Right Ventricular Function in Chronic Tricuspid Regurgitation

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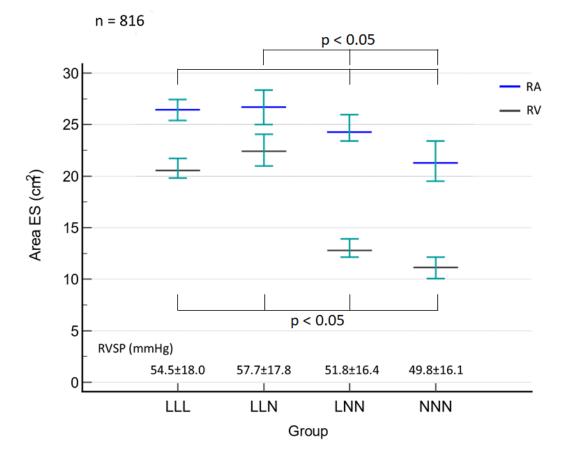
Beijing China



Conflict of Interest: None



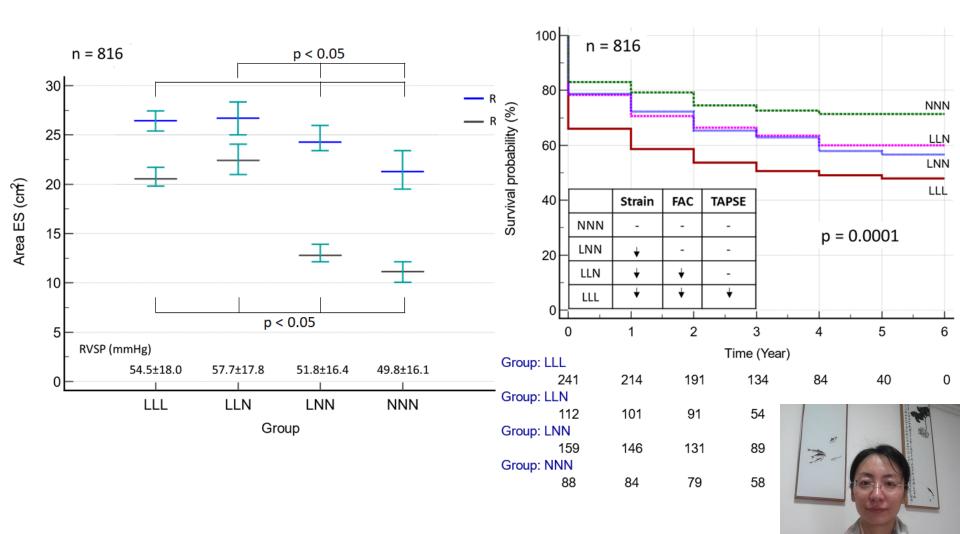
RV Free Wall Strain in TR



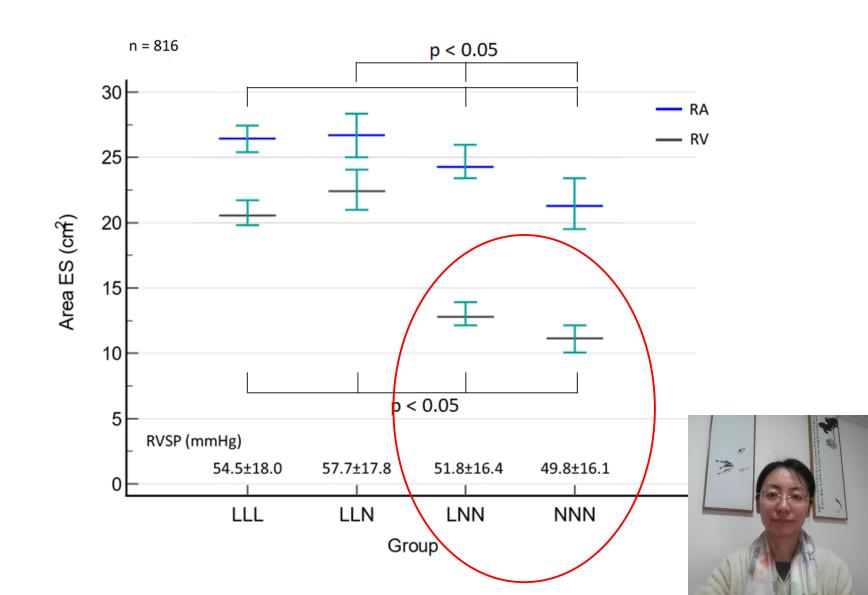
	Strain	FAC	TAPSE
NNN	-	-	-
LNN	¥	-	-
LLN	¥	¥	-
LLL	¥	¥	¥



RV Free Wall Strain in TR



RV Free Wall Strain in TR



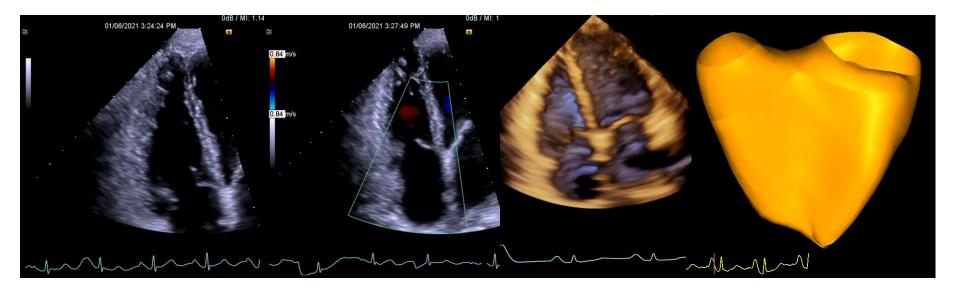
LNN– TR



RA Area = 30.3 cm^2 **RV Area = 16.1 \text{ cm}^2** RV Basal D = 42 mmTAPSE = 19 mmFAC = 46.6%FWS = 12.6% PISA EROA = 0.86 cm^2 VC = 8 mm **EDV/EDVi = 220 ml/110 ml m²** RVEF = 43%



NNN- TR



RA Area = 19.0 cm^2 **RV Area = 16.1 \text{ cm}^2** RV Basal D = 31 mmTAPSE = 23 mmFAC = 59.2%FWS = 28% PISA EROA = 0.38 cm^2 VC = 8 mm **EDV/EDVi = 92 ml/63 ml m²** EF = 61%



So, does normal Strain, FAC and TAPSE (NNN) in a normal-sized RV with normal EF indicate a compensated RV? How do we confirm this?

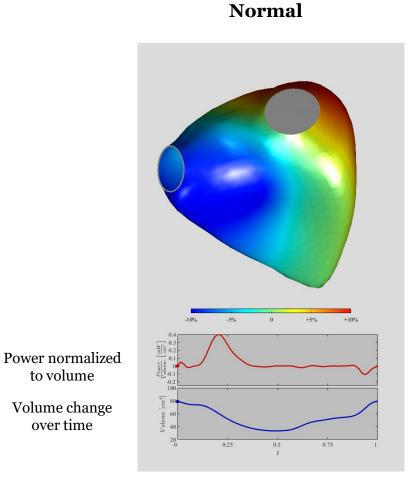


One non-invasive approach is to assess the **Hemodynamic power of the RV**

The hemodynamic power or the intra-ventricular pressure gradient vector considers the spatial contraction pattern (strain) and how it matches with blood flow forces thus providing information on wall stress that is incremental to strain and volume measurements.



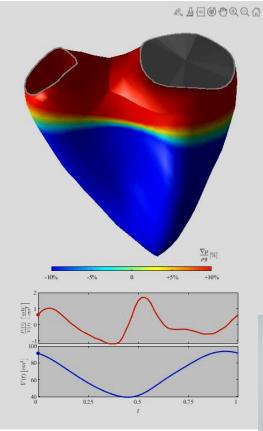
RV Hemodynamic Force



to volume

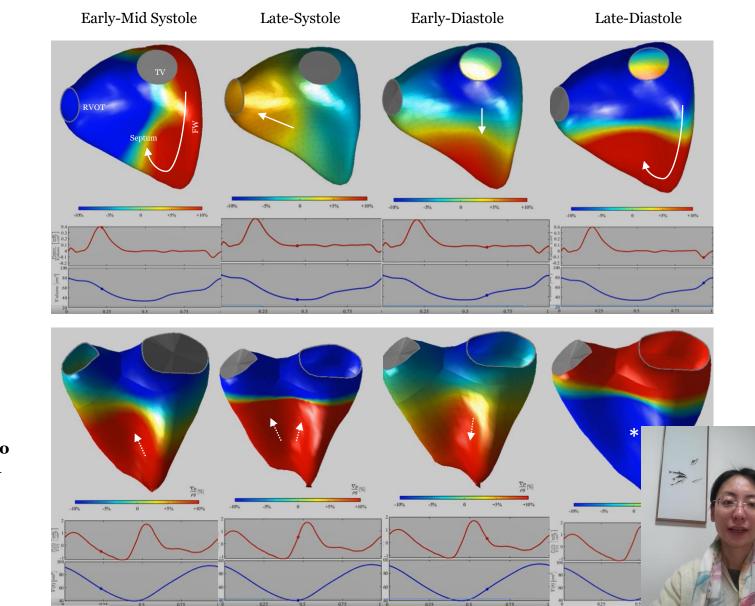
over time

Moderate to Severe TR with NNN





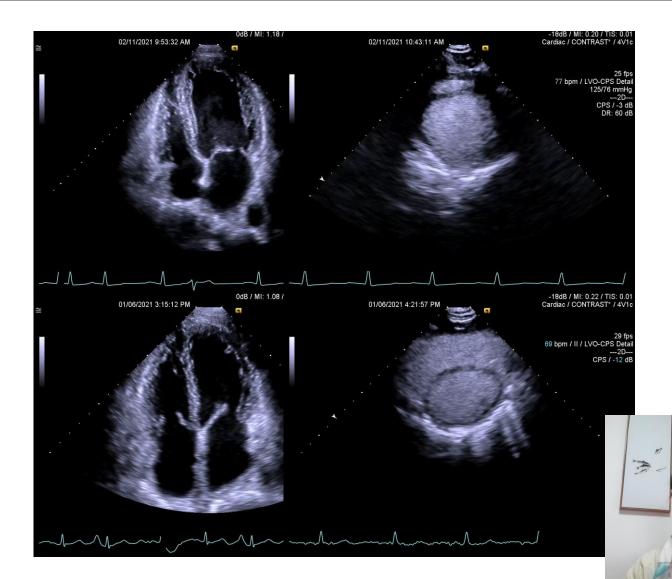
RV Hemodynamic Power Through Cardiac Cycle



Normal

Moderate to Severe TR with NNN

Septal Shape and RV Function



Normal

Moderate to Severe TR with NNN

Summary

Hemodynamic power which integrates direction of intraventricular pressure gradient developed by direction of blood flow, and the resulting wall stress may be an early marker of subclinical RV dysfunction, even before strain becomes abnormal.

This needs further studies.



Thank You

