Mechanisms of Myocardial Ischemia Inducing Sudden Cardiac Death in Anomalous Coronary Origin from the Opposite Sinus: Insights from a Computational Fluid Dynamic Study

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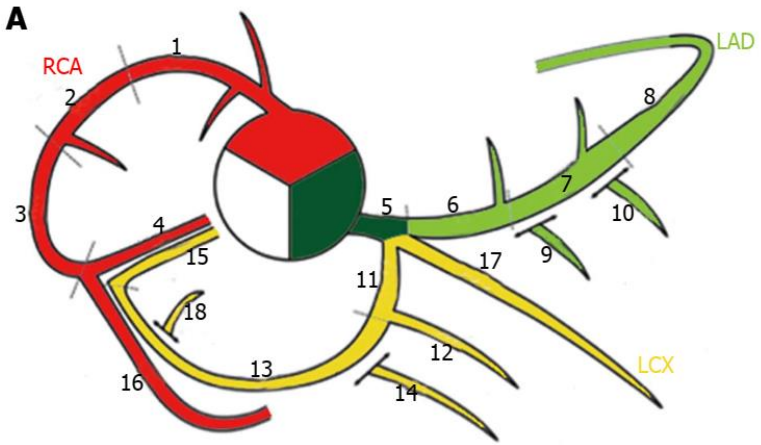
Overview

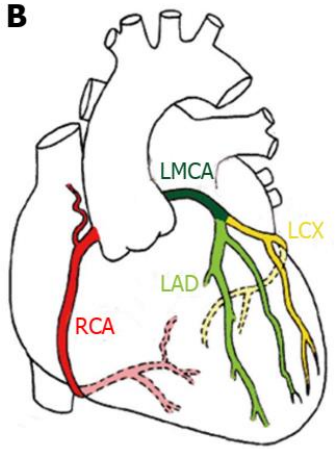
Fall 2010

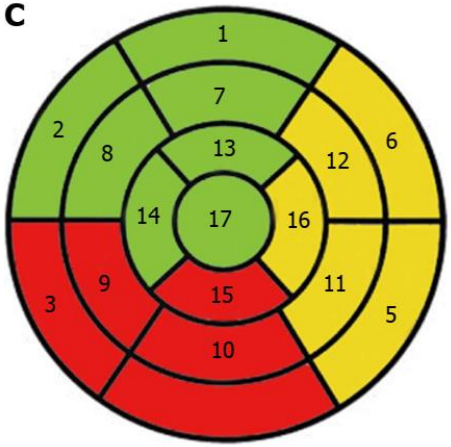
CFD study on effect of branch sizes in human coronary artery

Liza Shrestha
University of Iowa

Normal coronary anatomy







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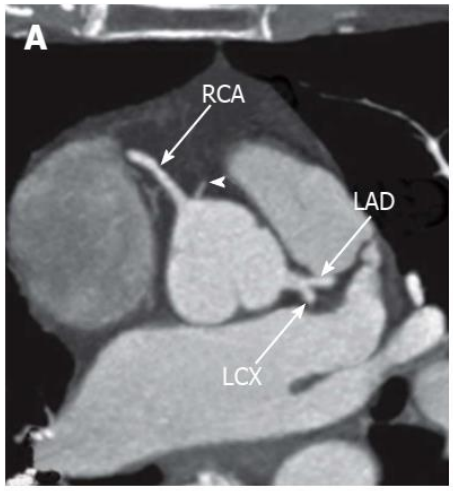
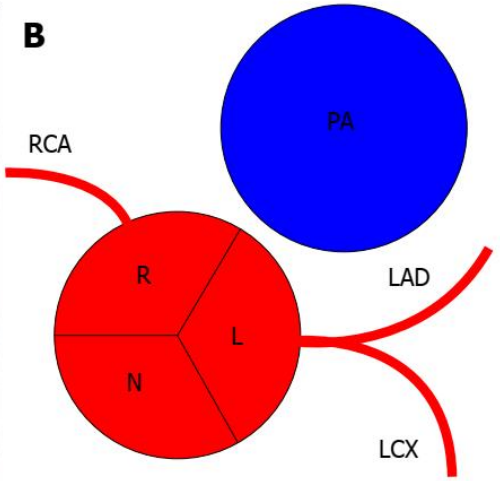
REVIEW

Coronary artery anomalies overview: The normal and the abnormal

Adriana DM Villa, Eva Sammut, Arjun Nair, Ronak Rajani, Rodolfo Bonamini, Amedeo Chiribiri





Coronary artery anomalies overview: The normal and the abnormal

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- According to the literature, CAAs affect around 1% of the general population, ranging from 0.3%-5.6% in studies on patients undergoing coronary angiography, and in approximately 1% of routine autopsy
- The commonest CAA is:
  - a separate origin of the LAD and LCX: 0.41%
  - LCX arising from the RCA: 0.37%
- The anomaly most frequently associated with SCD is the anomalous origin of a coronary artery (AOCA), in particular with a course between the aorta and the PA

Table 4 Classification of coronary artery anomalies based on anatomical features

Coronary artery anomalies			
Of ostium	Ostial atresia		
	valve-like ridge		
Of origin	From PA		RCA from PA
			LMCA from PA
			LAD from PA
			All from PA
			Accessory cor. from PA
	From aorta	SCA	SCA from LSV
			SCA from RSV
		RCA	RCA ectopic from RSV
			RCA from LSV
			RCA from PSV
		LMCA	LMCA from PSV
		LAD	LAD from RCA
			LAD from RSV
		LCX	LCX from RSV
			LCX from RCA
Of anatomy	Duplication		Duplication of RCA
			Duplication of LAD
			Duplication of LCX
Congenital absence	Congenital absence of LMCA		
	Atresia LMCA		
	Congenital absence of LCX		
Hypoplasia	Hypoplasia of RCA and LCX		
Of termination	Fistulae		
	Systemic termination		

Coronary artery anomalies overview: The normal and the abnormal

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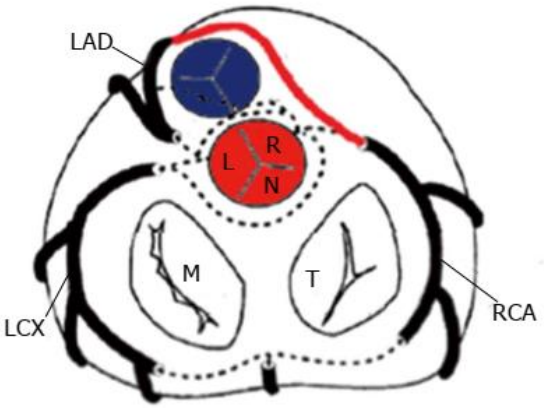
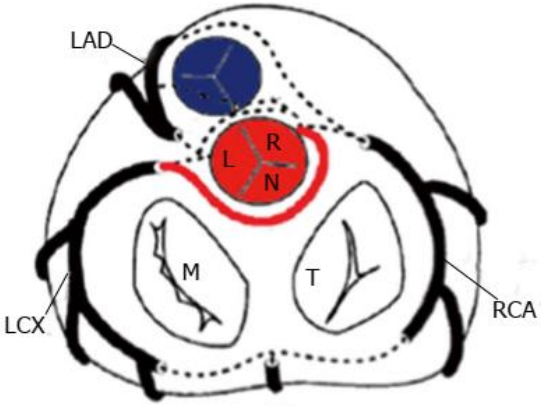
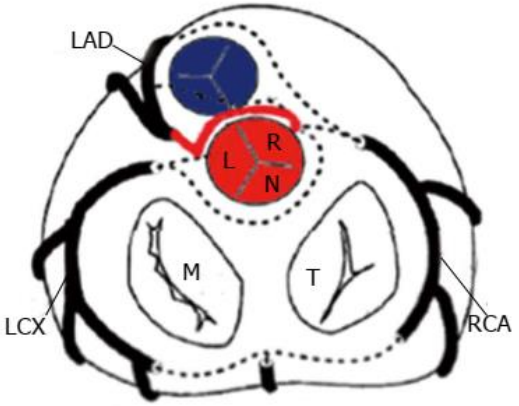


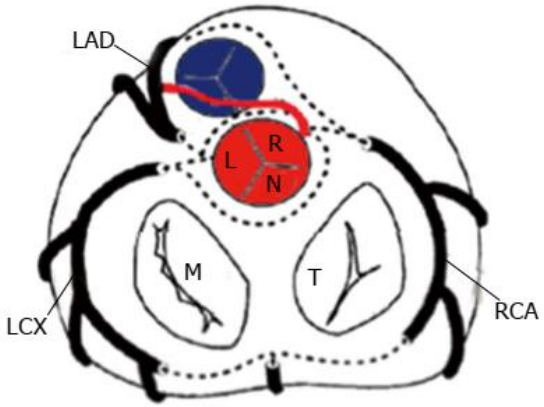
Pre-pulmonic



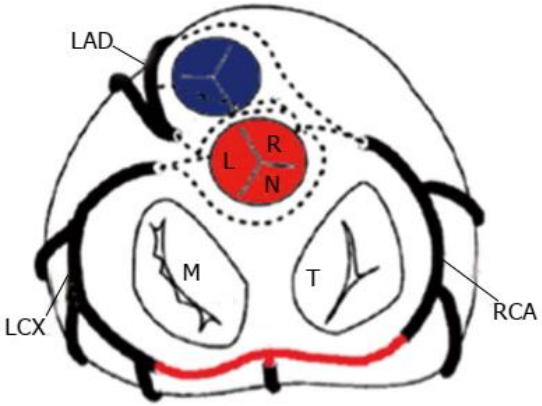
Petro-aortic



Inter-arterial



Trans-septal

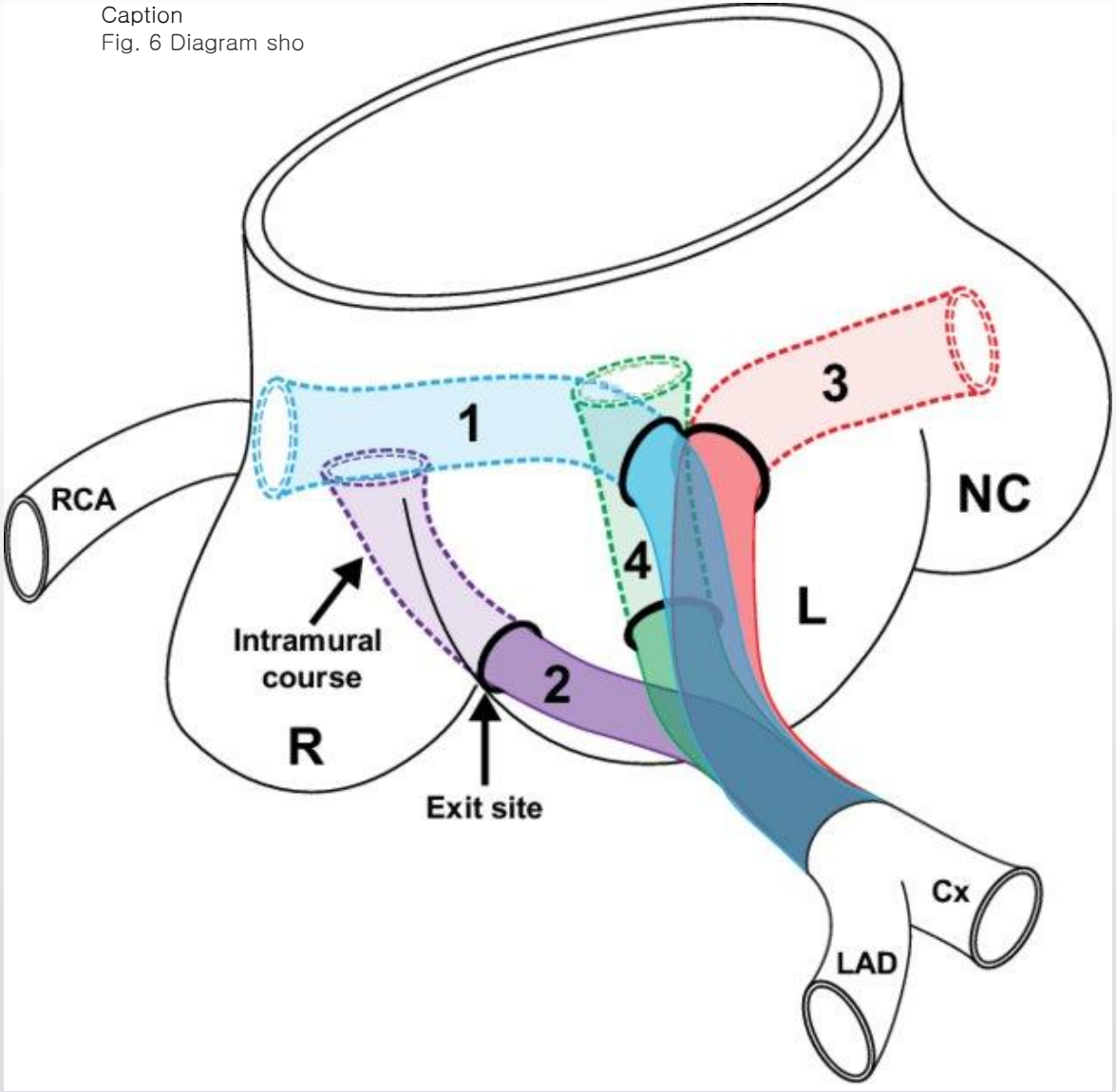


Retro-cardiac

Figure

Caption

Fig. 6 Diagram sho



https://www.researchgate.net/publication/262149870_Sudden_Cardiac_Arrest_at_the_Finish_Line_In_Coronary_Ectopia_the_Cause_of_Ischemia_Is_from_Intramural_Course_Not_Ostial_Location/figures?lo=1

OBJECTIVE

. To investigate the physiology of Left coronary anomalous origin from the opposite sinus (L-ACAOS) with and without intramural course (IM) by using the computational fluid dynamic (CFD) analysis.

Cardiovascular causes of sudden death in 1435 young (<35yo)

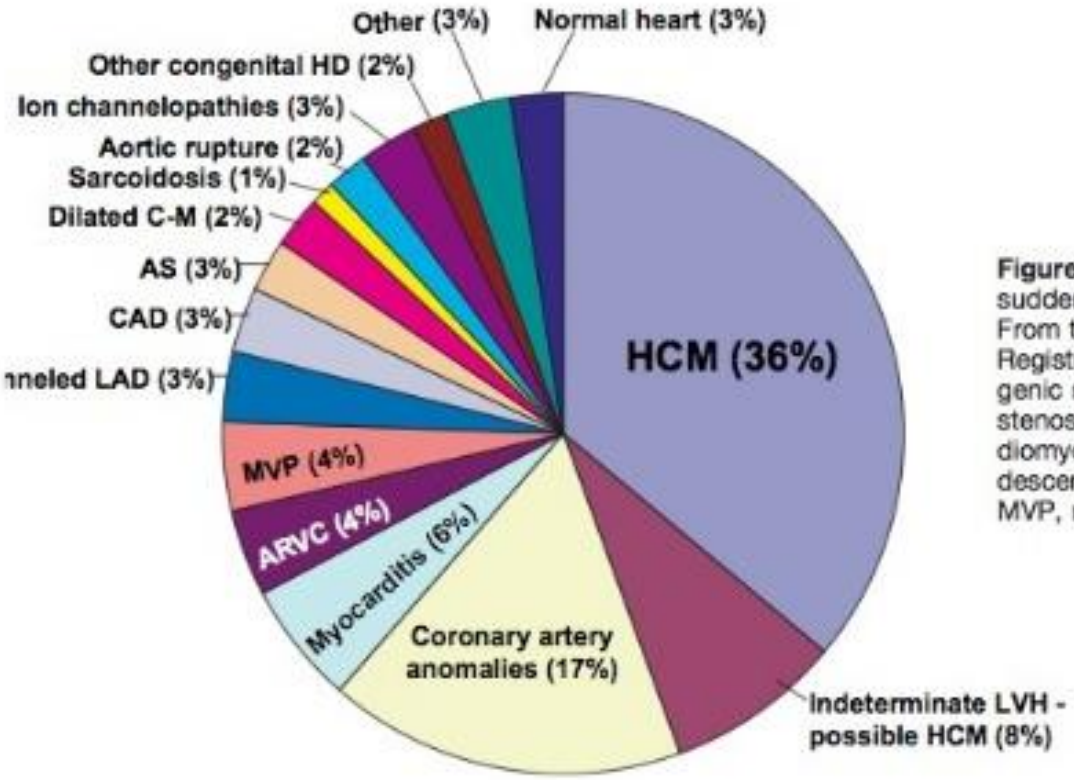


Figure. Distribution of cardiovascular causes of sudden death in 1435 young competitive athlete From the Minneapolis Heart Institute Foundation Registry, 1980 to 2005. ARVC indicates arrhythn genic right ventricular cardiomyopathy; AS, aorti stenosis; CAD, coronary artery disease; C-M, ca diomyopathy; HD, heart disease; LAD, left anteri descending; LVH, left ventricular hypertrophy; ar MVP, mitral valve prolapse.

Maron, Thompson et al Circulation 2007

Sydney Cardiology

BACKGROUND 1

L-ACAOS probably constitutes the most clinically relevant arterial pattern among the wide spectrum of coronary artery anomalies (CAAs), since is being in most sudden cardiac death in young athletes and military recruits.

Rigatelli G, Rigatelli A, Cominato S, Panin S, Nghia NT, Faggian G. A clinical-angiographic risk scoring system for coronary artery anomalies. Asian Cardiovasc Thorac Ann. 2012;20:299-303

	Mean± SD or (%)
Age (years)	55.1 ± 8.2 years
Male gender	10/13 (76.9)
Hypercholesterolemia	3/13 (23.0)
Hypertension	2/13 (15.3)
Diabetes	0/0 (0)
Valvular disease	1/13 (7.6)
Angiographic findings other than CAA:	
Superimposed CAD	2/13 (15.3)
Associated CAD	3/13 (23.0)

Presentation:	
-Atypical chest pain	4/13 (30.7)
-Unstable angina	3/13 (23.0)
-Acute myocardial infarction	1/13 (7.6)
Ventricular tachy-arrhythmias	3/13 (23.0)
Positive Stress test	2/13 (15.3)

Demographic and clinical data of the collected patients used for the modeling and CFD analysis

METHOD 1

The coronary artery circulation with L-ACAOS with and without IM has been segmented and then reconstructed, after reviewing both the angiographic and computed tomography(CT) findings:

- 13 consecutive patients

(10 males, mean age 55.1 ± 8.2 years) with L- ACAOS

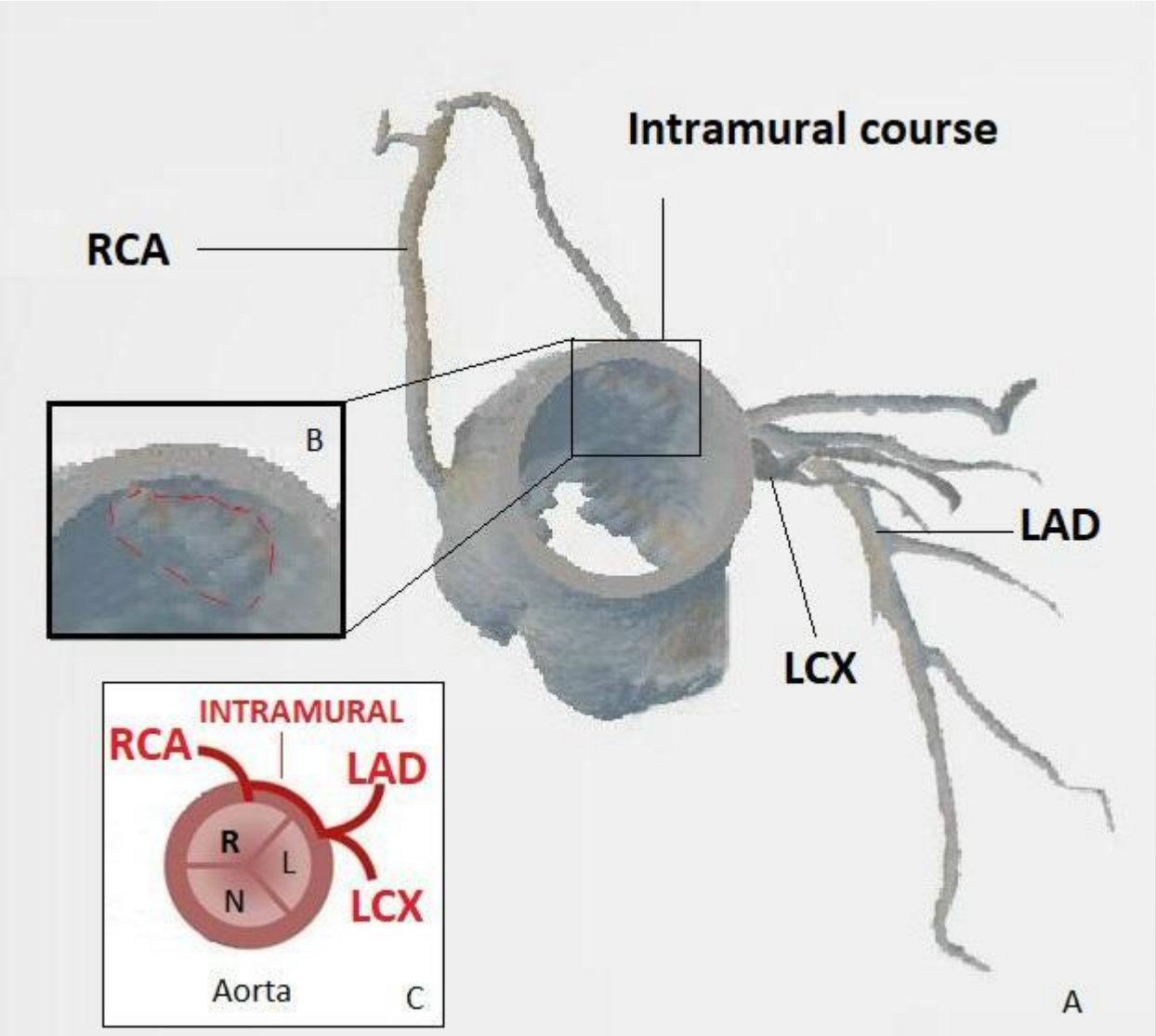
- In our institution
- Between 1st January 2003 and 1st January 2018.

METHOD 2: Model Reconstruction

- The vessels were virtually reconstructed based on the measurements of vessel size and length obtained by quantitative coronary angiography (QCA, GE Innova 2100 IQ, General Electric, USA) and coronary computed tomography (GE Light Speed 64 slices, General Electric, USA) analyses.
- Both the coronary circulation and the proximal segment of the aorta were reconstructed through manual segmentation using the software OsiriX (OsiriX Foundation, Geneva, Switzerland) as a plugin.

METHOD 3: Model Reconstruction

- Specifically, the user interface allowed setting various parameters (size of the rectangular box, resolution levels, and regularization parameters).
- The model was subsequently optimized using the Rhinoceros v. 4.0 Evaluation software (McNeel & Associates, Indianapolis, IN), as previously described [9]



(A) 3D-model reconstruction; (B) intramural course of left coronary artery; (C) Schematic representation of an ACAOS with an intramural course

METHODS 4

Blood was modelled as an incompressible non Newtonian fluid

All the simulations were carried out using ANSYS CFX 18.0.

METHODS 5

The velocity profiles of the blood flow were modelled in order to simulate the rheologic conditions of a hemodynamically stable patients.
Only the diastolic phase, with a mean blood pressure of 80 mmHg (10665 Pa) was considered.

METHODS 6

Vorticity magnitude, static ressure and wall shear stress (WSS) were analysed in a model of L-ACAOS with no IM course and in L-ACAOS-IM at rest and during exercise.

RESULTS

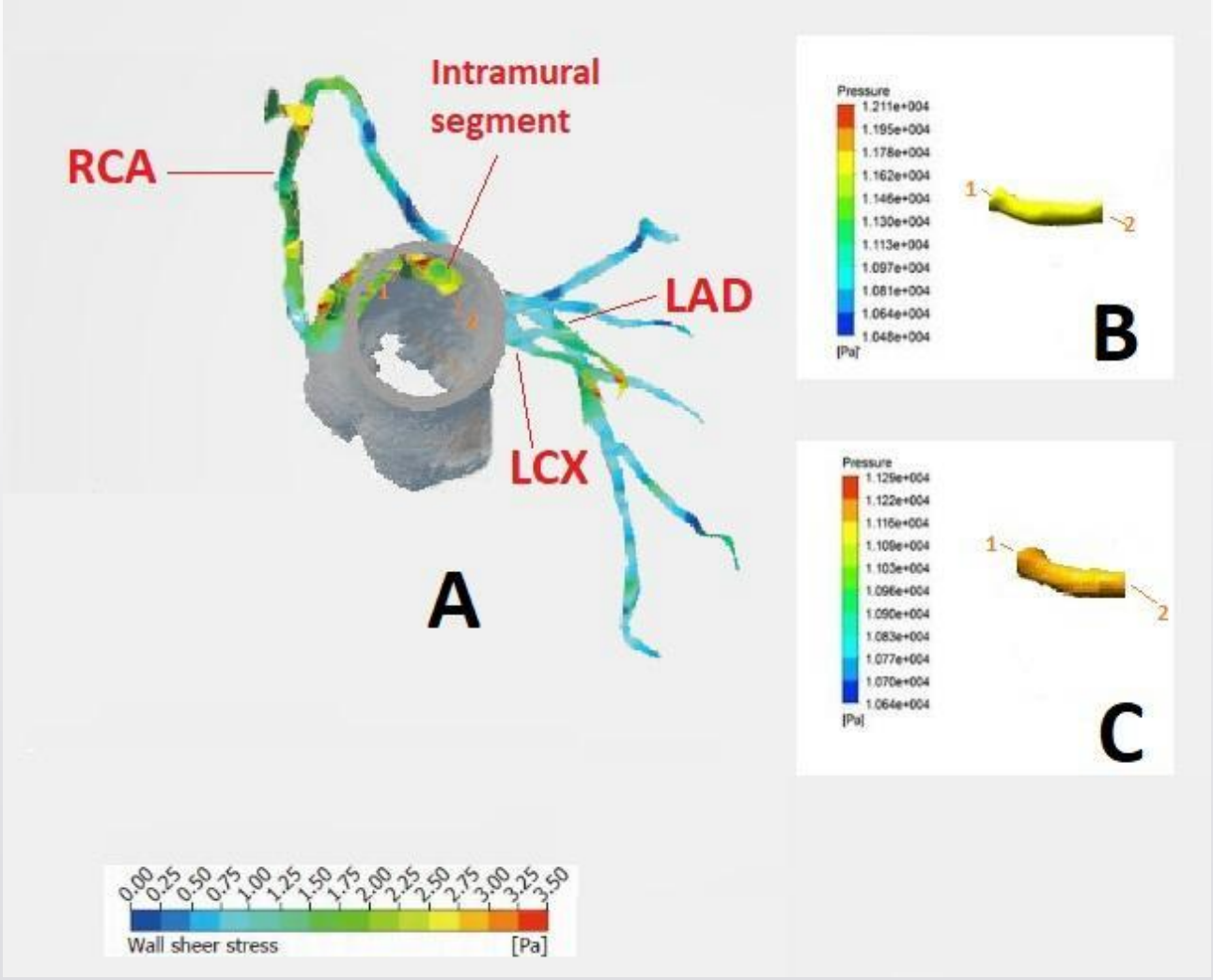
Among the consecutive 13 patients collected for building up the 3D model reconstruction, 7 have been classified as L-ACAOS-RA with interatrial course and 6 as L-ACAOS-IM (table 1).

By QCA, the mean diameter of proximal left main (LM), left anterior descending artery (LAD), and left circumflex (LCX) were 4.32 ±0.82 mm, 3.21±0.65 mm, and 2.52±0.88 mm

•The mean LAD-LCX bifurcation angle, measured after the diagnostic angiography using an electronic goniometer was 52.6±10.4 ° whereas the mean length of the LM was 18.5±1.7 mm.

•The mean length of the IM course in L-ACAOS-IM by angio-CT was 8.2±1.1 mm

RESULTS 1



The mean vorticity magnitude and WSS significantly increased from rest to exercise in both models, in right coronary artery, left anterior descending and left circumflex coronary arteries.

RESULTS

Table 3. Computational fluid dynamic analysis of L-ACAOS with IM course at rest and during exercise. *Vorticity magnitude Intramural segment basal vs exercise p<0.001, **Mean WSS intramural segment basal vs exercise, p<0.001,

	RCA	LAD	LCX	IM segment	RCA	LAD	LCX	IM segment
			Basal				**Exercise**	
MeanVorticity magnitude (1/s)	6160.31	6215.19	6188.54	7012.78*	7871.25	7798.61	7775.12	9019.56*
Mean WSS (Pa)	2.21	1.75	1.79	2.11**	2.62	2.45	2.49	3.02**

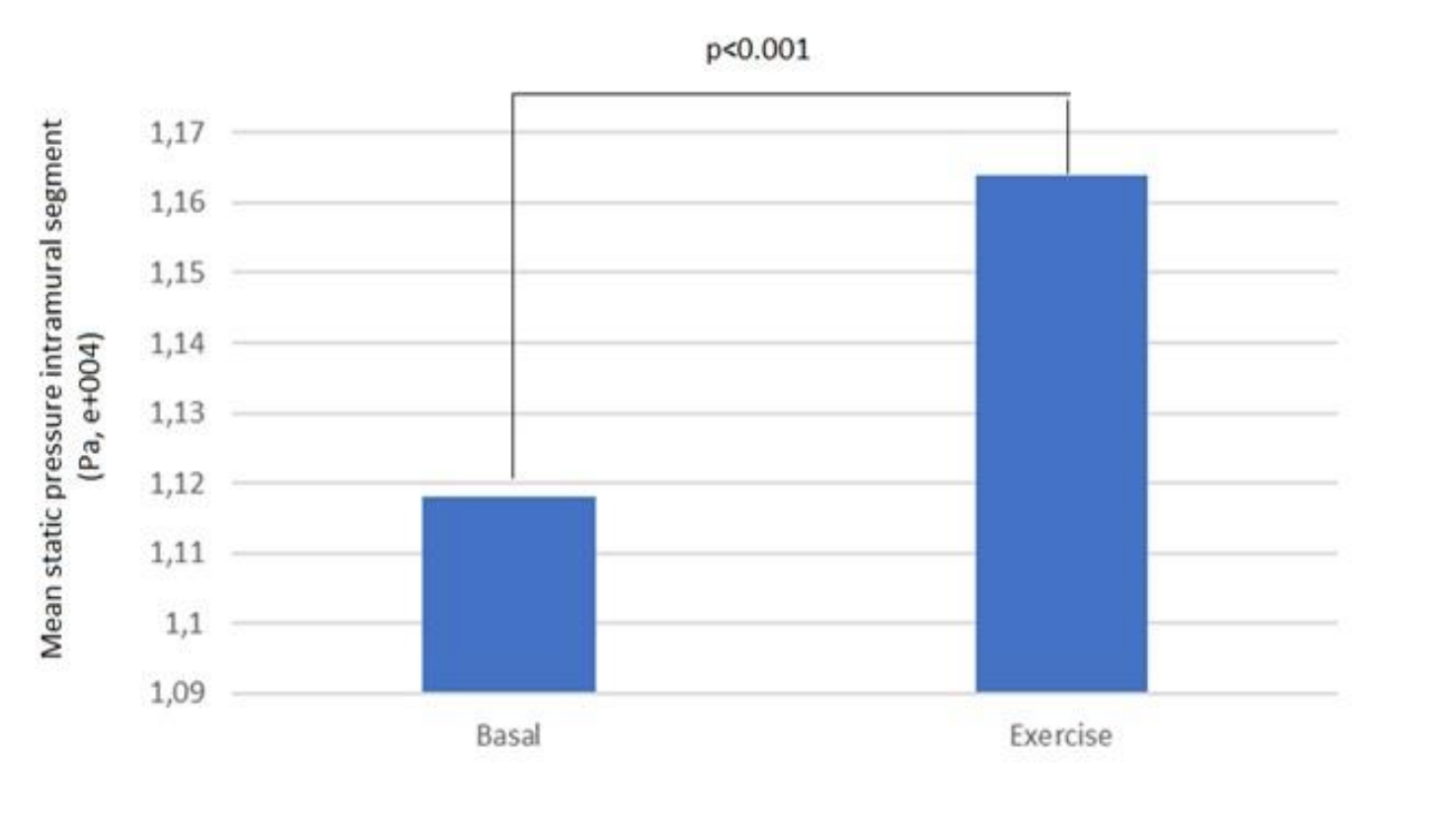
RESULTS

Table 2. Computational fluid dynamic analysis of L-ACAOS with no IM at rest and during simulated exercise. P resulted not significant for all.

	RCA	LAD	LCX	Retroaortic	RCA	LAD	LCX	Retroaortic
	Baseline				Exercise			
Mean Vorticity magnitude (1/s)	6104.23	6005.85	6002.21	6112.36	4109.54	6013.21	6001.32	6118.45
Mean WSS (Pa)	2.18	2.02	2.08	2.25	2.16	2.05	2.03	2.28

RESULTS 2

The mean static pressure significantly increased with exercise in IM (1.118e+004 vs 1.164e+004 Pa, p<0.001)



RESULTS 3

The mean vorticity magnitude: 7012.78 1/s vs 9019.56 1/s, $p<0.001$, $\Delta=2006.78$ 1/s

The mean WSS: 3.02 Pa vs 2.11 Pa, $p<0.001$, $\Delta=0.91$ Pa

This net increment was transmitted to the entire left coronary system in L-ACAOS-IM but not in L-ACAOS with no IM.

CONCLUSIONS

In L-ACAOS, different hemodynamic parameters observed in the intramural segment seems to confirm that that IM is compressed during exercise.

Moreover, these rheological properties also propagated along the left coronary system, predisposing distal coronary segments to a higher risk of spasm and thrombosis.

LIMITATIONS

Our study suffers from a number of limitations.

1. Firstly it considers virtual L-ACAOS model and the vessel has been considered non-compliant with a steady diastolic blood flow in a virtual hemodynamically stable patient

2. Secondly, we investigated in our model only the diastolic phase while the phasic obstruction in L-ACAOS-IM is thought to occur during cardiac systole

3. Finally, we did not evaluate the time averaged WSS, oscillatory index, and the relative residence time, which had a recognized role in the development of arterial atherosclerosis

THANK FOR YOUR LISTENING