 ± 4.1	0.035
RV strain (%)	27.2 ± 3.4	23.7 ± 3.7	25.6 ± 3.0	0.001
RV SRs (s <sup>-1</sup> )	1.42 ± 0.24	1.26 ± 0.23	1.29 ± 0.19	0.008
Left Ventricular Measures				
LVEF (%)	56.4 ± 5.2	57.5 ± 5.6	58.8 ± 5.1	0.147
LV strain (%)	18.4 ± 3.7	16.9 ± 2.8	17.7 ± 2.3	0.071
LV SRs (s <sup>-1</sup> )	0.98 ± 0.26	0.95 ± 0.15	0.89 ± 0.13	0.13

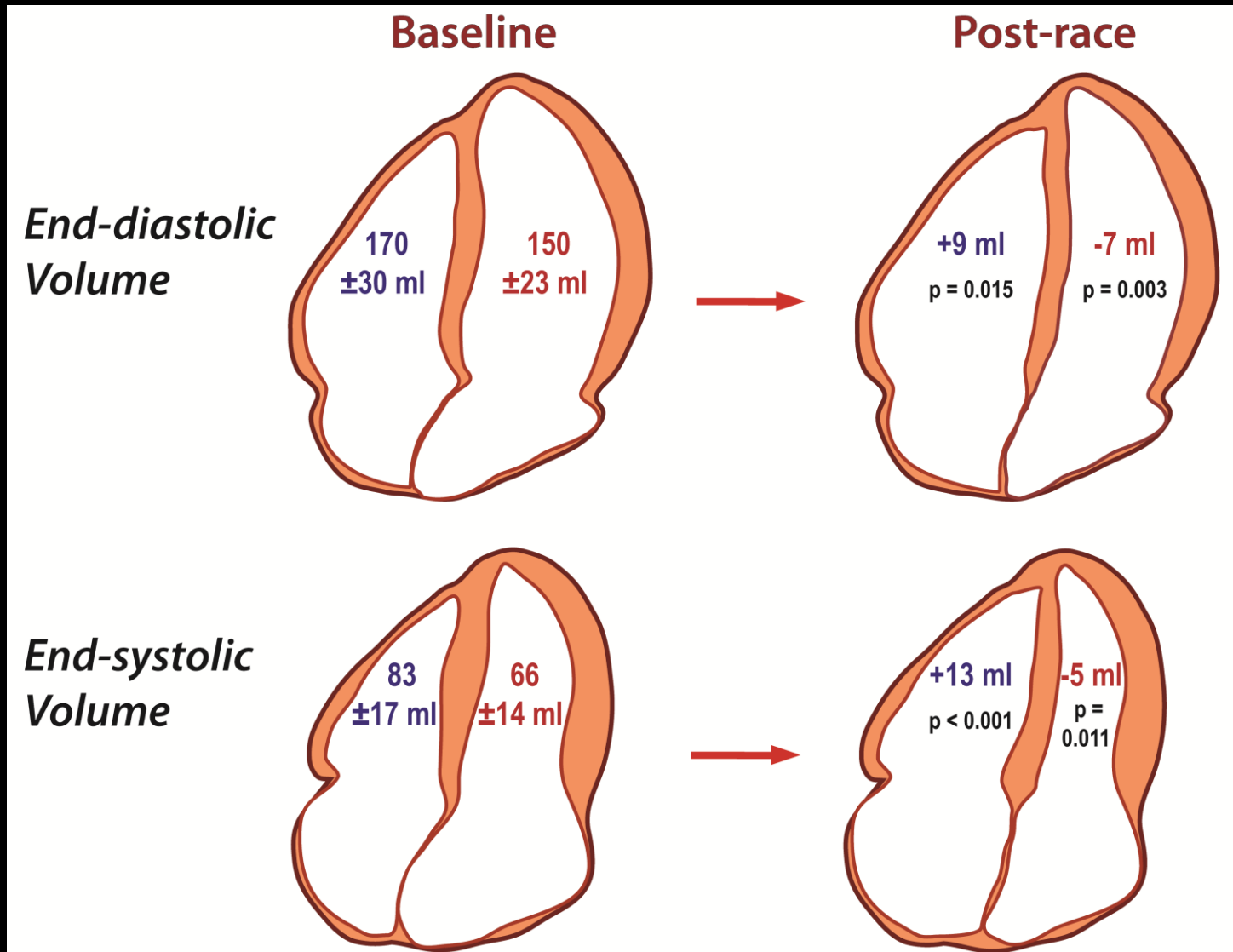
**ALL RV measures decreased whilst NO LV measures changed**

La Gerche, Heidbuchel, Prior et al. Eur Heart J In Press

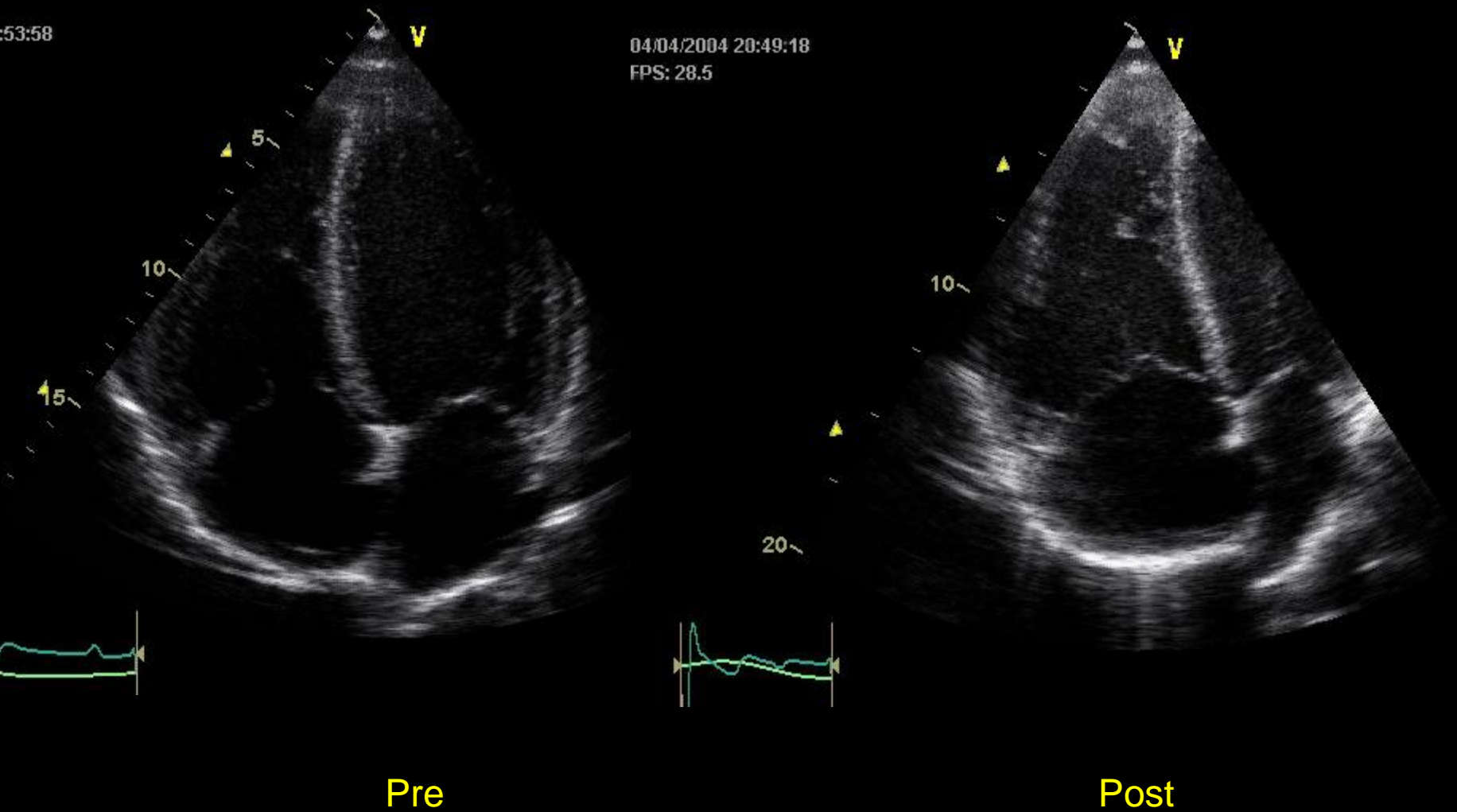




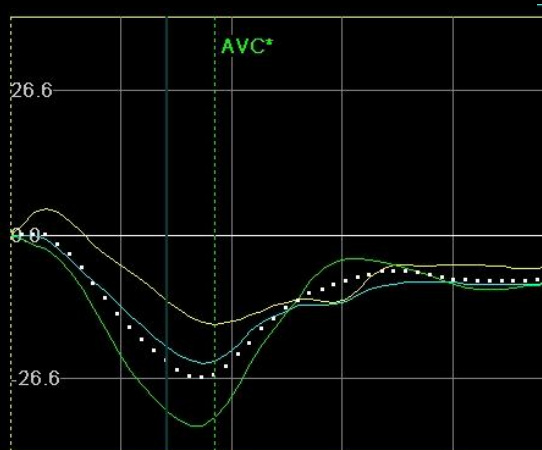
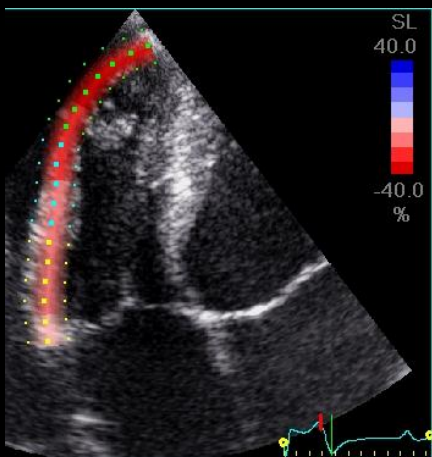
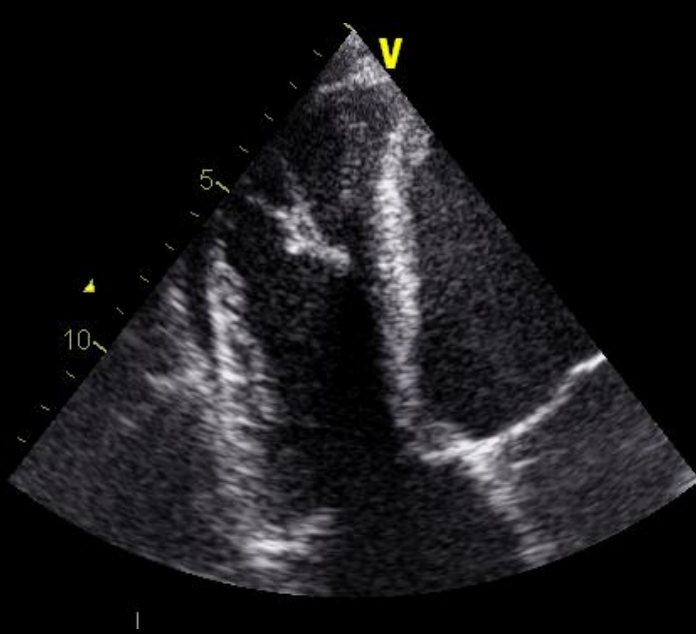
# RV *dilates* whilst the LV *shrinks*



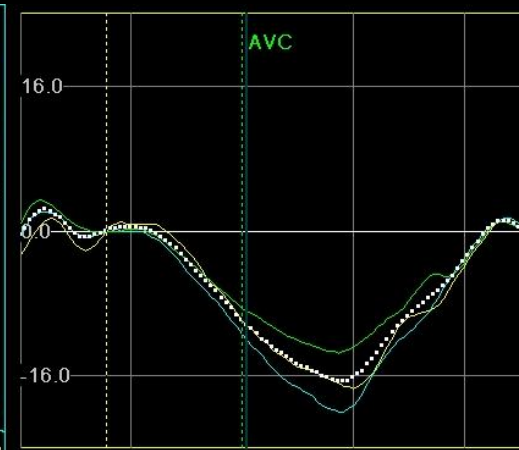
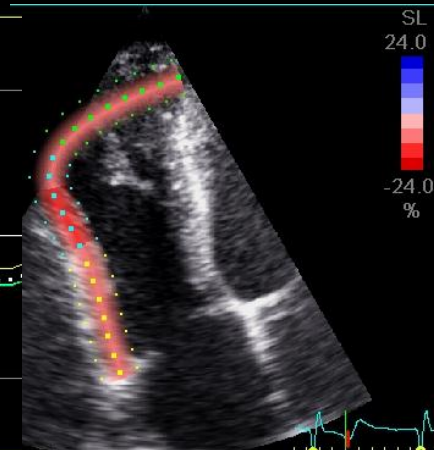
# Effect of prolonged intense exercise on the RV



# Effect of prolonged intense exercise on the RV

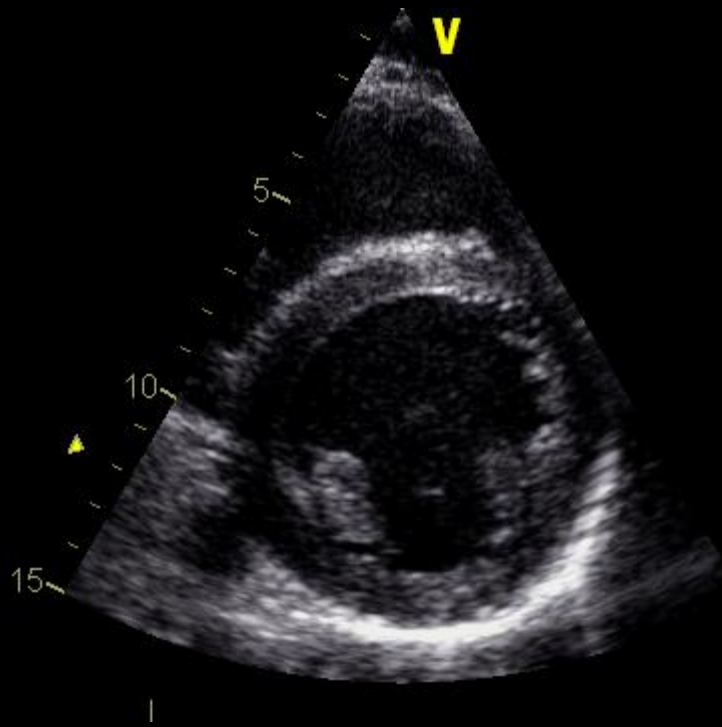


Pre

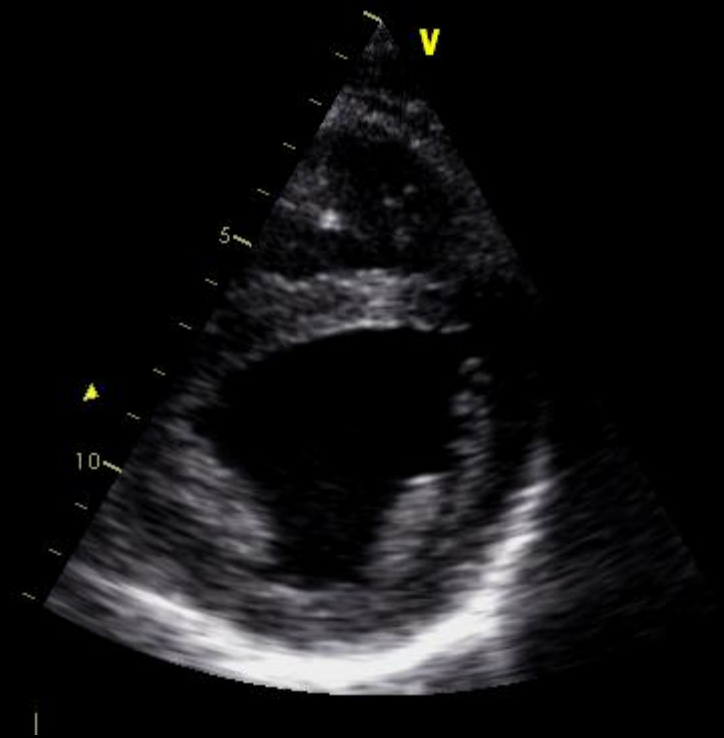


Post

# Effect of prolonged intense exercise on the RV



Pre



Post

# The RV

“the *Achilles heel* of the  
exercising heart”



# Why is the RV the Achilles heel?

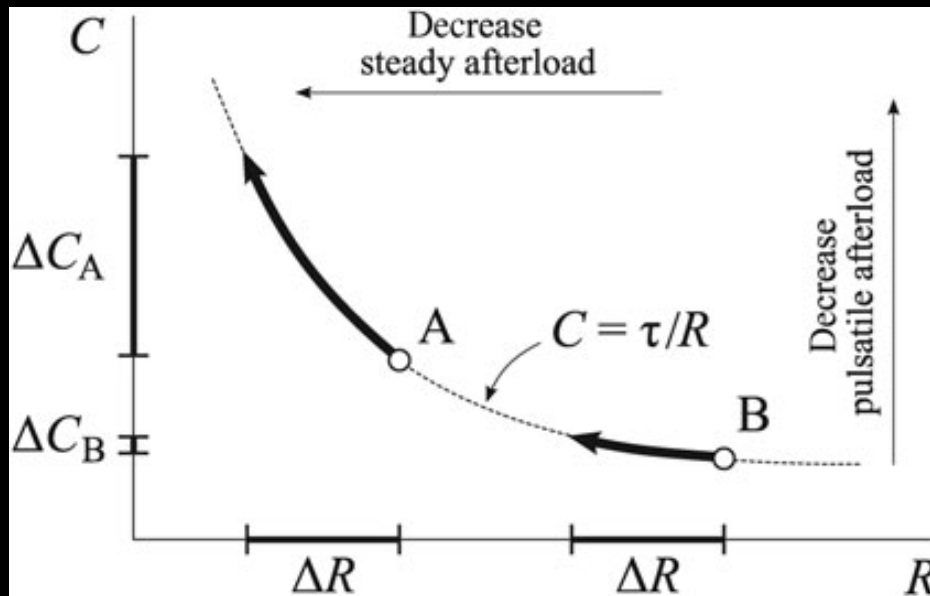
	Left Ventricle	Right Ventricle
Cardiac Output (L/min)	5	5
Vascular resistance (dyne-sec.cm <sup>5</sup> )	1100	70
Afterload Pressure (mmHg)	130/ 75 (85)	25/ 9 (15)
<b>Exercise</b>		
Cardiac Output (L/min)	25	25
Vascular resistance (dyne-sec.cm <sup>5</sup> )	↓↓↓	↓
Afterload Pressure (mmHg)	↑	↑↑↑



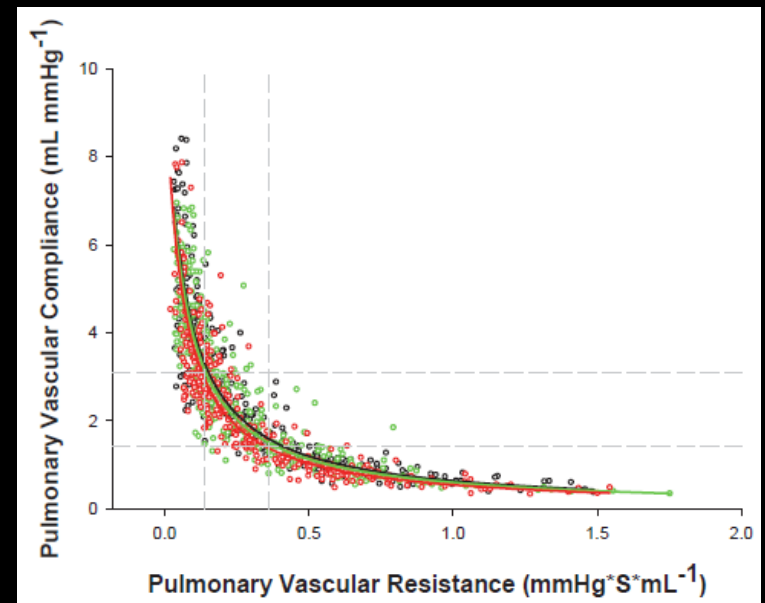
**It's all about hoses**



# In a pulsatile circulation we need to consider compliance



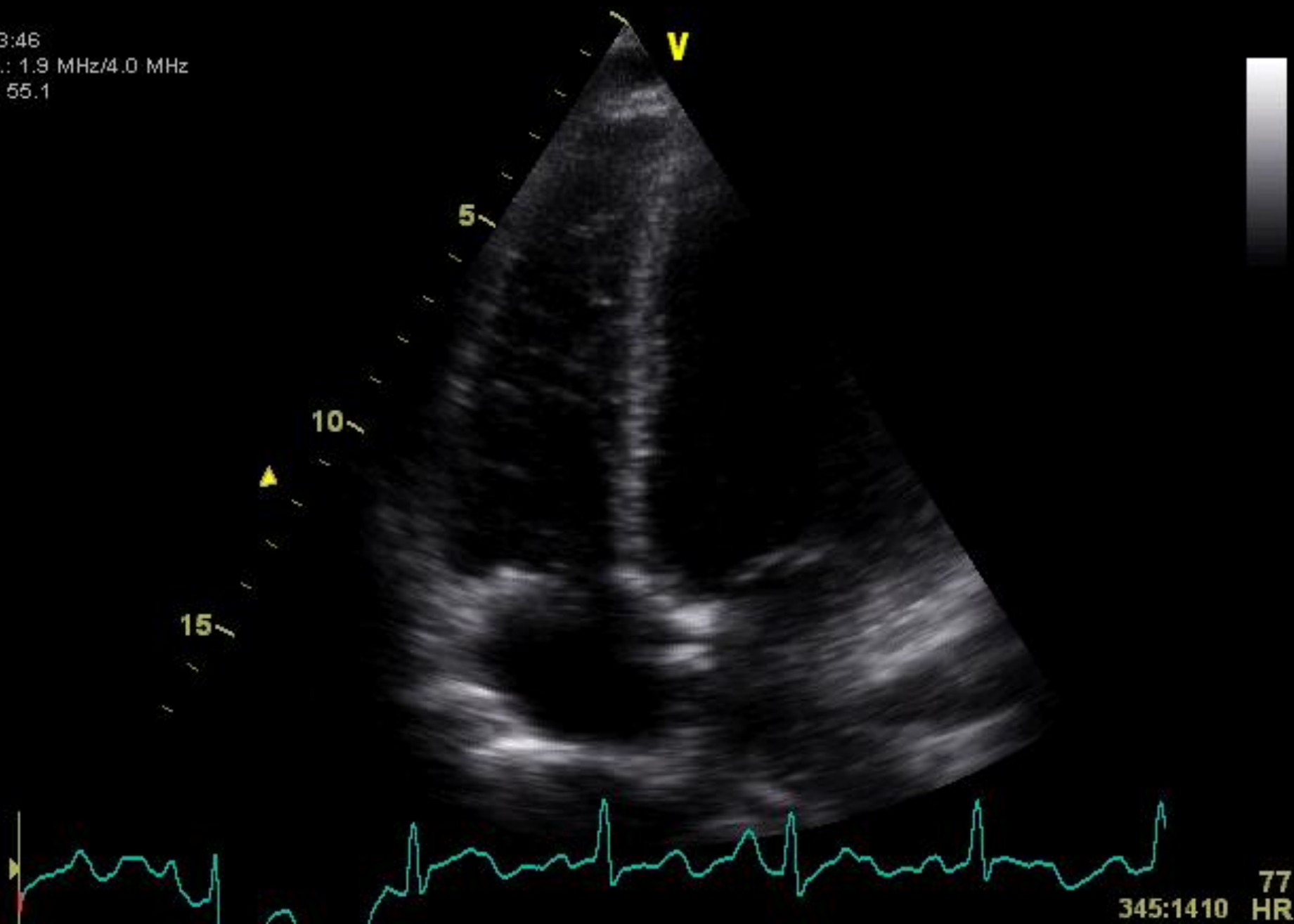
Lankhaar et al. *EHJ* 2008



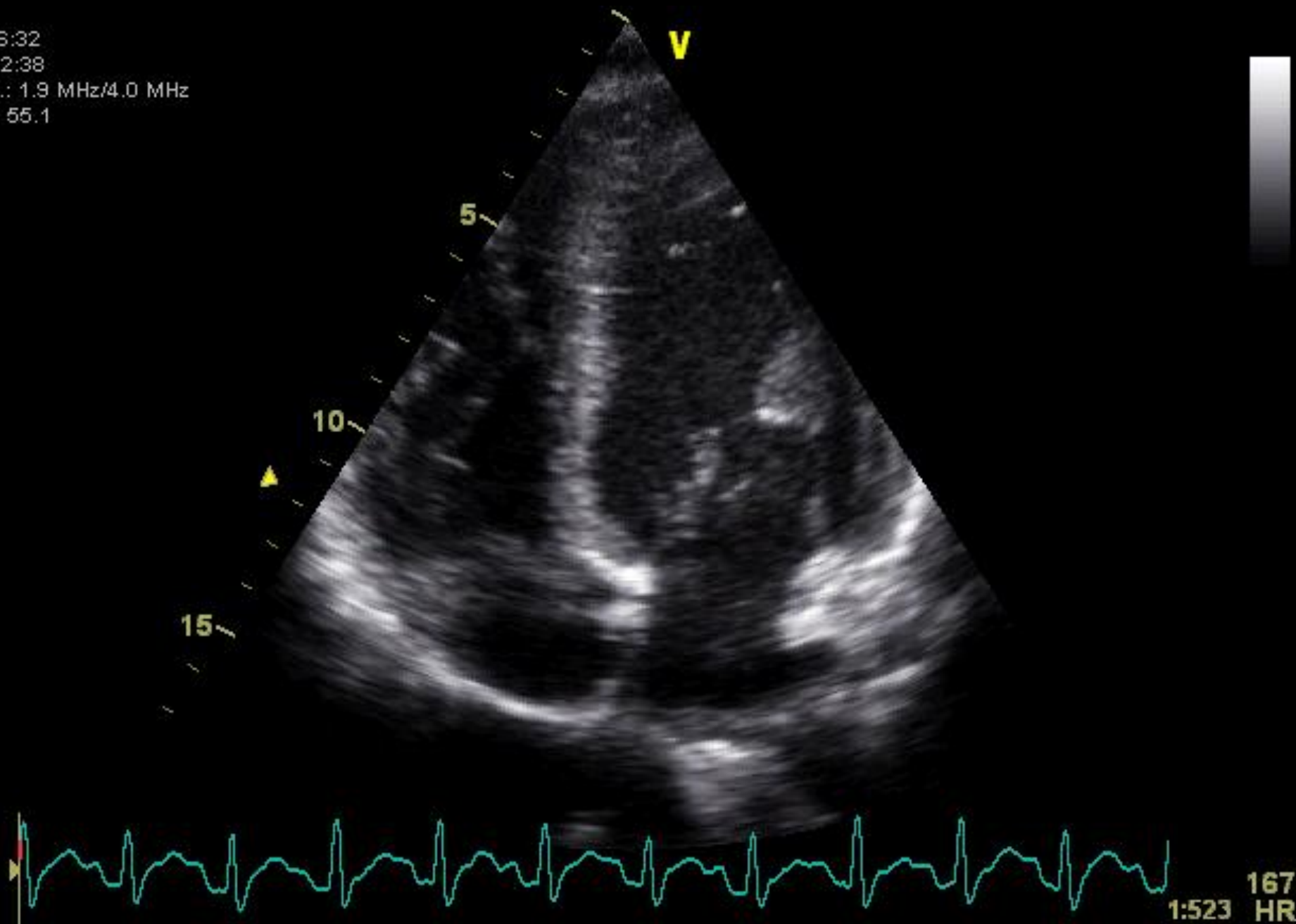
Tedford, Kass et al. *Circ* 2011

- To maximise *pulsatile* flow you want big vessels that can distend
- Flow  $\propto P \times r^4 / \eta \times L$

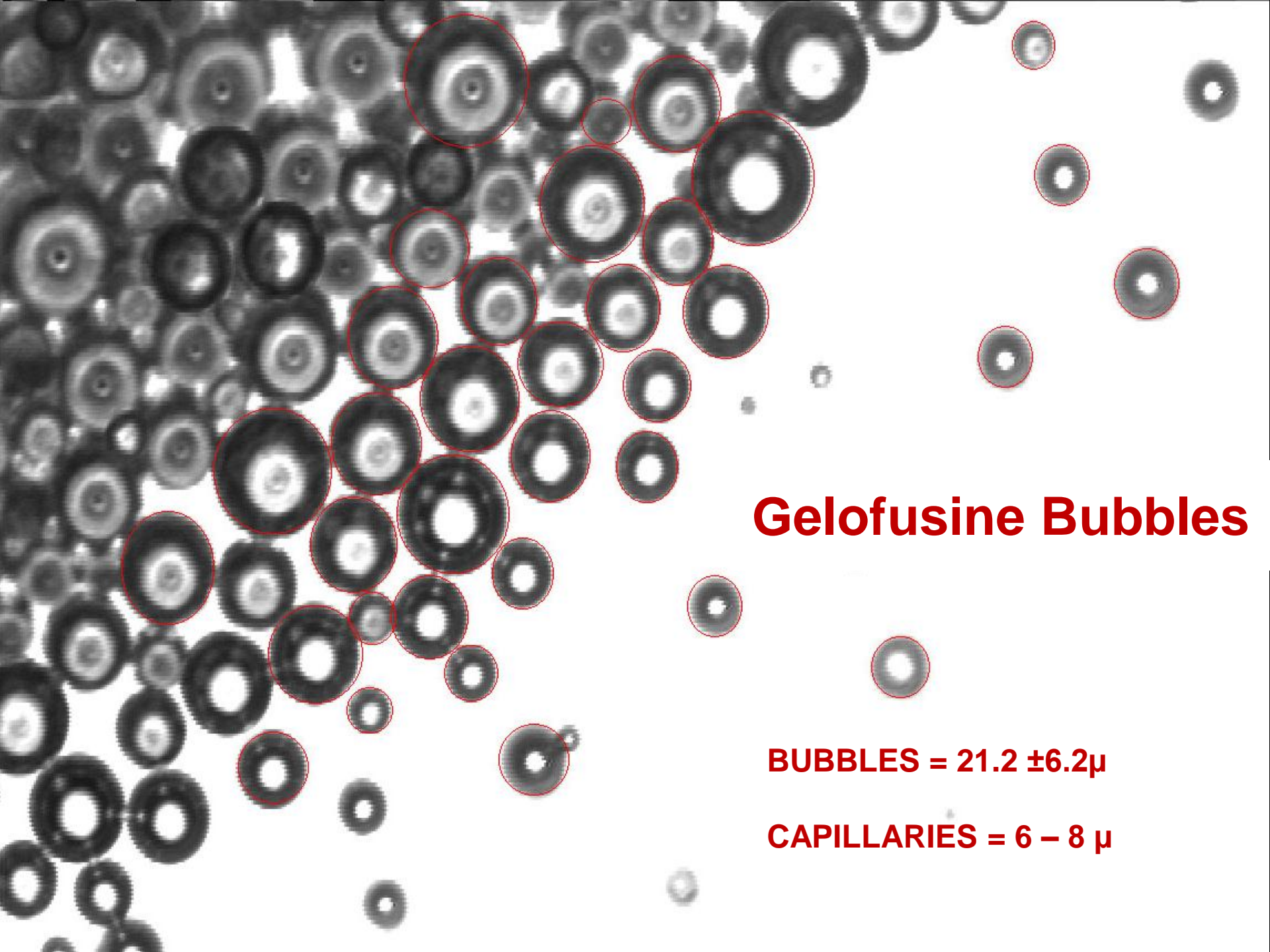
19:43:46  
Freq.: 1.9 MHz/4.0 MHz  
FPS: 55.1



19:26:32  
T1: 32:38  
Freq.: 1.9 MHz/4.0 MHz  
FPS: 55.1





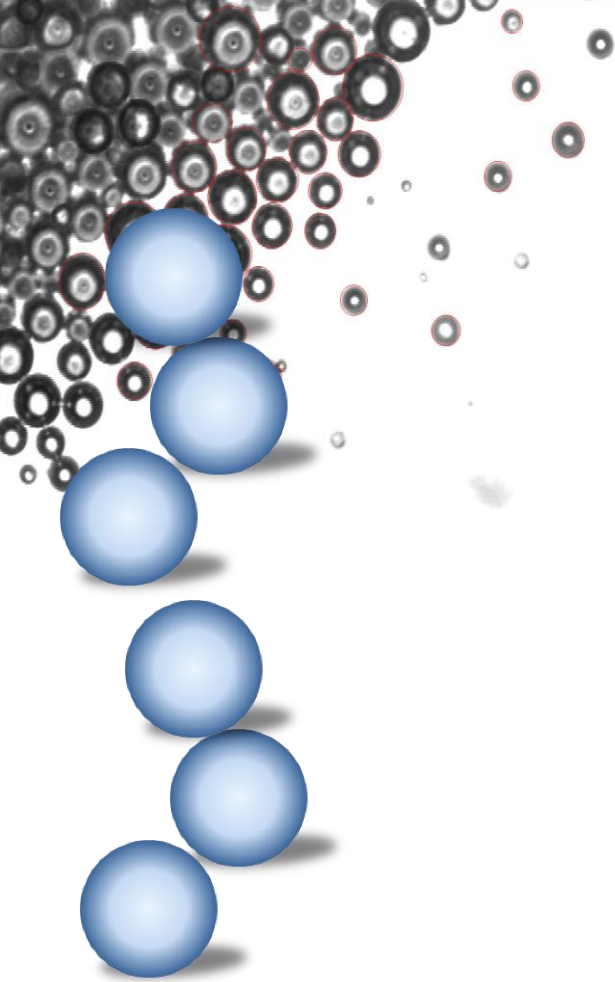


## **Gelofusine Bubbles**

**BUBBLES =  $21.2 \pm 6.2 \mu$**

**CAPILLARIES =  $6 - 8 \mu$**

? indicates larger vessels



**exercise**



.....and larger calibre vessels result in  
lower pressures and resistance  
and higher flows



# Pulmonary transit of contrast (PTAC)

- 40 athletes and 15 non-athletes
- Graded PTAC
- Equal proportion of PTAC

## Low Pulmonary Transit of Agitated Contrast

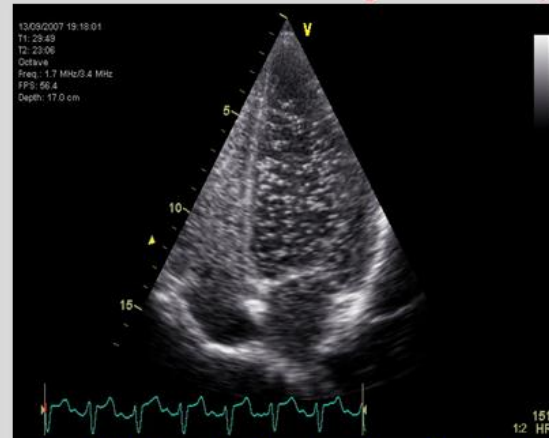


Grade I:  $\geq 4$  bubbles in LV

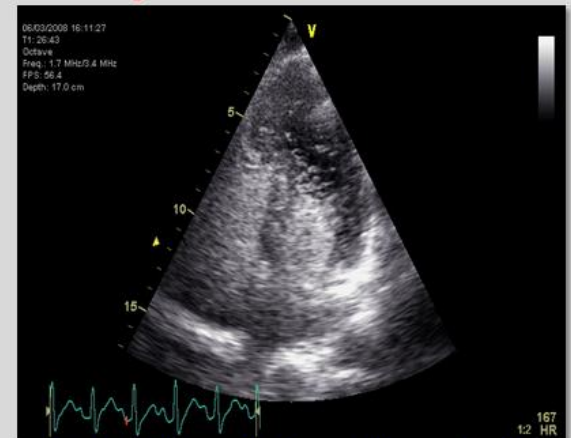


Grade II:  $\geq 20$  bubbles in LV

## High Pulmonary Transit of Agitated Contrast



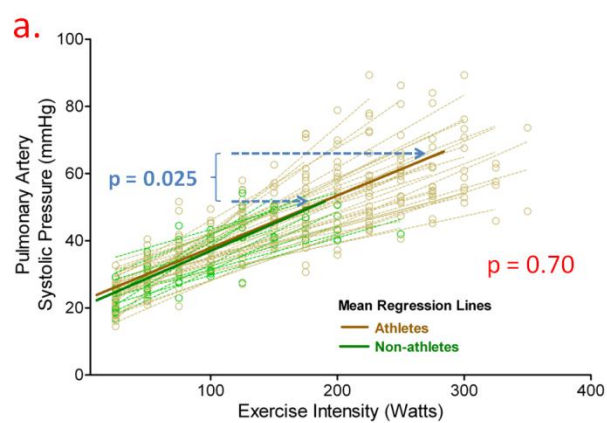
Grade III: LV opacification less intense than the RV



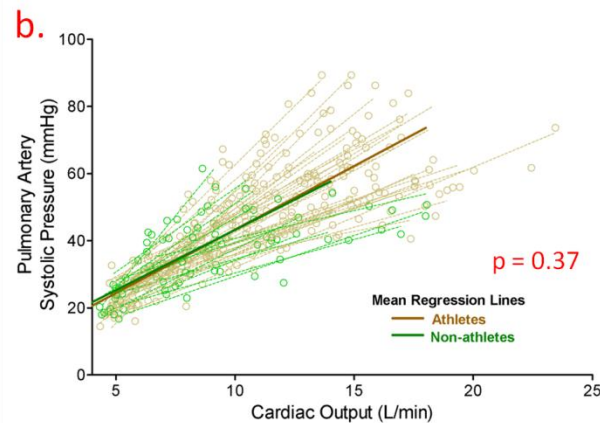
Grade IV: LV opacification as intense as the RV

# Lower pulmonary pressures and resistance

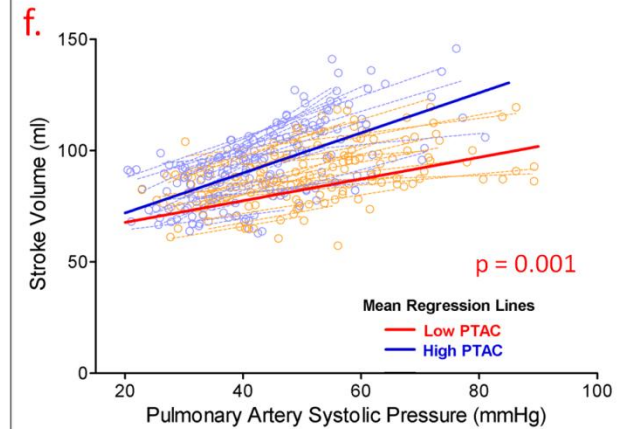
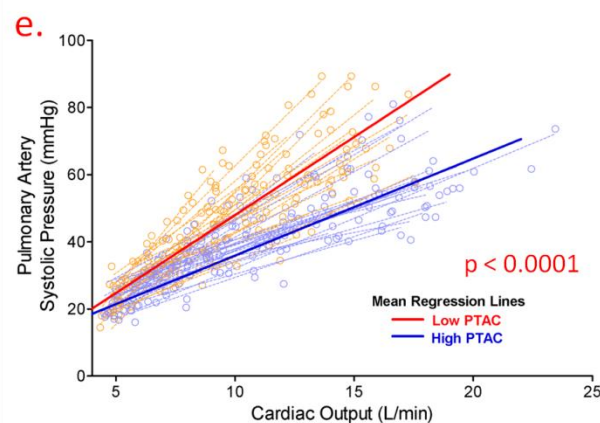
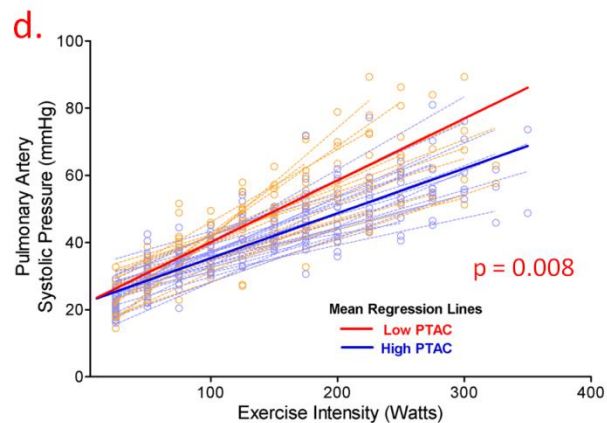
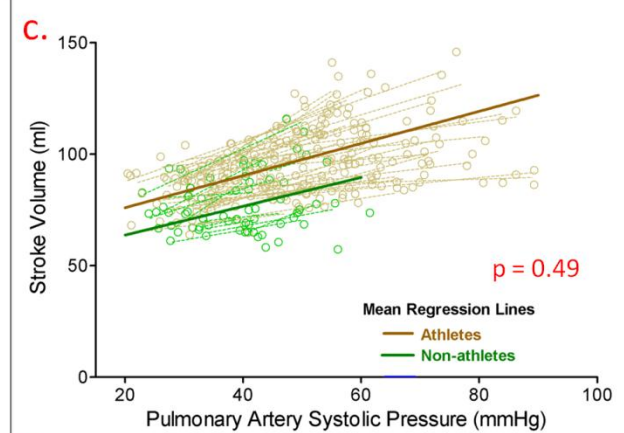
Pulmonary artery pressure



Pulmonary vascular resistance



Pulmonary vascular compliance



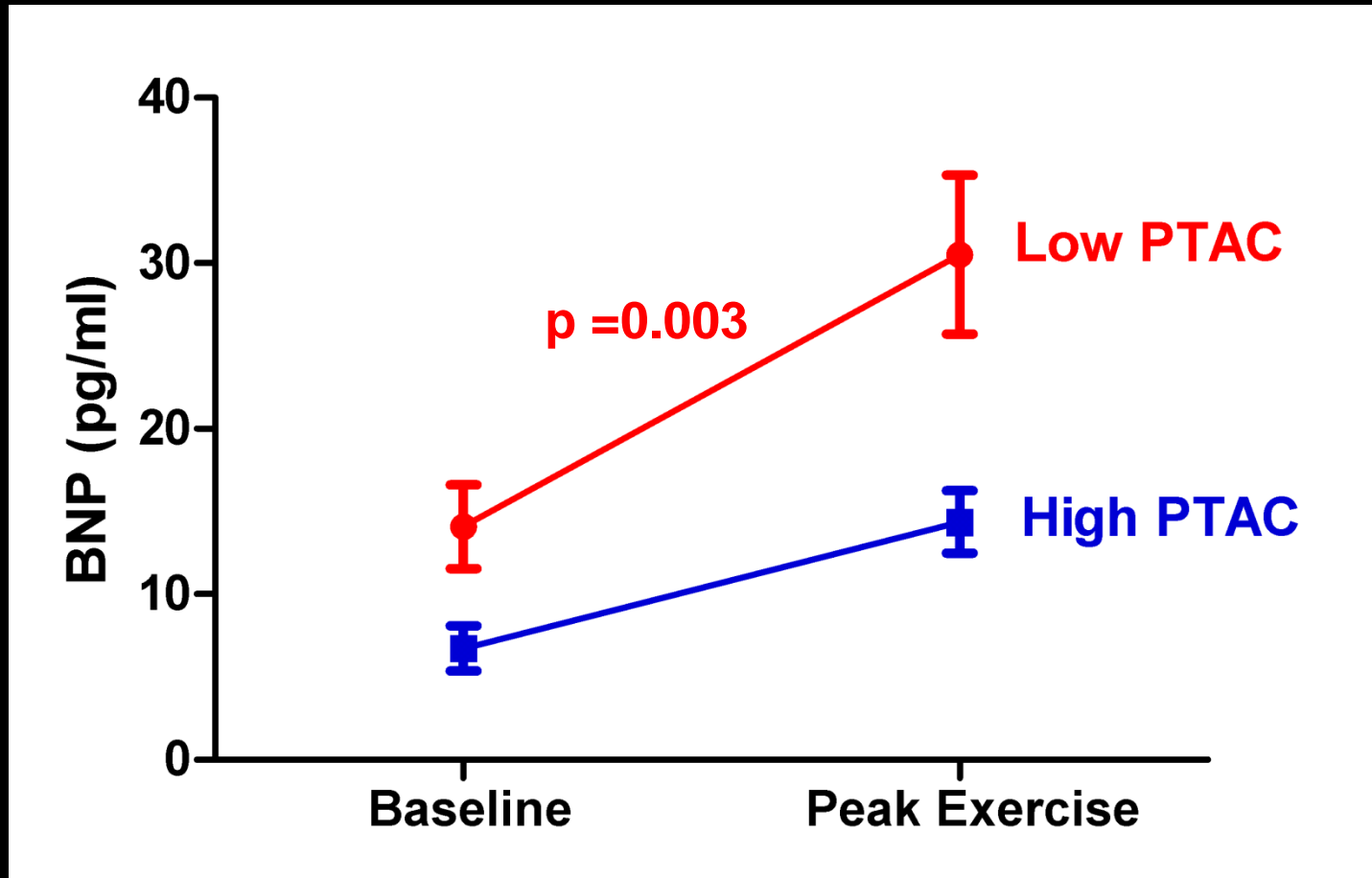
# PTAC predicts performance

- PTAC was associated with a 16% greater peak exercise CO
- PTAC was an independent predictor of  $\text{VO}_2$  max and maximal exercise output

# Better RV function associated with PTAC

	Rest		p-value	Peak-Exercise		p-value
	High-PTAC	Low-PTAC	Baseline	High-PTAC	Low-PTAC	Interaction with exercise*
<i>RV Function</i>						
RV Sm (cm/s)	11.2±2.4	11.1±1.4	0.72	21.5±4.5†	18.9±2.9†	0.009
RV Em (cm/s)	11.3±3.4	10.9±2.1	0.98	33.6±6.7†	30.4±6.8†	0.010
RV IVA (cm/s <sup>2</sup> )	1.7±0.7	1.7±0.8	0.83	6.9±2.7†	5.1±1.9†	0.001
<i>LV function</i>						
LV Sm (cm/s)	6.4±1.6	6.3±1.2	0.64	13.5±3.2†	12.3±2.5†	0.10
LV Em (cm/s)	9.0±1.8	8.7±1.9	0.63	19.6±3.7†	17.6±4.0†	0.21
LV IVA (cm/s <sup>2</sup> )	1.4±1.4	1.3±1.0	0.75	3.4±1.1†	2.9±1.5†	0.35

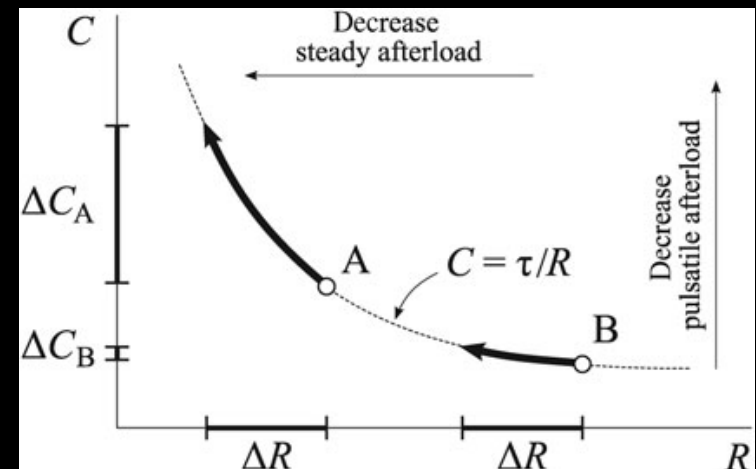
# Attenuated BNP increase in high PTAC



La Gerche et al. *J App Physiol* 2010

# Trans-pulmonary bubbles

- Are associated with enhanced pulmonary vessel hemodynamics, RV function and exercise capacity
- May be a surrogate measure of pulmonary vessel size and compliance
- ??? Early marker of pulmonary vessel pathology



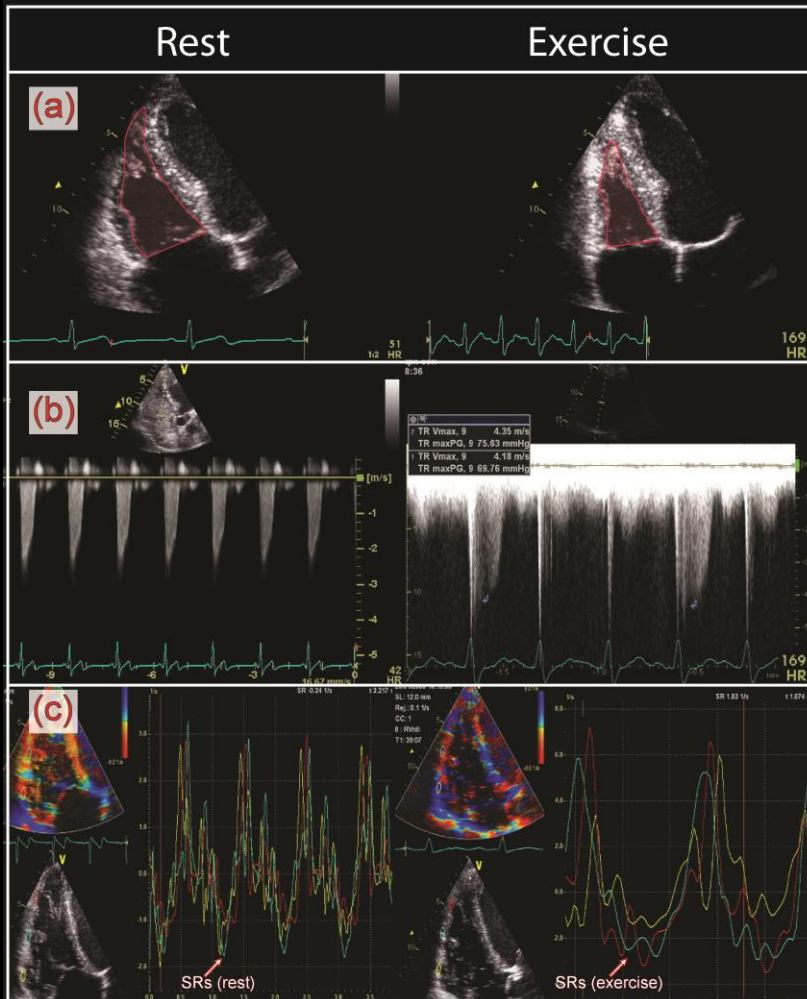
Lankhaar et al. *EHJ* 2008



So, RV function during  
exercise may be important  
but how can I measure it?



# RV Stress test



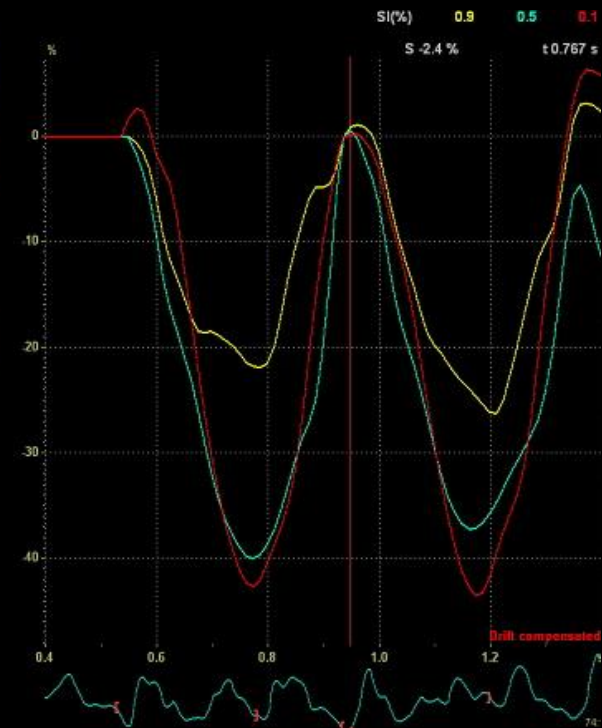
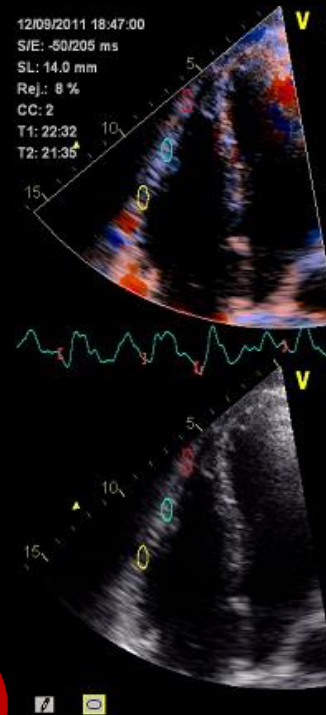
RV areas

PASP  
estimates

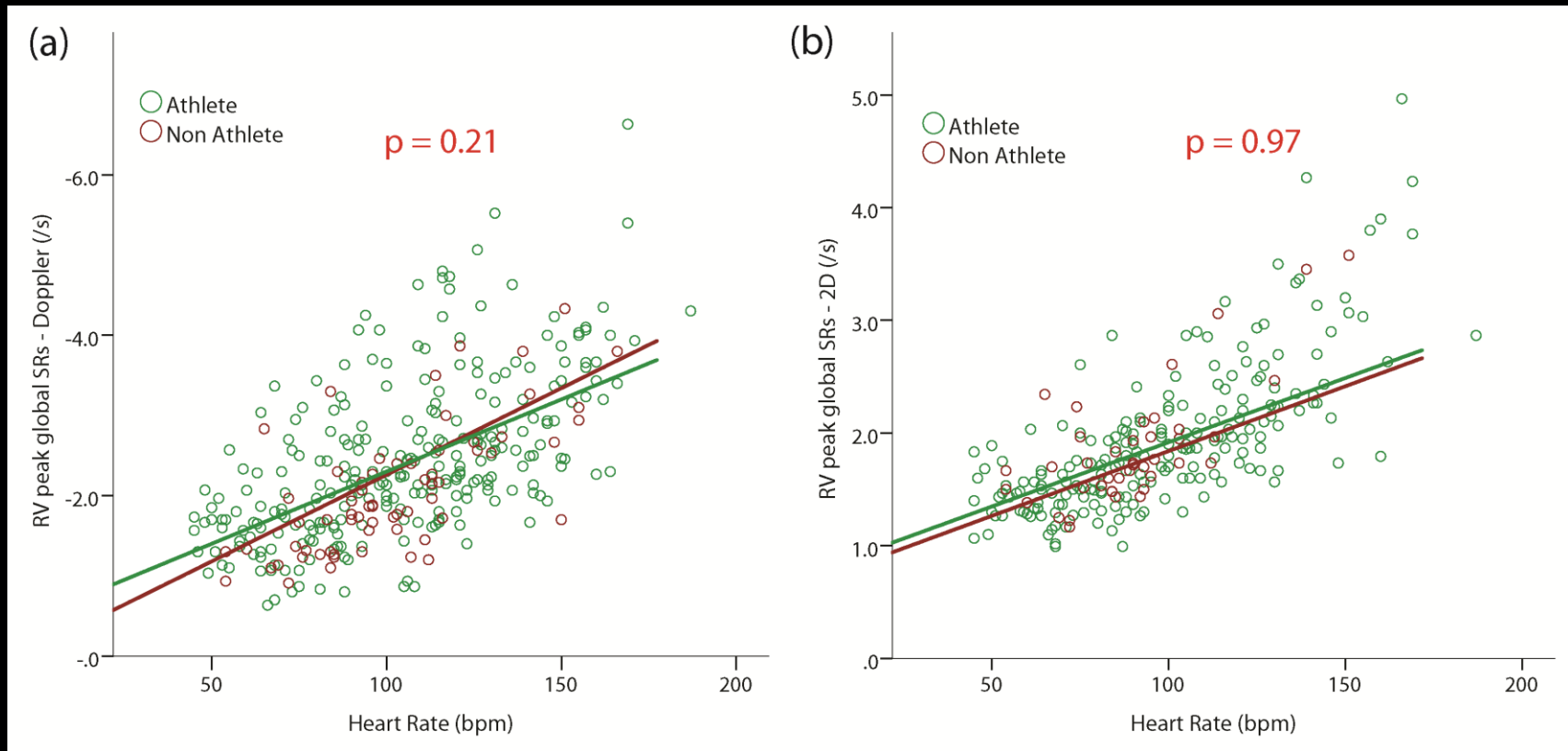
Pressure/area  
relationship

RV velocities, strain/ rate

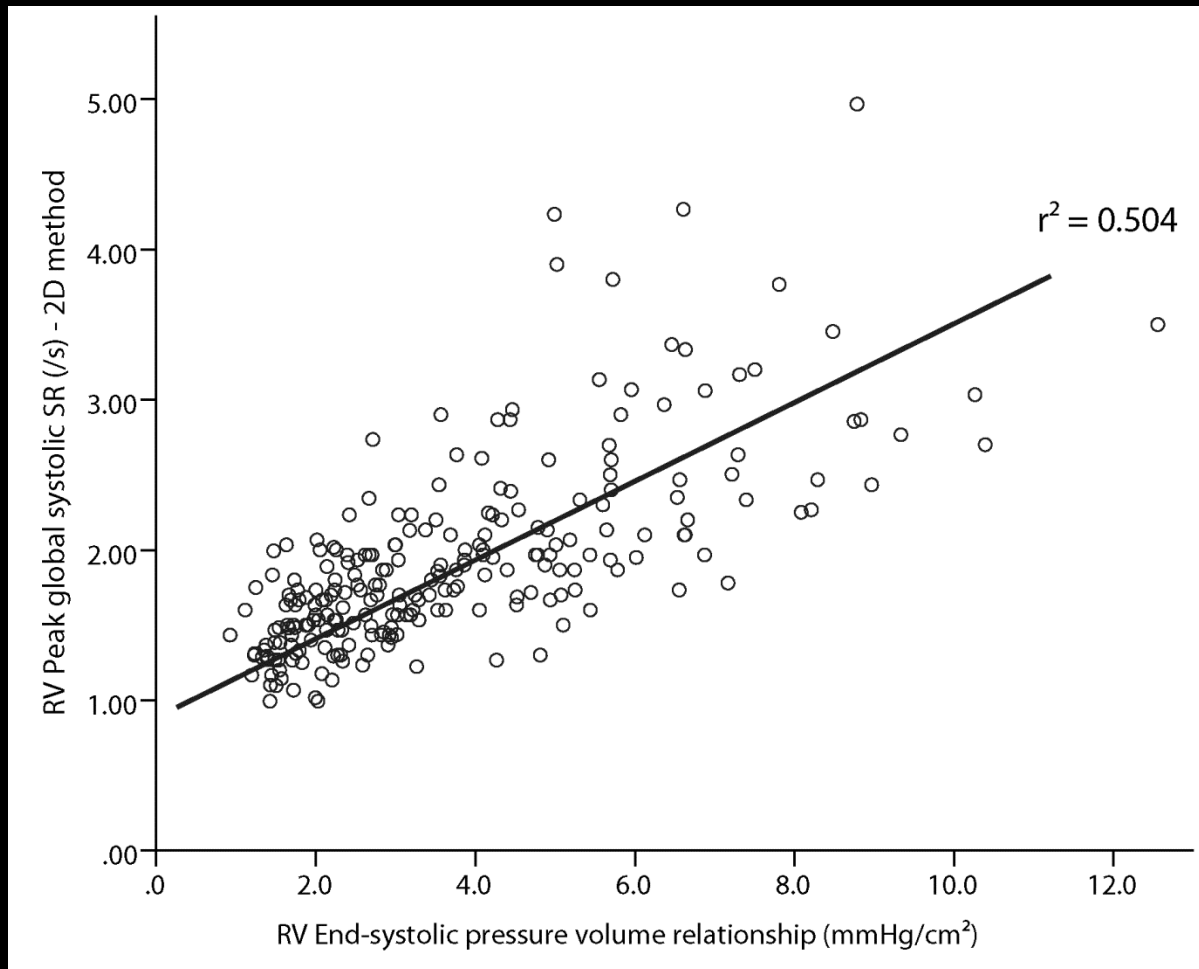
# Deformation during exercise



# RV strain rate increases with exercise

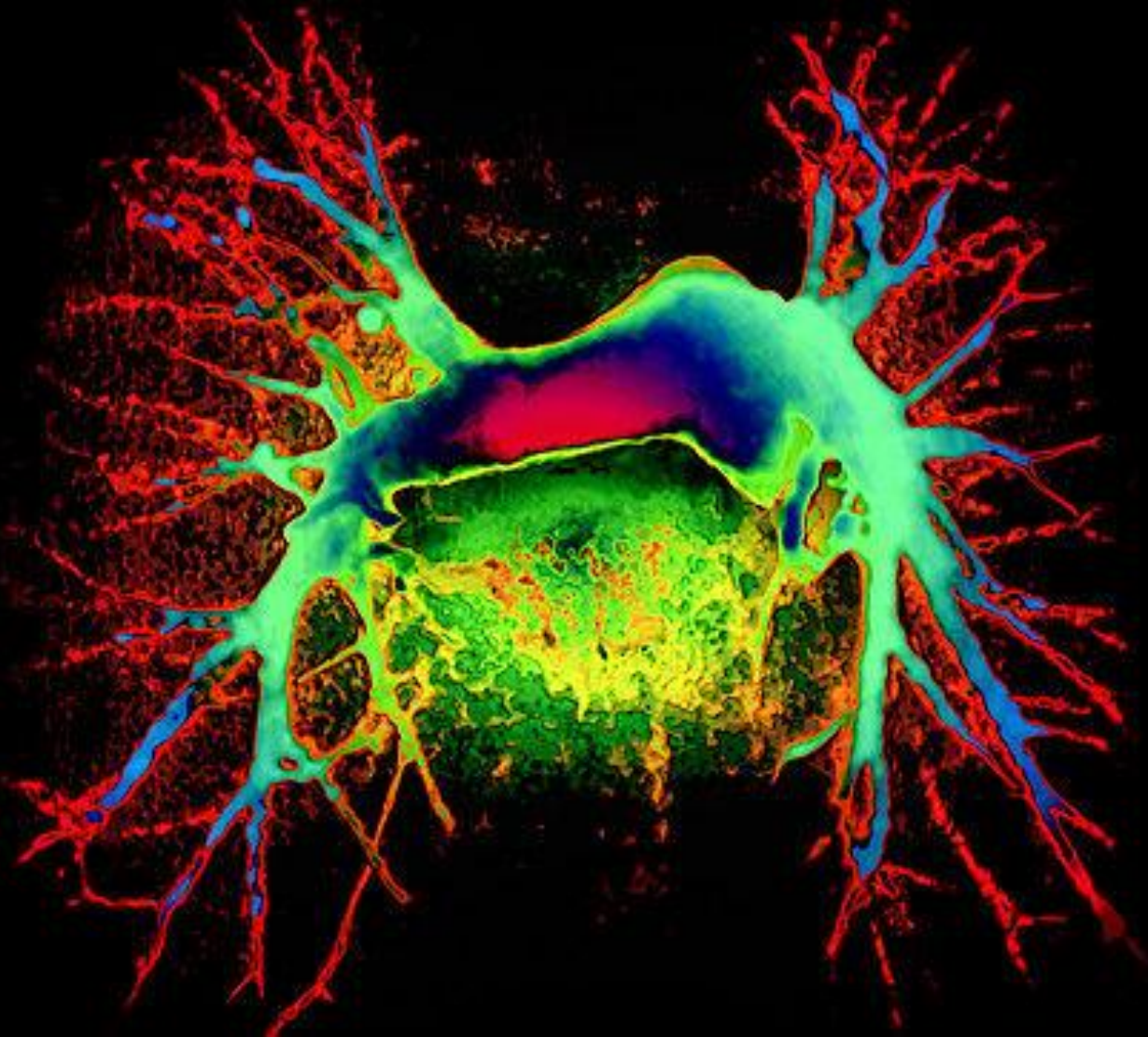


# Two non-invasive surrogates singing the same tune?

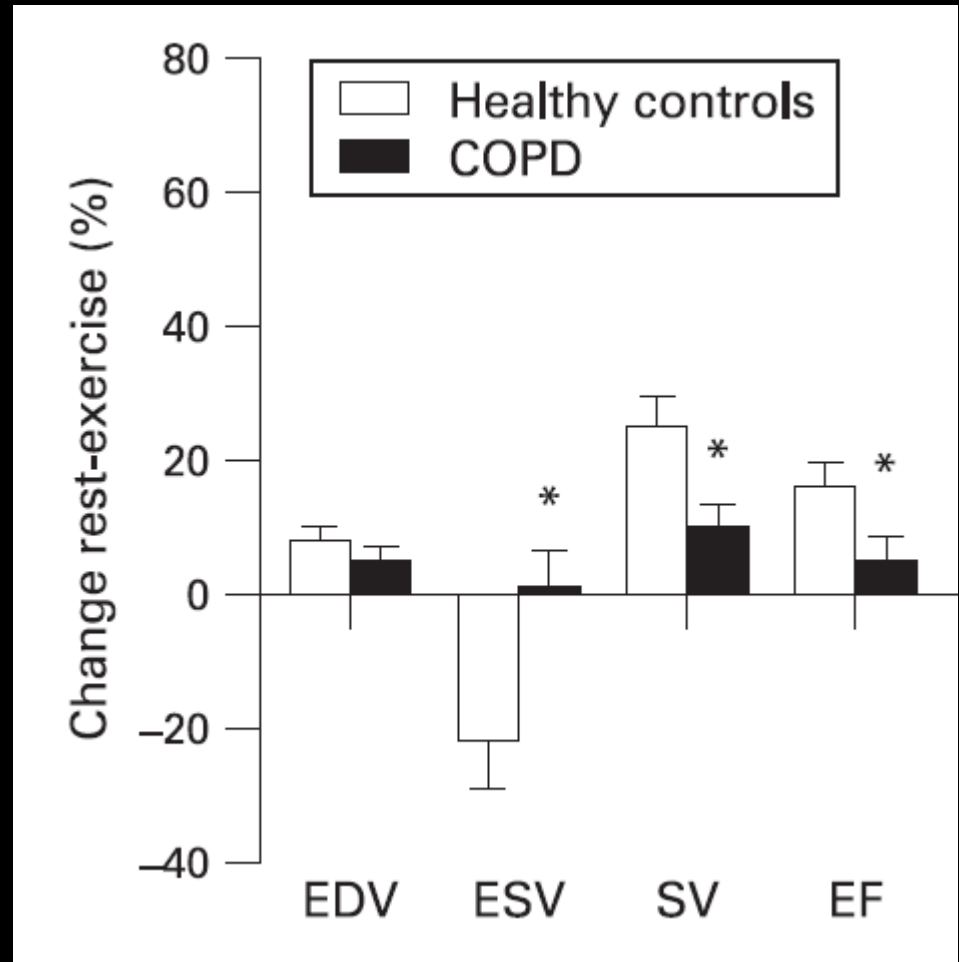




# Translational exercise science - from normal to pathology



# Failure of RV function limits exercise cardiac output in PHTn

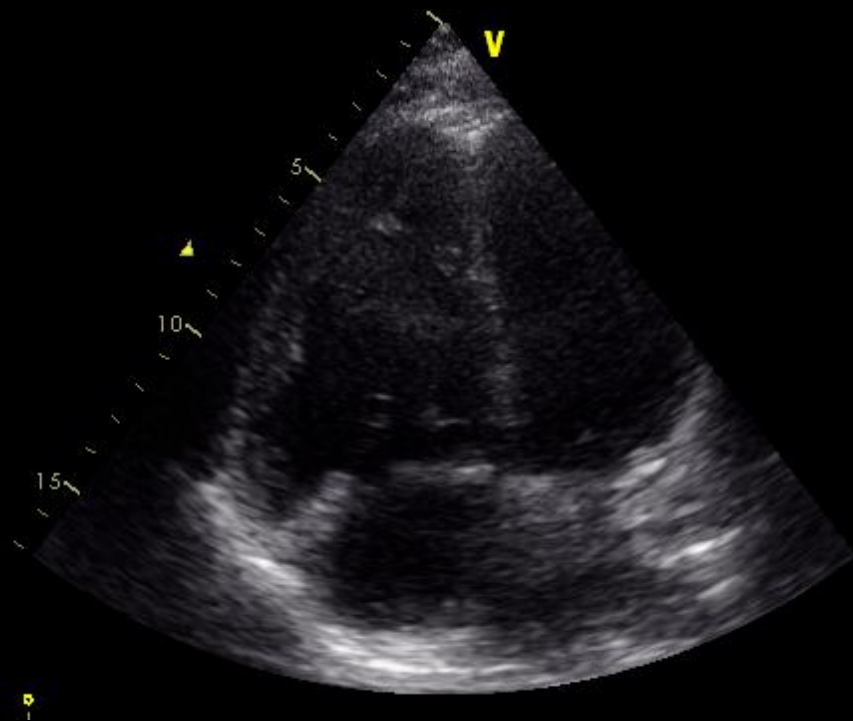


Holverda, Vonk Noordegraaf et al. *Heart* 2009

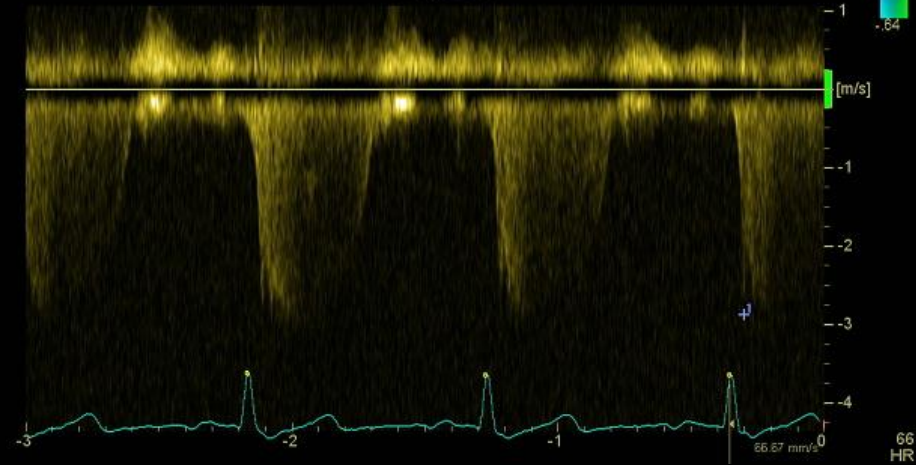
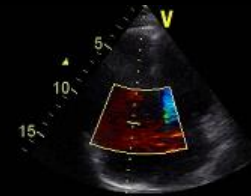
# Elite cyclist with ? subtle ARVC



# 28 yo lady with recently diagnosed ASD

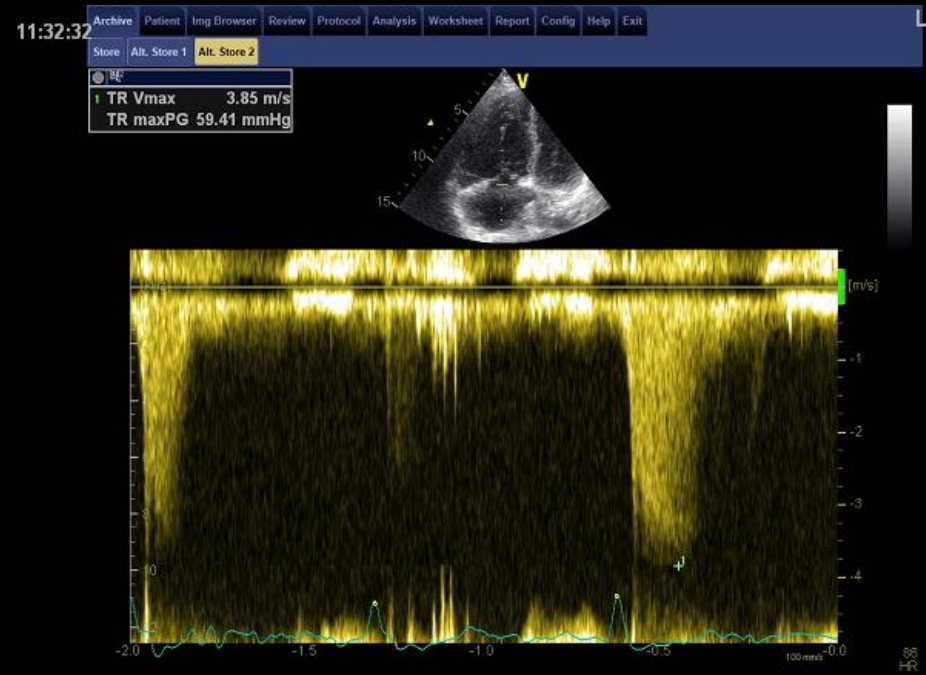
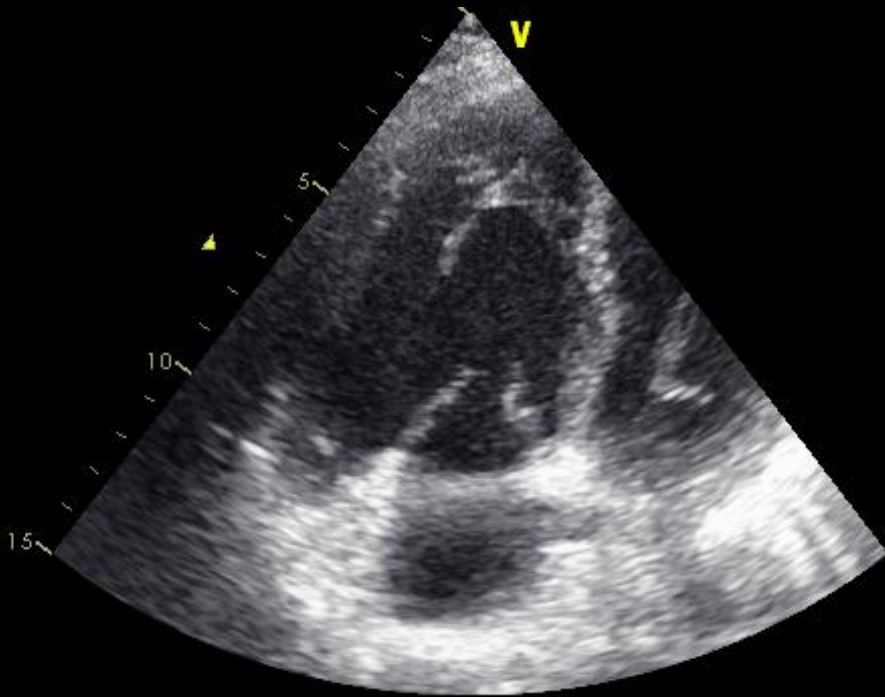


TR Vmax 2.88 m/s  
TR maxPG 33.21 mmHg



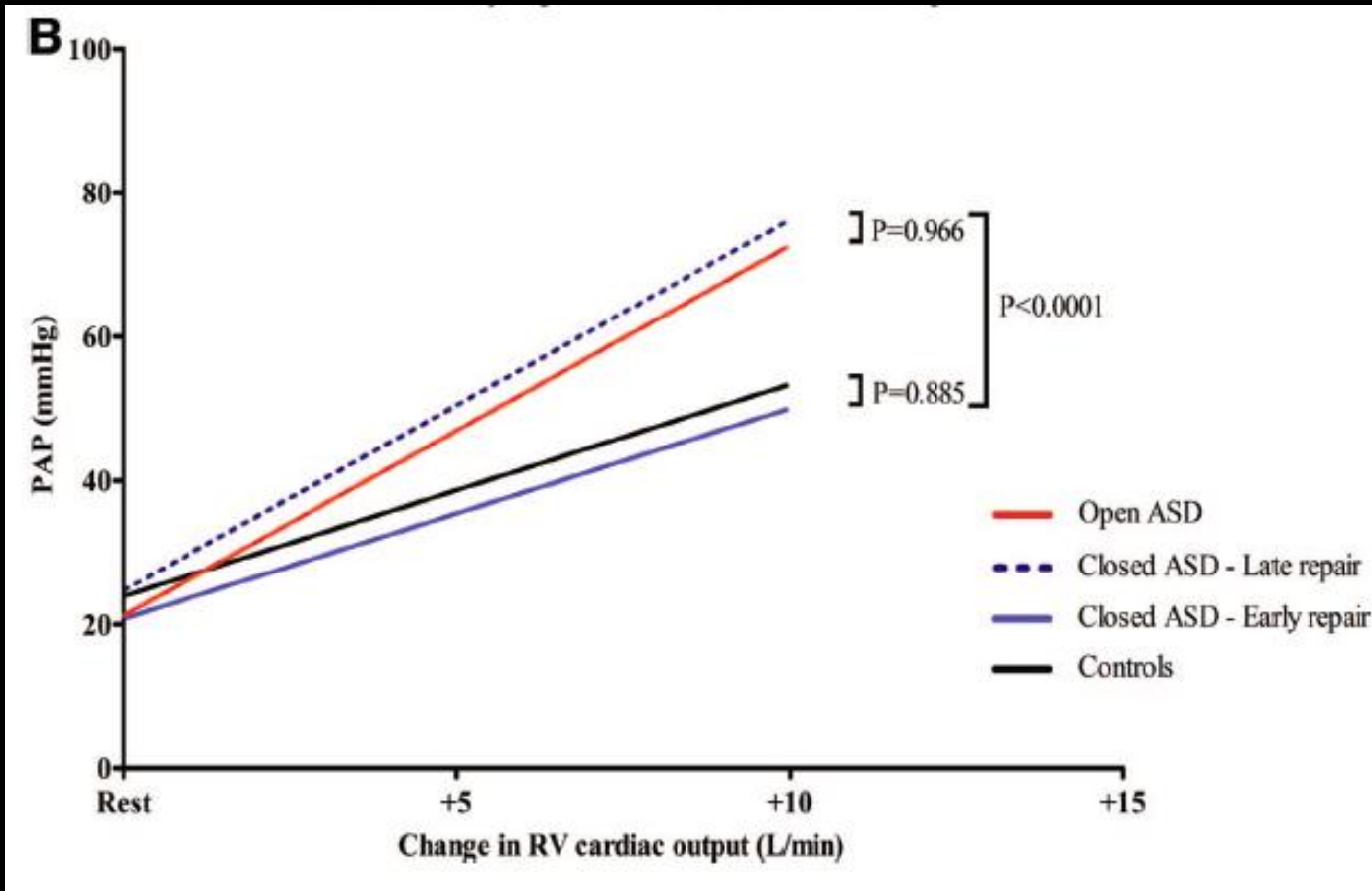


# ASD exercise





# Increased pulmonary vascular resistance in unrepaired ASD's



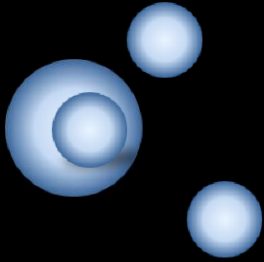
# Conclusions

- Recognise the limitations of assessing the RV at rest
- Exercise places an important (and possibly disproportionate) load on the RV
- RV/ pulmonary vascular function can limit exercise performance – in disease and health
- ‘RV reserve’ *may* be useful in predicting functional decline and prognosis

Thank you

# How can big bubbles get through a little tube?

**Bubbles break up**



**AV shunts open**

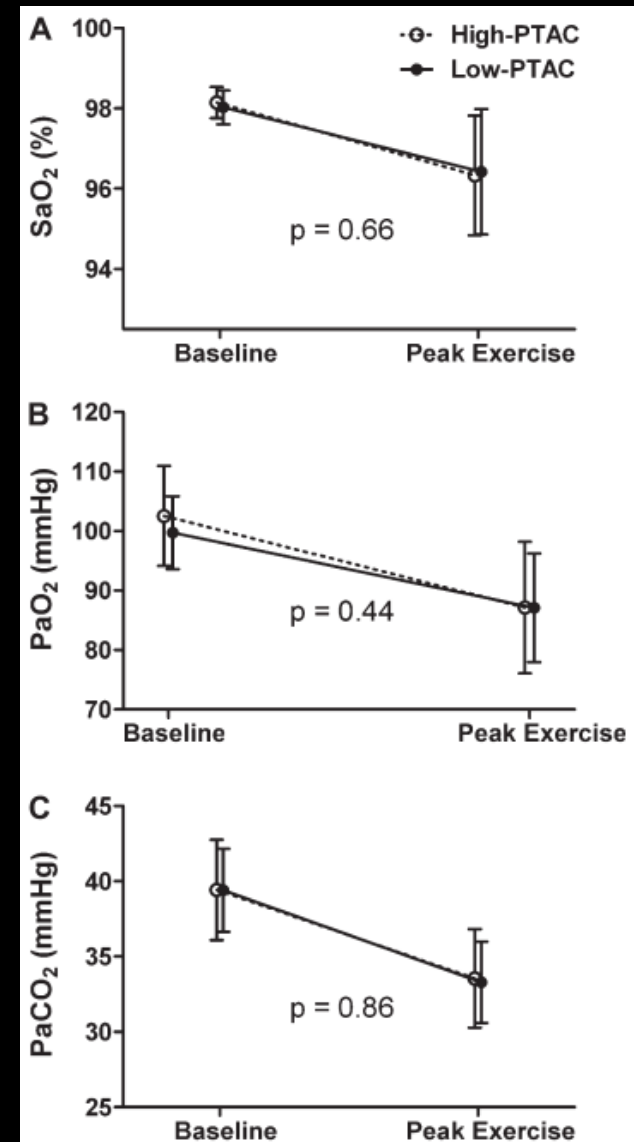


**Pulmonary vessels distend**



# AV Shunts ??

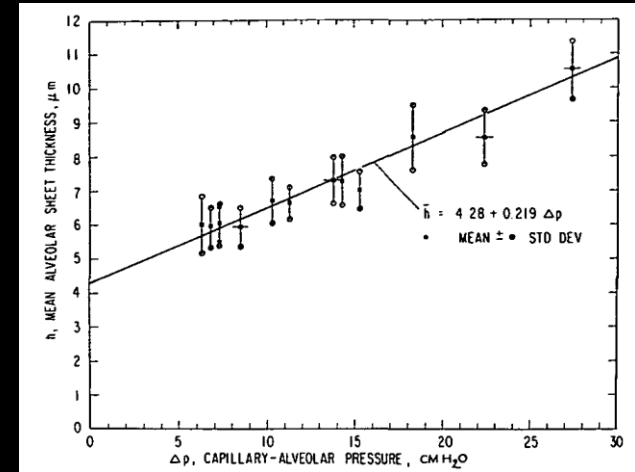
- $\text{SaO}_2$  and  $\text{PaO}_2$  decreased in athletes but there was *no* relationship between exercise induced hypoxemia and PTAC
- PTAC is unlikely to represent shunting (alone)



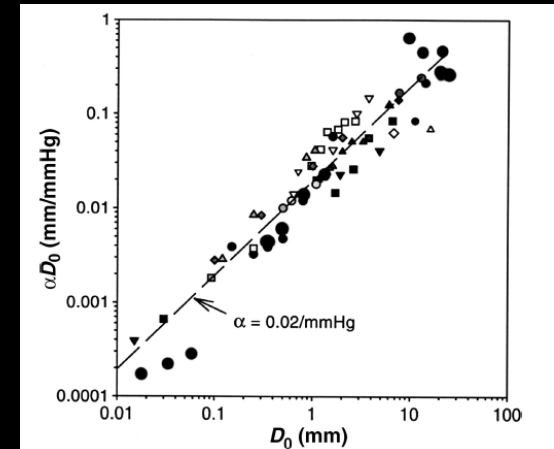


# Distensibility ??

- Pulmonary capillary distensibility has been demonstrated in rapid freeze animal models (Sobin 1972, Glazier 1969)
- Reeves compared a theoretical schema with human data and determined that:
  - pulmonary vessels distend 2% for every 1mmHg increase in mPASP



Sobin et al. *Circ Res* 1972



Reeves et al. *Am J Physiol Lung Cell Mol Physiol* 2005



# Increased afterload

- Pulmonary hypertension, pulmonary arterial hypertension etc.
- Need to consider both deformation and load

