Basic Concepts and Interpretation: IVUS Pre-Intervention

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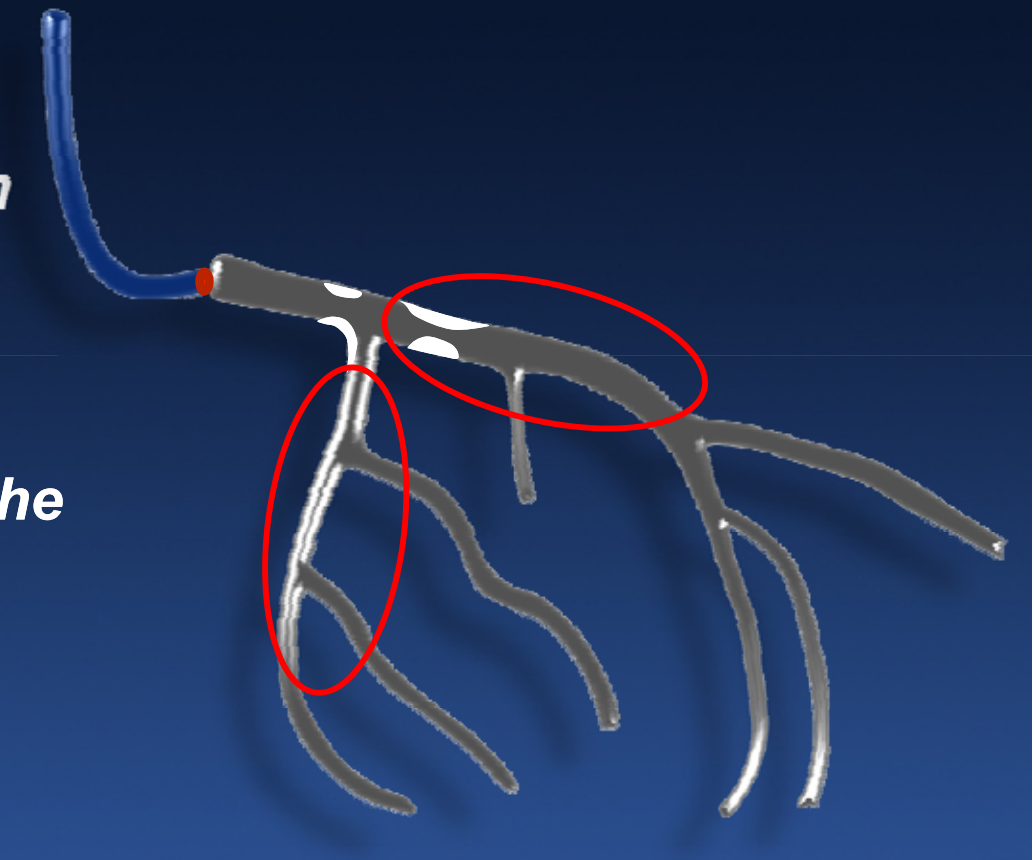
Cardiovascular Research Foundation

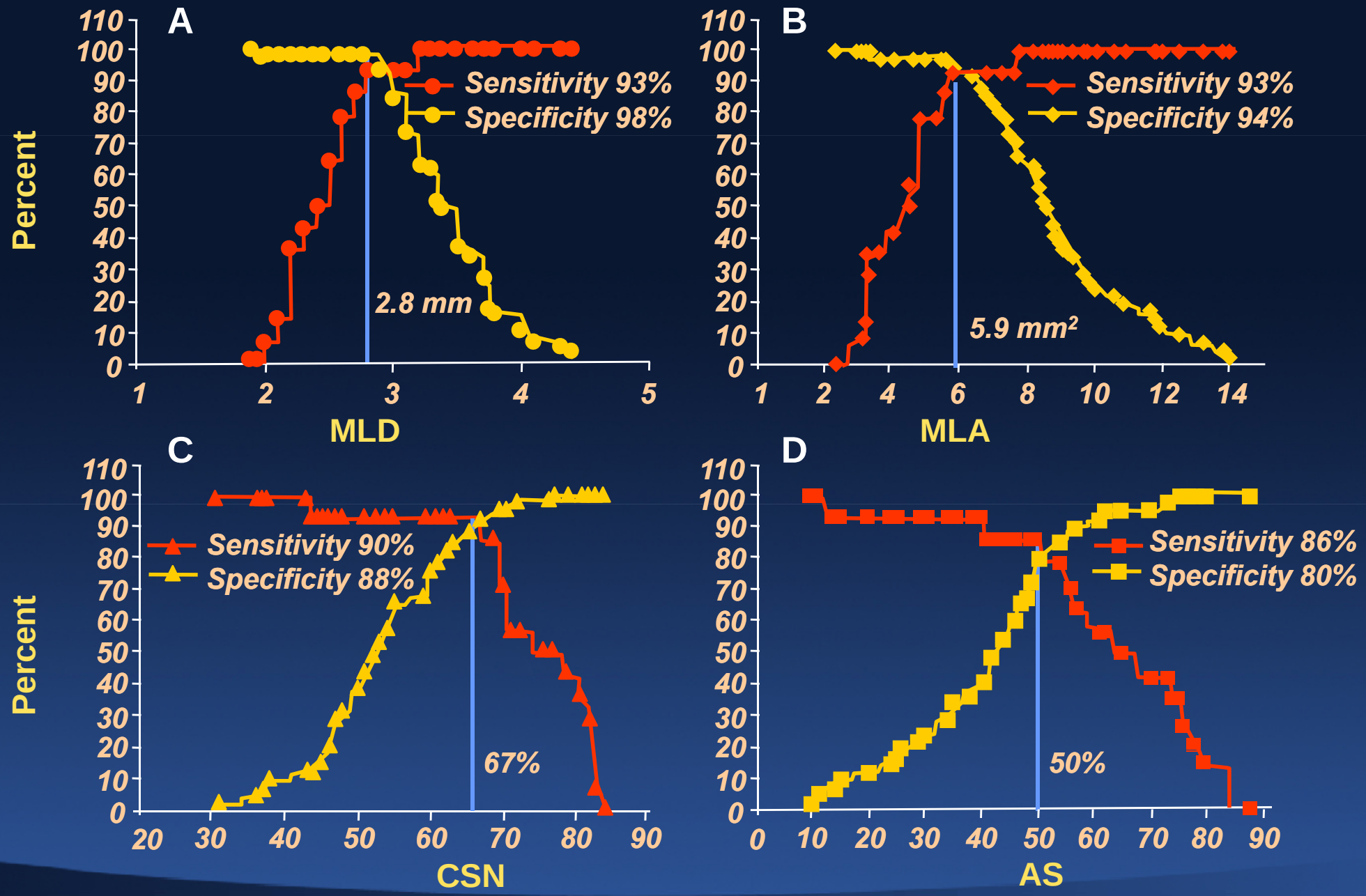
	Abizaid AJC 1998; 82: 423-8	Nishioka JACC 1999; 33: 1870-8	Takagi. Circ. 1999; 100: 250-5	Briguori AJC 2001; 87: 136-41	Takayama CCI 2001;53:48-55	Lee AJC 2010; 105: 1378-84	Kang Circ CV Interv 2011; 4:65-71 (AJC, 2012;109:947-53)	Ahn JACC CV Interv 2011;4:665-71	Ben-Dor Eurointervent 2011;7:225-33 (Cardiovasc Revasc Med 2012;13:177-82)	Yang CCI 2013	Koo JACC CV Interv 2011;4:803-11	Waksman JACC 2013;61:917-23	Gonzalo JACC 2012;59:1080-9	Stone TCT2012 (VERDICT - FIRST)	Chen Int J Cardiol 2012; in press	Kwan Chin Med J 2012;125:4249-53
	CFR	SPECT	FFR	FFR	FFR	FFR	FFR	SPECT	FFR	FFR	FFR	FFR	FFR	FFR	FFR	FFR
N	112	70	51	53	14	94	236	170	205	206 LAD	267	367	47	544	323	169 LAD
% abnormal	40%	65%	49%	23%	50%	40%	21%	26%	26%	44%	33%	28%	46%	31%	54%	59%
IVUS																
Ref lumen (mm^2)	8.3 7.4	11.9 10.6	9.3	7.8	10.3	5.5 5.9	7.6		8.6		7.8 6.7		7.0			
MLA (mm^2)	4.4	4.3	3.9	3.9	3.5	2.3	2.6	2.1	3.5	3.1	3.0	3.5	2.6	3.3	2.9	3.0
MLA Cut-off (mm^2)	***4.0***	***4.0***	***3.0***	***4.0***	***n/a***	***2.0***	***2.4***	***2.1***	***3.2***	***3.2 2.5***	***2.8***	***3.1***	***2.4***	***2.9***	***3.0***	***3.0***
C-statistic							***0.80***	***0.69***	***0.73***	***0.78***	***0.81***	***0.65***	***0.63***	***0.66***	***0.77***	***0.86***
NPV							***96%***	***91%***					***65%***	***81%***	***76%***	***82%***
PPV							***37%***	***39%***			***47%***		***67%***	***47%***	***73%***	***84%***
Other determinants of ischemia	*LL*				*MLA/ LL*	*PB LL*	*PB LAD*	*PB*	*Vessel size*	*Prox-Mid LL PB*	*Prox-Mid LAD*	*Vessel size PB*		*LAD EEM*	*PB LL LAD*	*PB LL*
QCA																
Length (mm)			14	8.5	17.9	15.1		21.2	11.4	22.7	16.5	15.0	7.1	13.9	18.4	20.6
Ref (mm)			3.0	3.1	2.9	2.7		3.3	3.3	3.3	3.1	2.9	2.6	2.9	3.1	3.4
DS (%)			46	52	53			55	48	54	50	45	51	48	64	65

IVUS vs FFR in LMCA Disease

- *There is more agreement between IVUS and FFR in assessing LMCA than in assessing non-LMCA lesions because of limited variability in*
  - *LMCA length*
  - *LMCA size*
  - *Amount of supplied myocardium*
- *Both have theoretical and practical limitations*
  - *FFR: Significant LAD disease*
  - *IVUS: Need to image from both the LAD and LCX*



IVUS Determinants of LMCA FFR <0.75



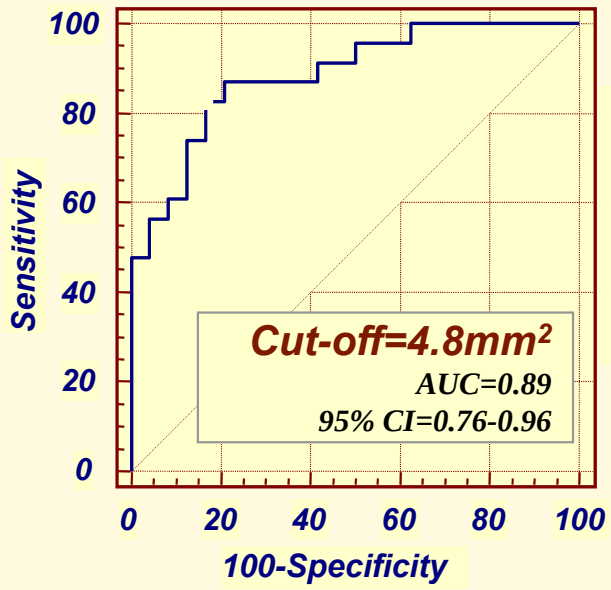
Jasti et al. Circulation 2004;110:2831-6

IVUS determinants of LM FFR (n=47)

Independent predictors for FFR (continuous variable)

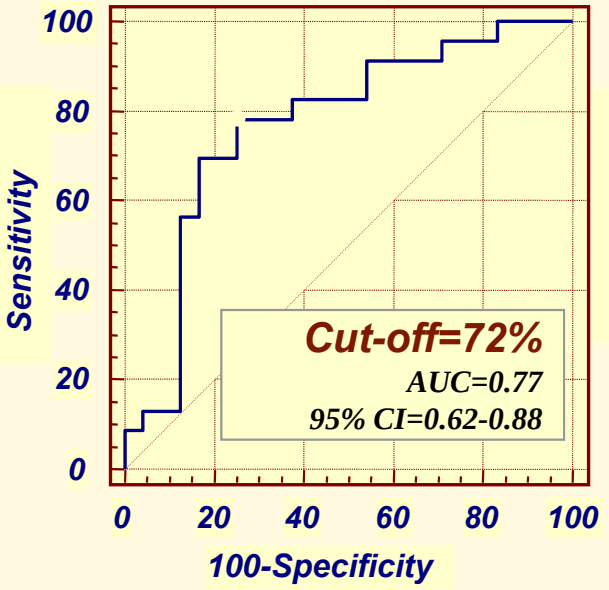
- MLA (β=0.58, 95% CI=0.02-0.04, p<0.001)
- Plaque rupture (β=-0.24, 95% CI= -0.09-0.01, p=0.036)

MLA predicting FFR<0.80



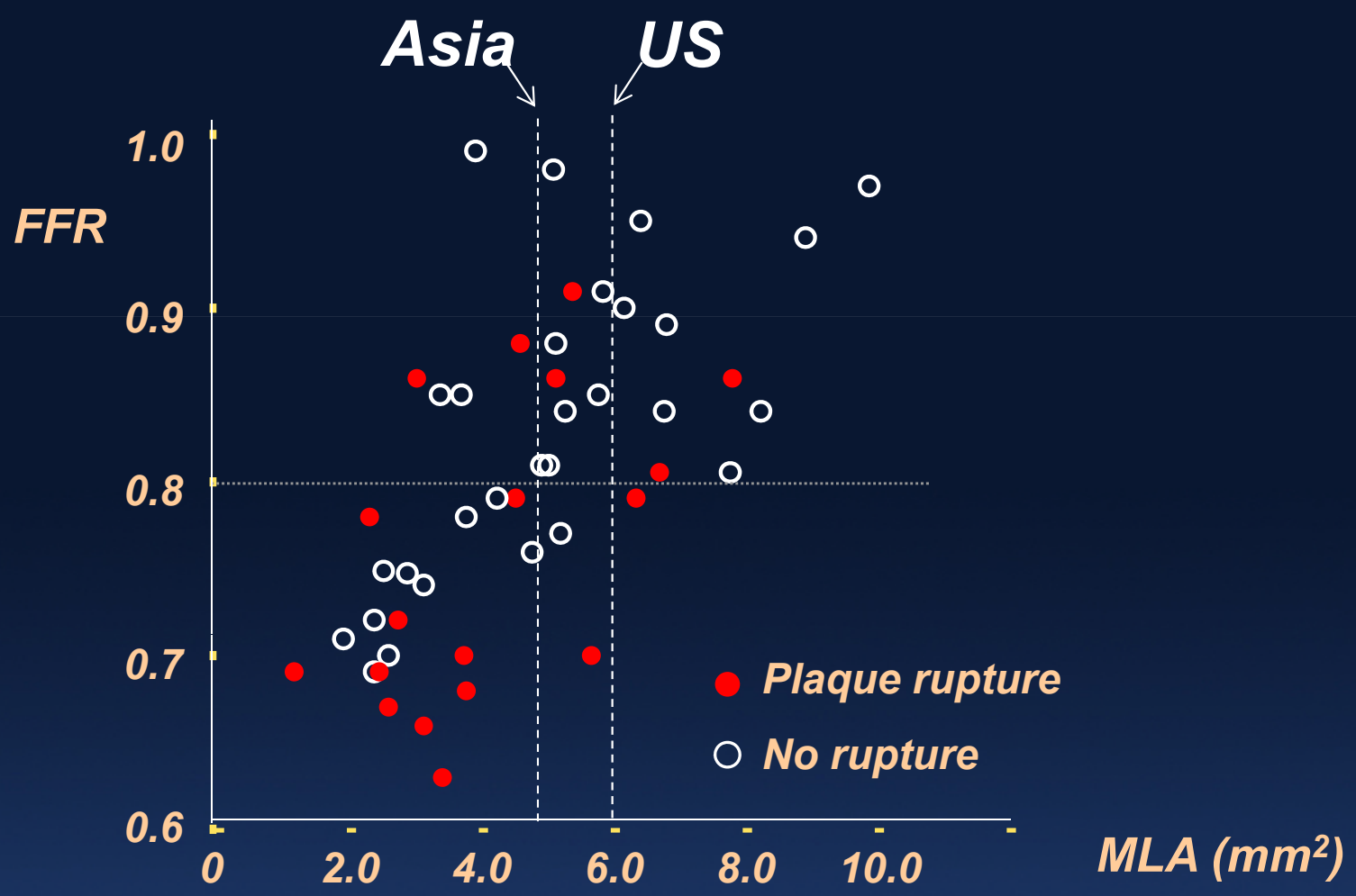
Sensitivity 83%
Specificity 83%
PPV 83%
NPV 83%
Accuracy 83%

PB predicting FFR<0.80



Sensitivity 78%
Specificity 75%
PPV 75%
NPV 78%
Accuracy 77%

Kang et al. JACC Cardiovasc Interv 2011;4:1168-74

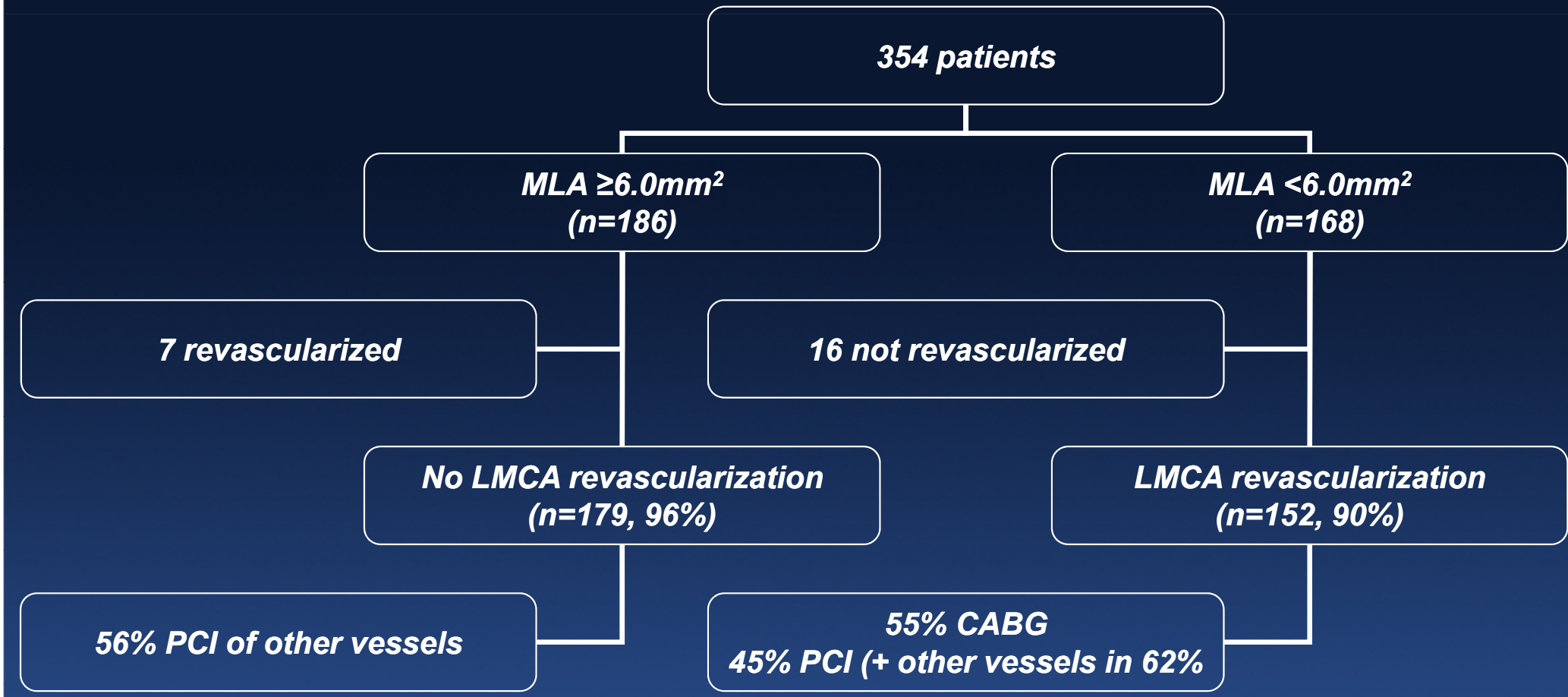


	Men	Women
South Korea	68.6 kg	56.5 kg
US	88.3 kg	74.7 kg

Heart weight correlates directly with body weight (r=0.8-0.9)

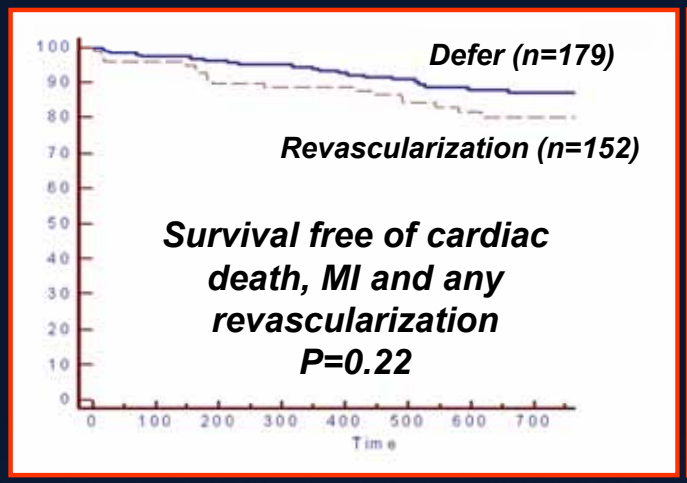
Kang et al. JACC Cardiovasc Interv 2011;4:1168-74
Yoon et al. Korean J Path 1999;33:1-8
Seo et al. J Korean Med Sci 2000;15:641-6

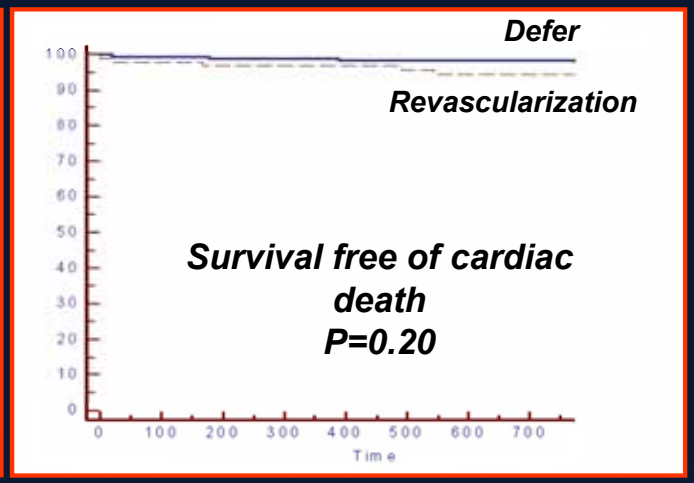
Prospective application of predefined IVUS criteria for revascularization of intermediate LM lesions: Results at 2 years from the LITRO study



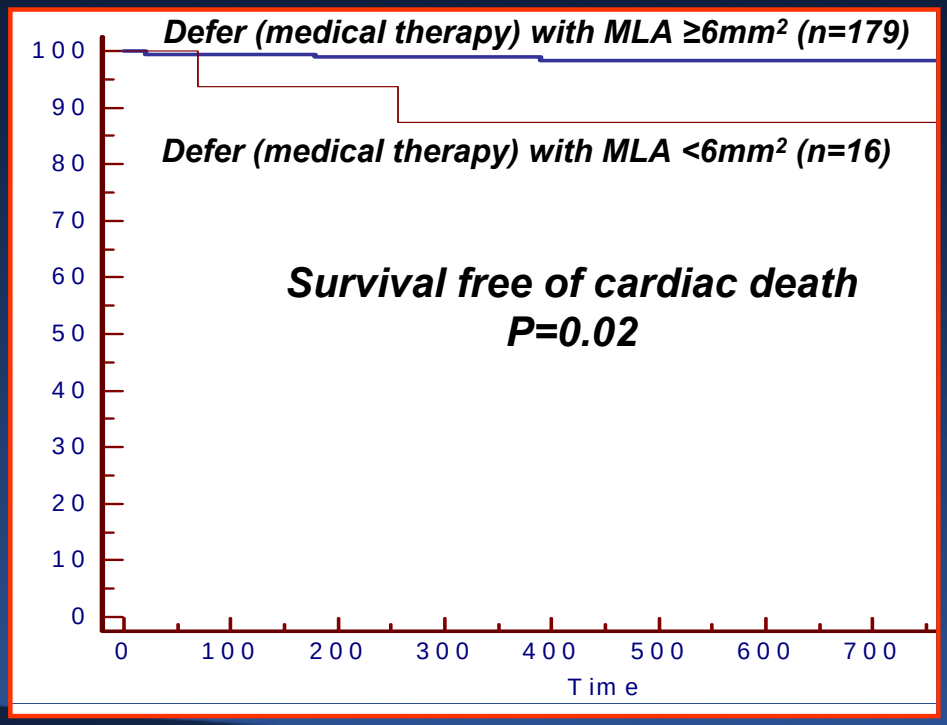
De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

Clinical outcome of pts with vs without revascularization



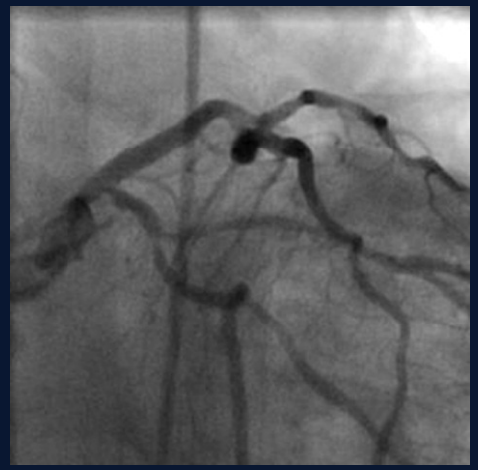


Clinical outcome of pts without revascularization according to the MLA

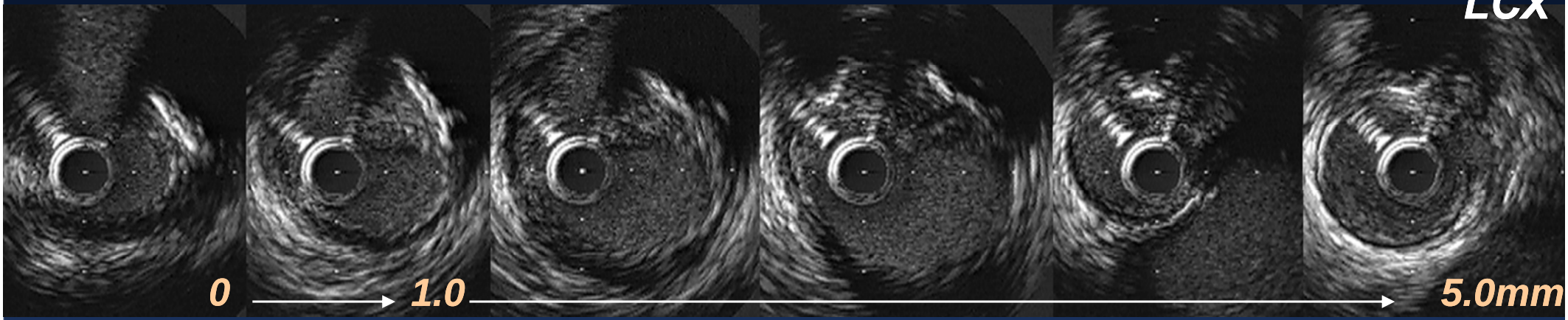


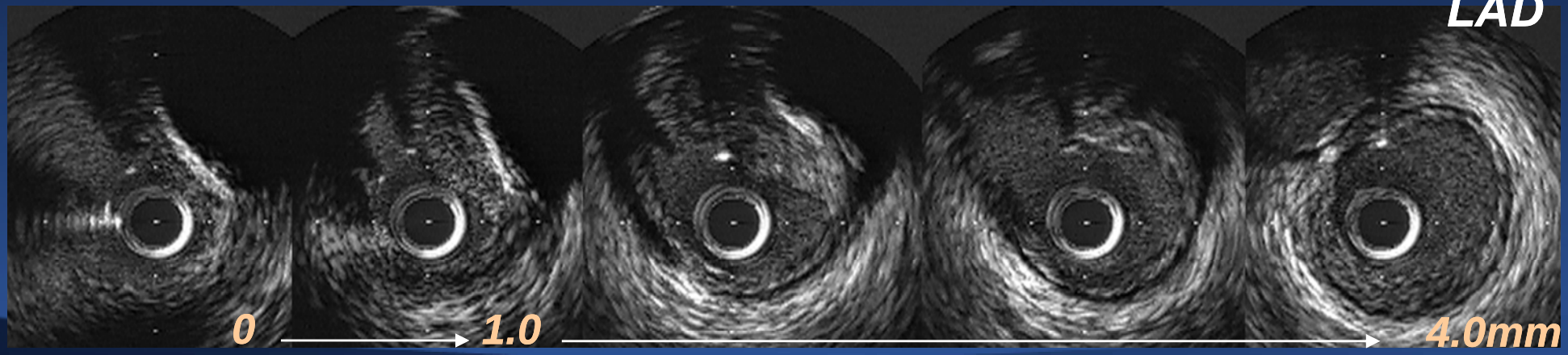
In the group of 16 patients with MLA <6mm² who were treated medically, cardiac death-free survival to 2 years was 86% (97.7% in the deferred group; p=0.04), and survival free of cardiac death, MI, and revascularization was 62.5% (87.3% in the deferred group; p=0.02).

De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

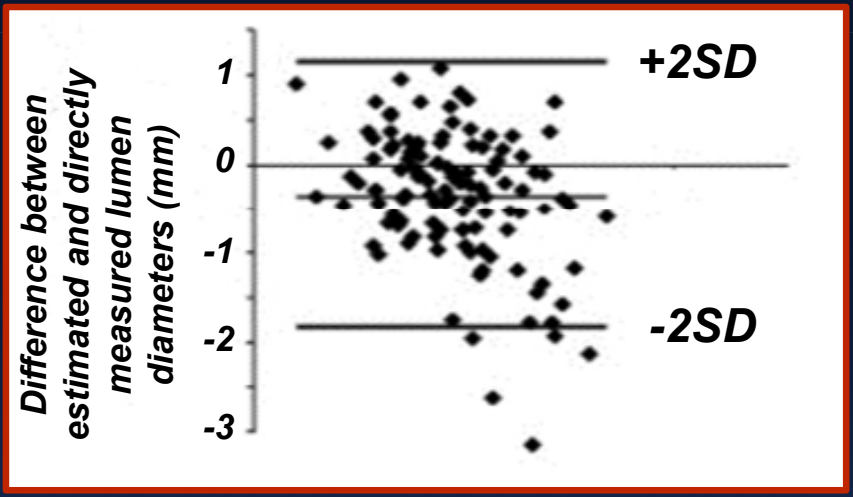


- *In 25% of patients, MLA differs by 1mm² when imaged from a pullback beginning in the LAD vs the LCX.*
- *Since IVUS can artificially increase, but not decrease lumen dimensions, the smallest MLA is always the most accurate*



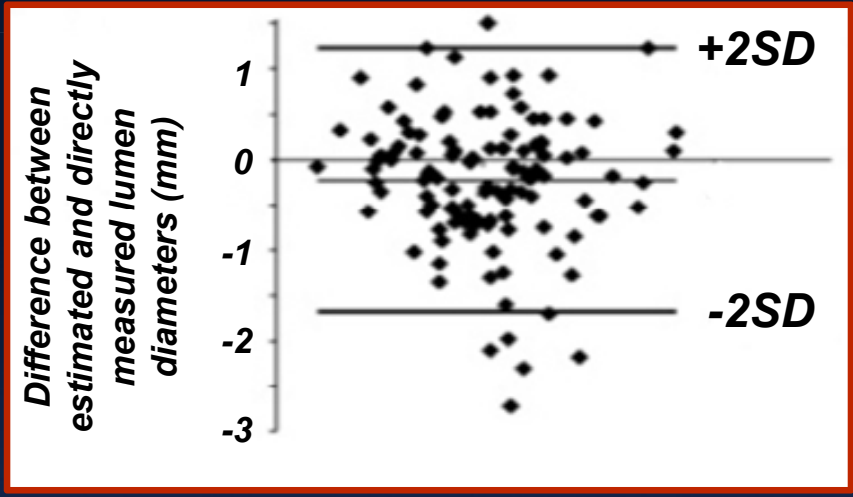


Evaluation of the LAD from the LM-LCX pullback



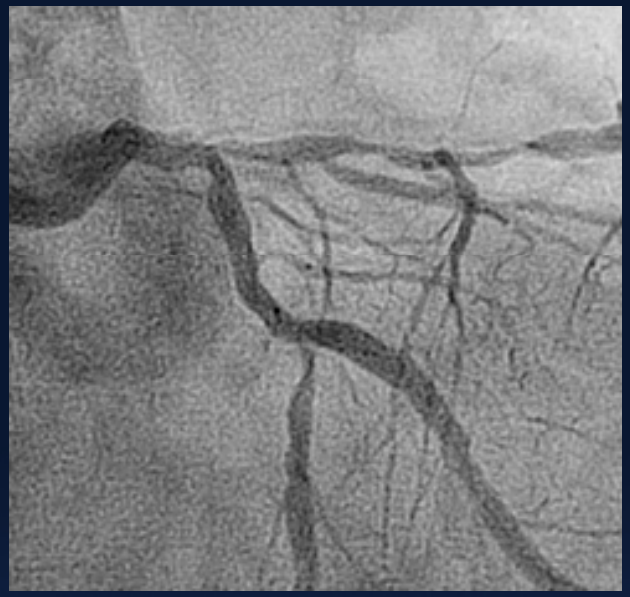
	Sensitivity	Specificity
Plaque burden >40%	59%	45%
Plaque burden >70%	78%	42%

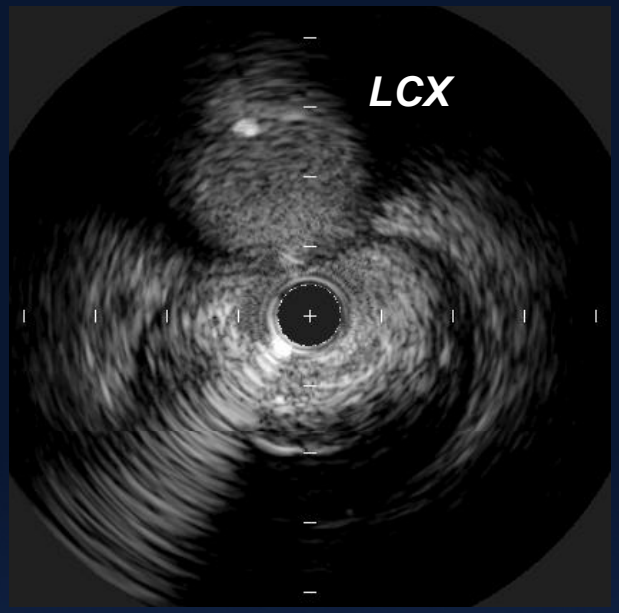
Evaluation of the LCX from the LM-LAD pullback

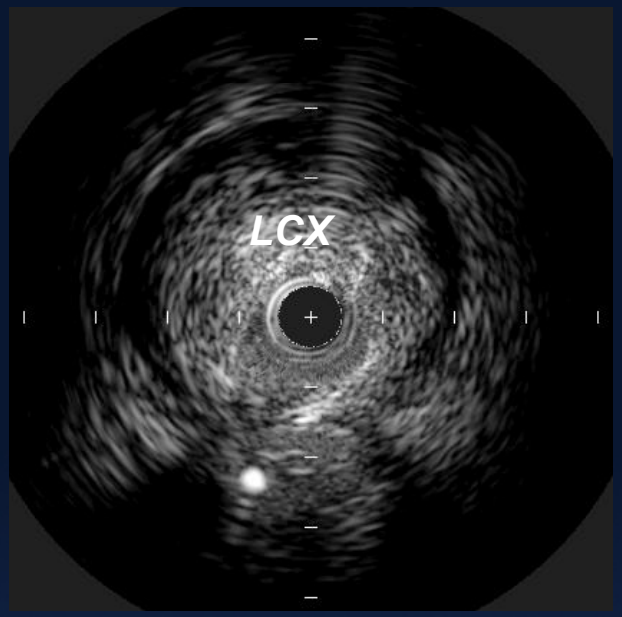


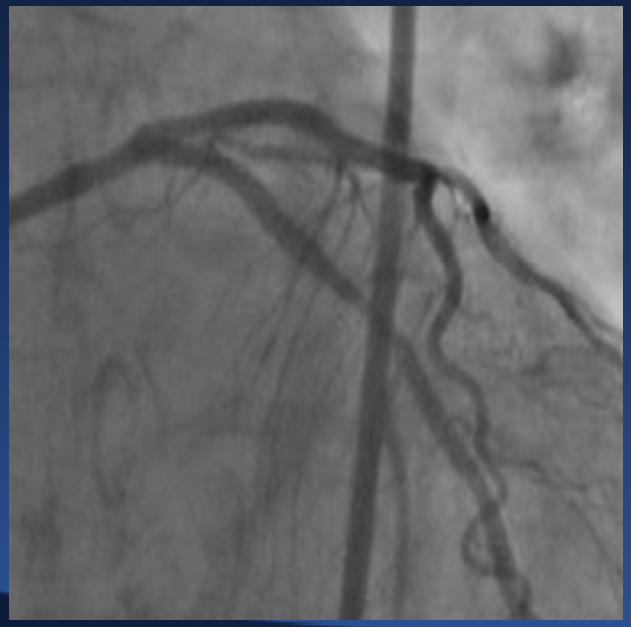
	Sensitivity	Specificity
Plaque burden >40%	67%	55%
Plaque burden >70%	88%	42%

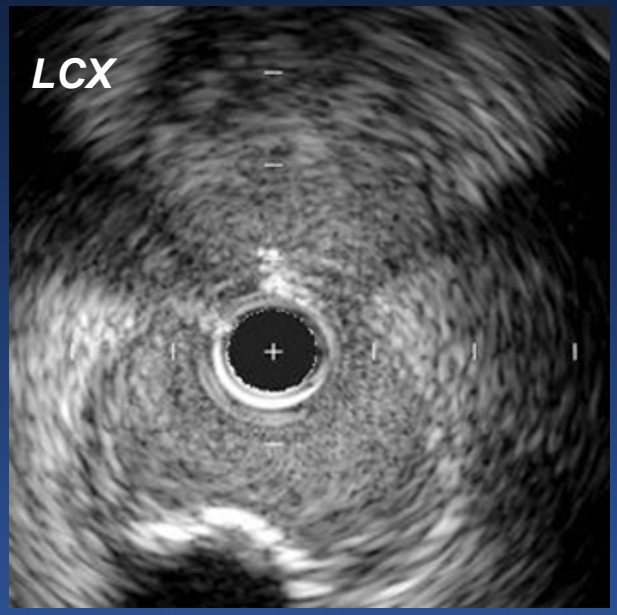
Oviedo et al. Am J Cardiol 2010;105:948-54

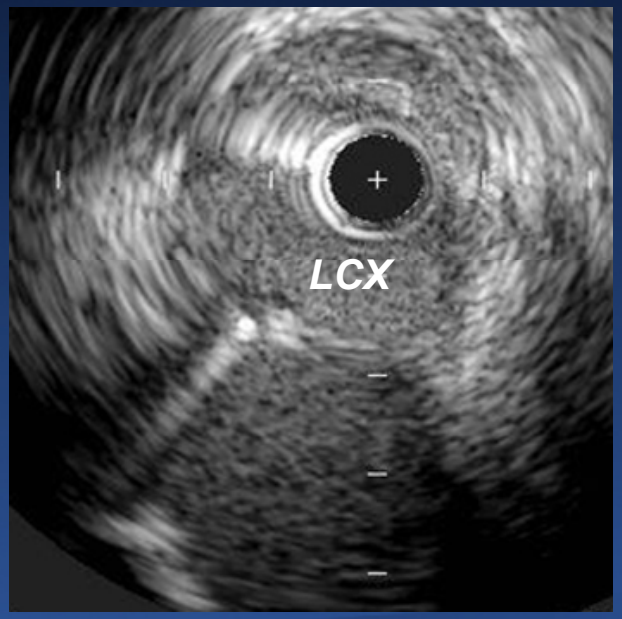



LCX

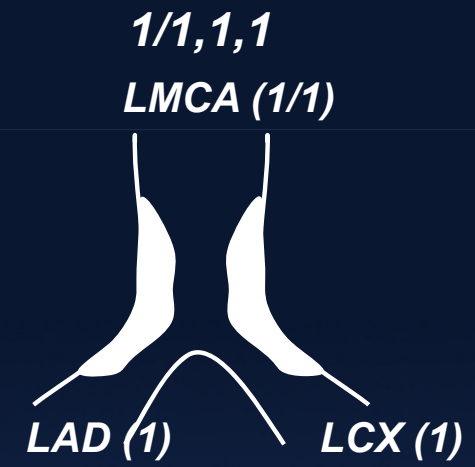

LCX



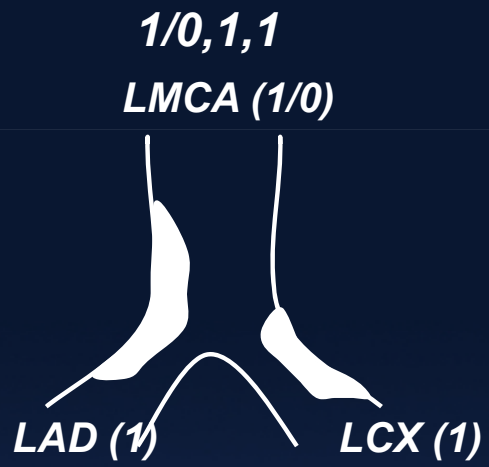

LCX


LCX

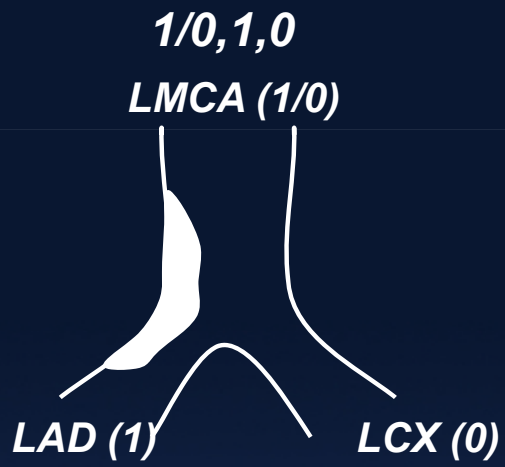
IVUS plaque distribution in 140 distal LMCA bifurcation lesions



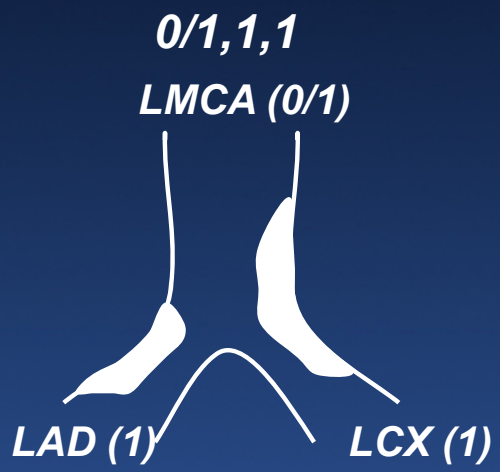
62%



14%



14%



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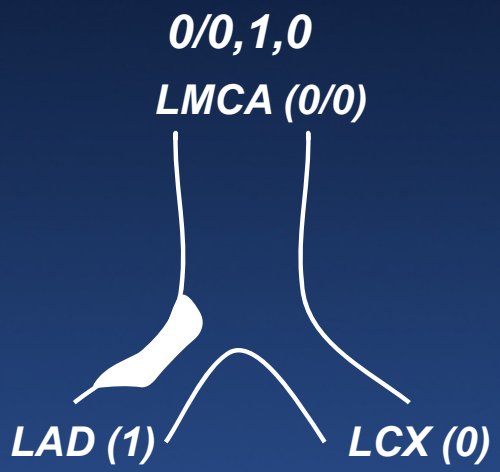

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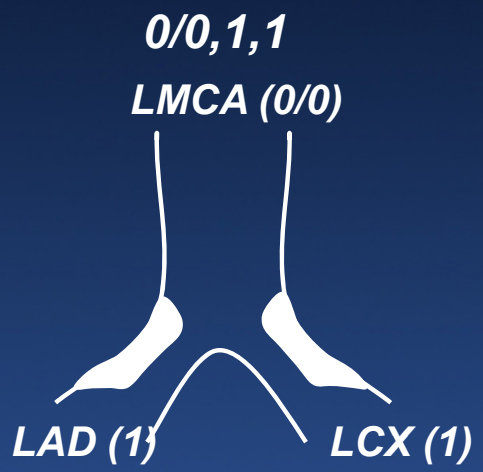

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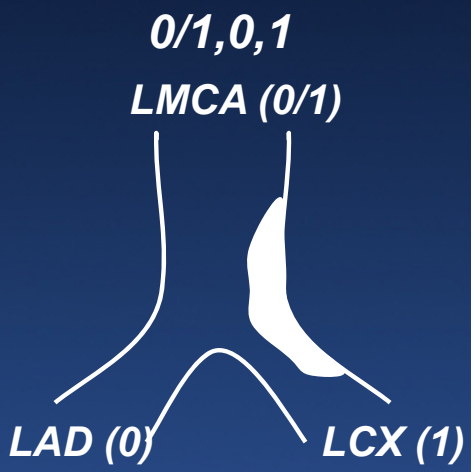

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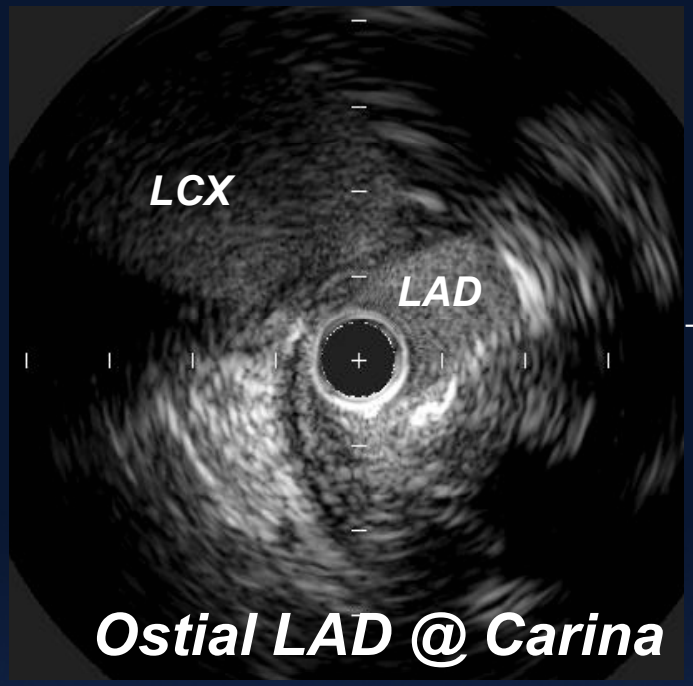
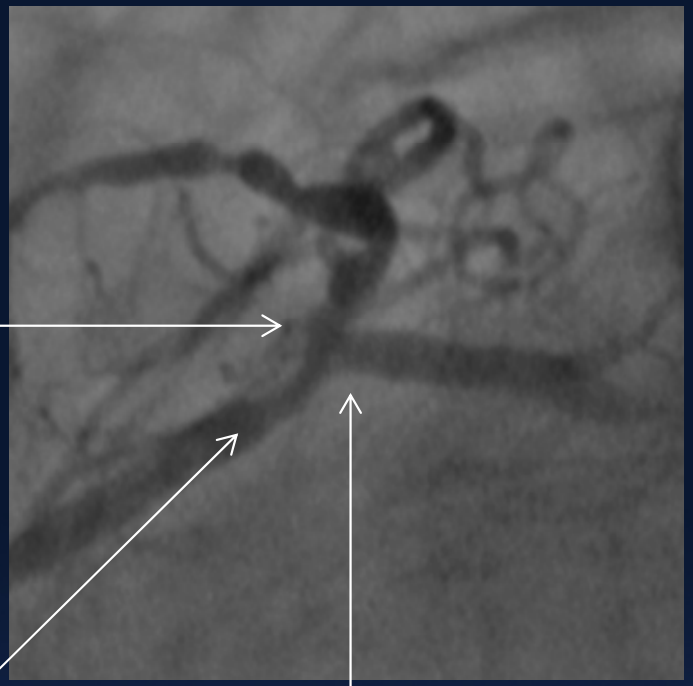
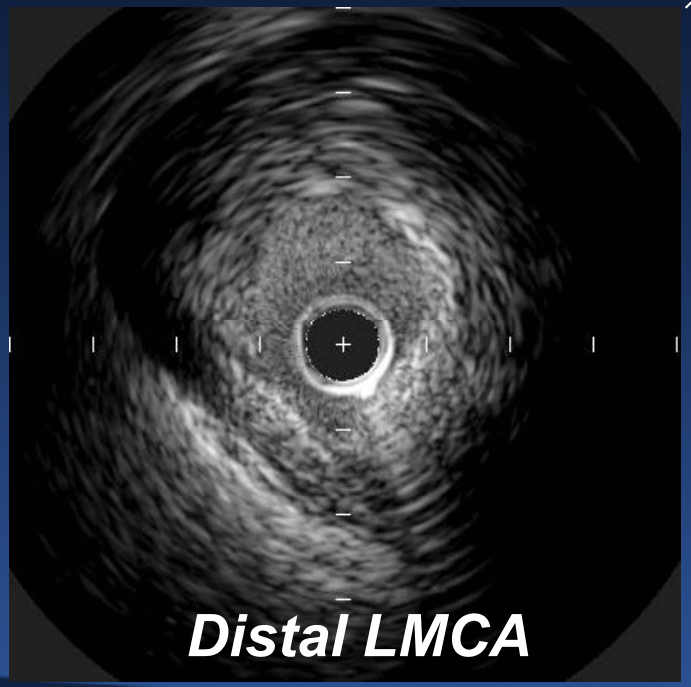
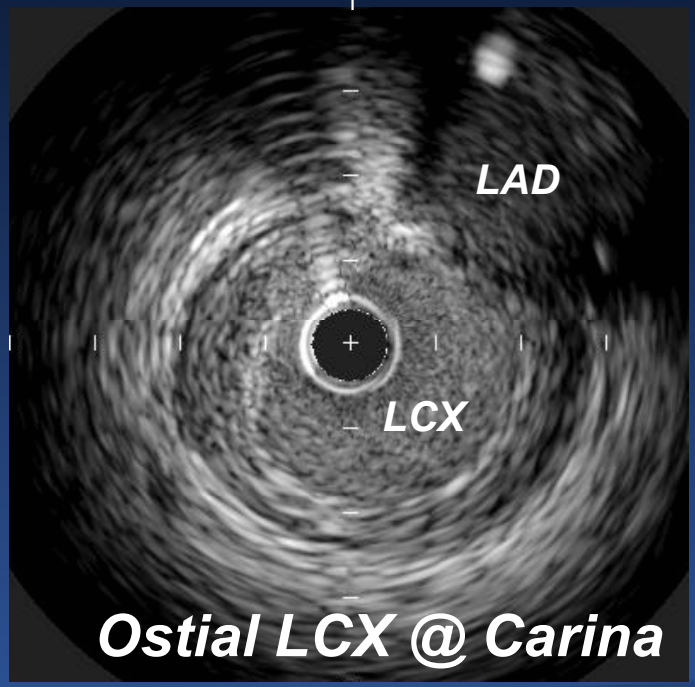
Oviedo et al. Circ Cardiovasc Interv. 2010;3:105-12

Oviedo et al. Circ Cardiovasc Interv. 2010;3:105-12


LCX
LAD
Ostial LAD @ Carina



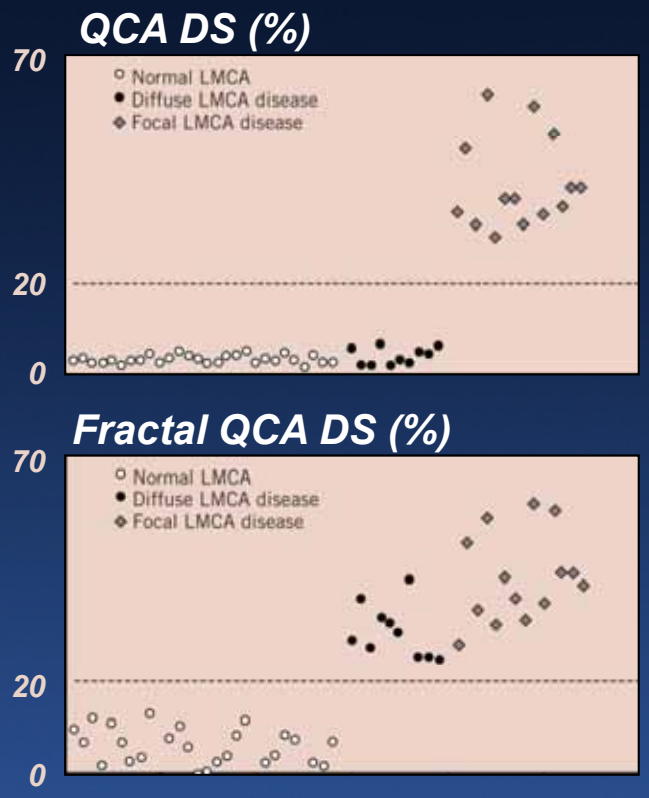

Distal LMCA

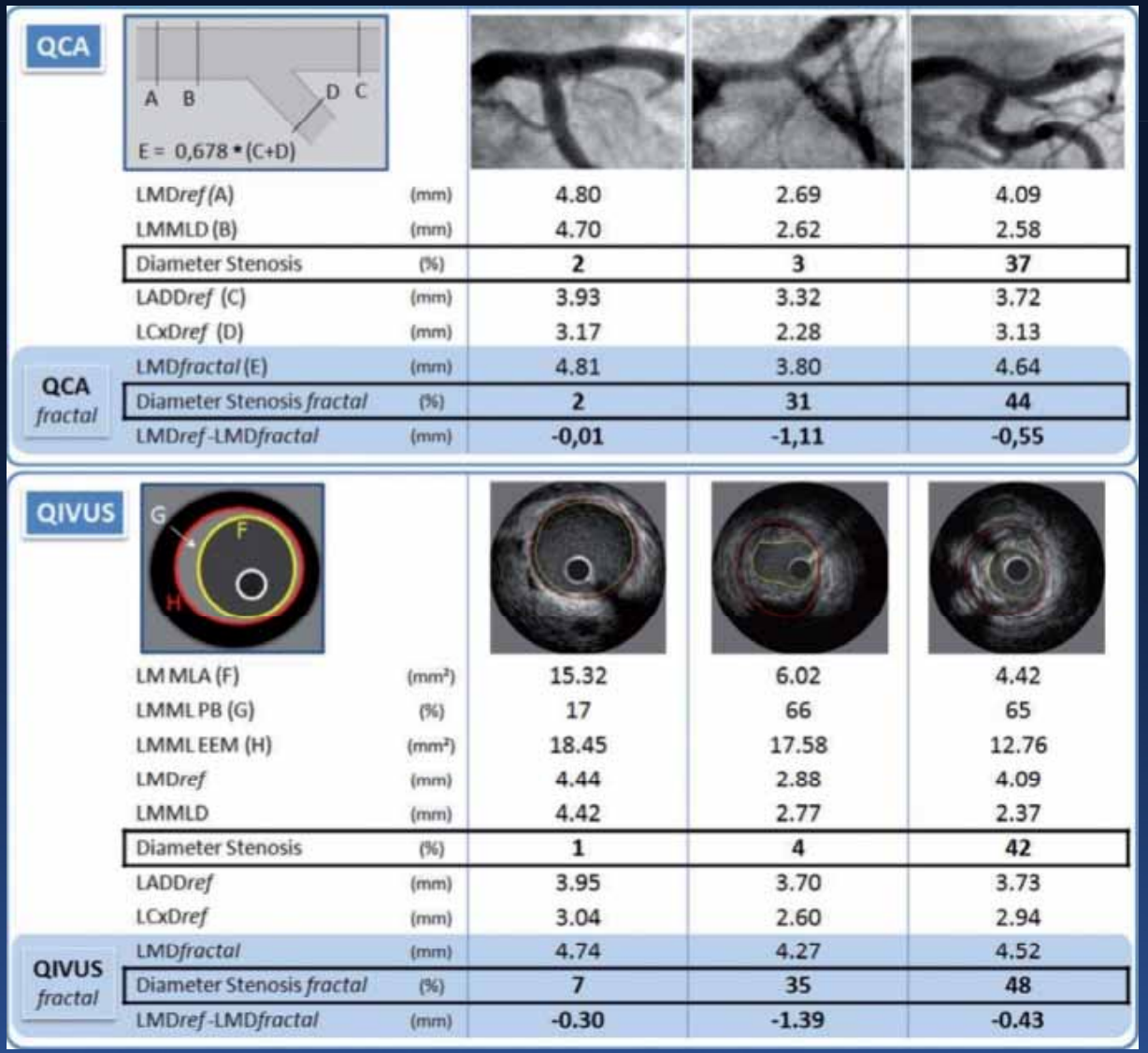

LAD
LCX
Ostial LCX @ Carina

These concepts regarding plaque distribution and the need for direct assessment of a sidebranch (rather than tangential imaging) also apply to non-LMCA bifurcations

“Small” LM = Diffuse LMCA disease

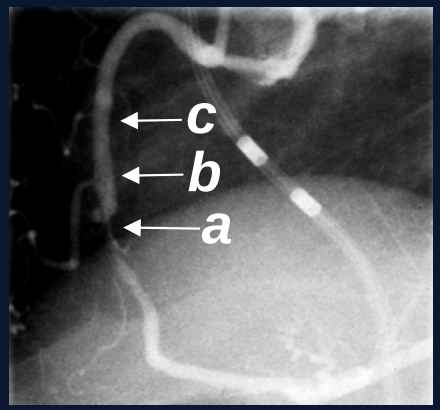
- **Murray’s Law**
  - $_{LMCA}r^3 = {}_{LAD}r^3 + {}_{LCX}r^3$
- **Fractal Geometry**
  - $_{LMCA}D = 0.678\ (_{LAD}D + {}_{LCX}D)$

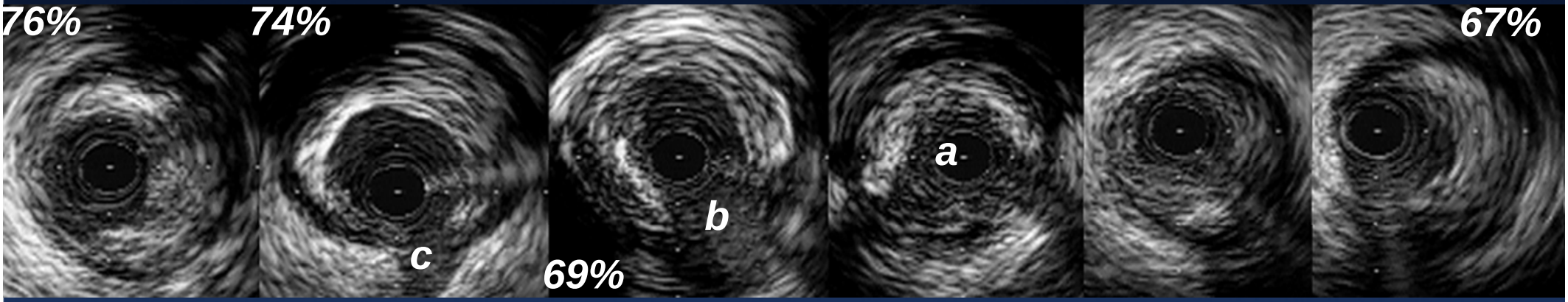




	LMD*ref* (A)	(mm)	4.80	2.69	4.09
	LMMLD (B)	(mm)	4.70	2.62	2.58
	Diameter Stenosis	(%)	2	3	37
	LADD*ref* (C)	(mm)	3.93	3.32	3.72
	LCxD*ref* (D)	(mm)	3.17	2.28	3.13
QCA *fractal*	LMD*fractal* (E)	(mm)	4.81	3.80	4.64
QCA *fractal*	Diameter Stenosis *fractal*	(%)	2	31	44
QCA *fractal*	LMD*ref* -LMD*fractal*	(mm)	-0,01	-1,11	-0,55
	LM MLA (F)	(mm²)	15.32	6.02	4.42
	LMML PB (G)	(%)	17	66	65
	LMML EEM (H)	(mm²)	18.45	17.58	12.76
	LMD*ref*	(mm)	4.44	2.88	4.09
	LMMLD	(mm)	4.42	2.77	2.37
	Diameter Stenosis	(%)	1	4	42
	LADD*ref*	(mm)	3.95	3.70	3.73
	LCxD*ref*	(mm)	3.04	2.60	2.94
QIVUS *fractal*	LMD*fractal*	(mm)	4.74	4.27	4.52
QIVUS *fractal*	Diameter Stenosis *fractal*	(%)	7	35	48
QIVUS *fractal*	LMD*ref* -LMD*fractal*	(mm)	-0.30	-1.39	-0.43

Matreff et al. Eurointervention 2010;5:709-15

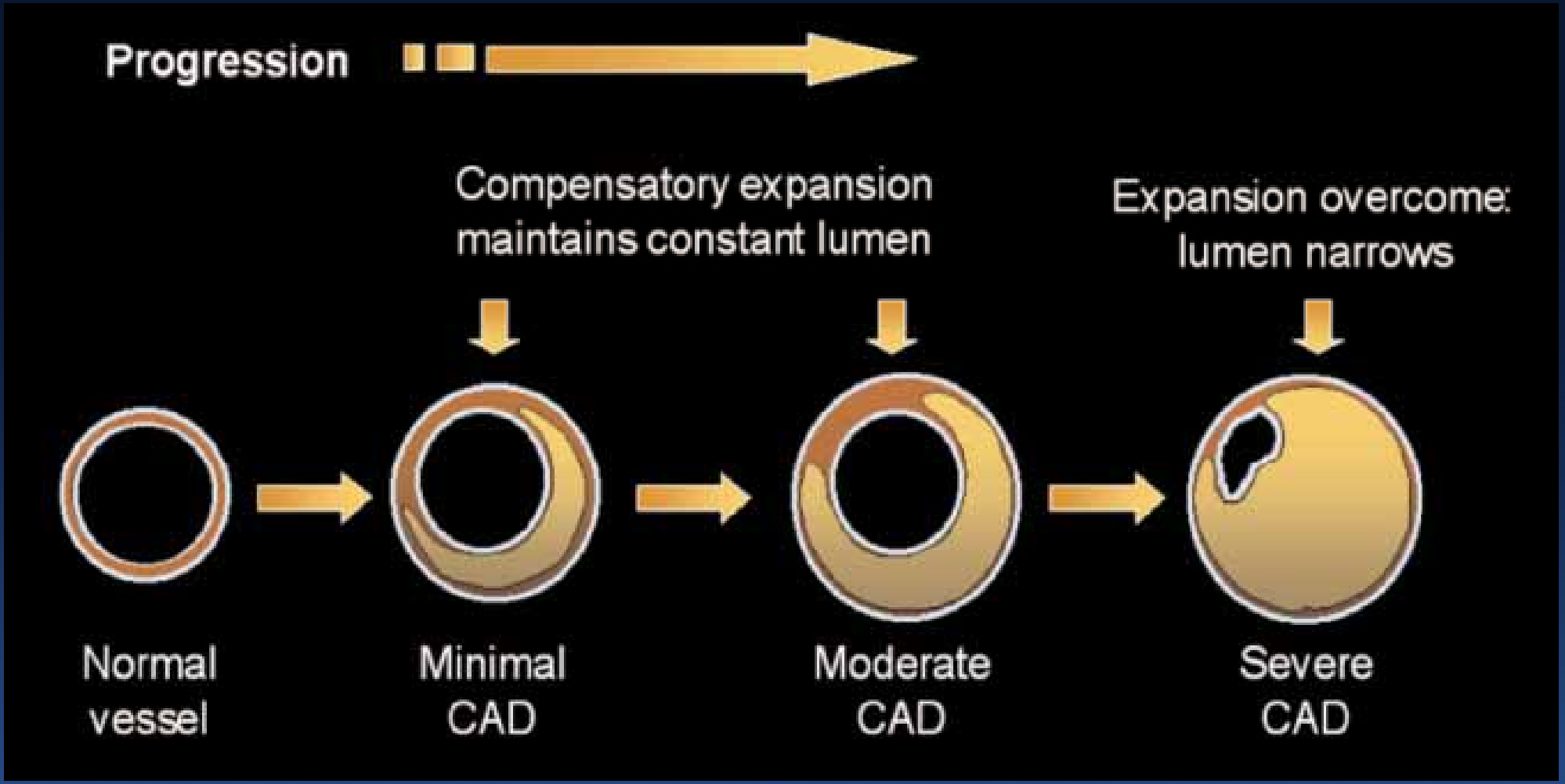




In 884 native coronary arteries, the plaque burden in the angiographically "normal" reference segment was 51±13% and 72±12% of plaque volume (range 46-86%) is in non-stenotic segments

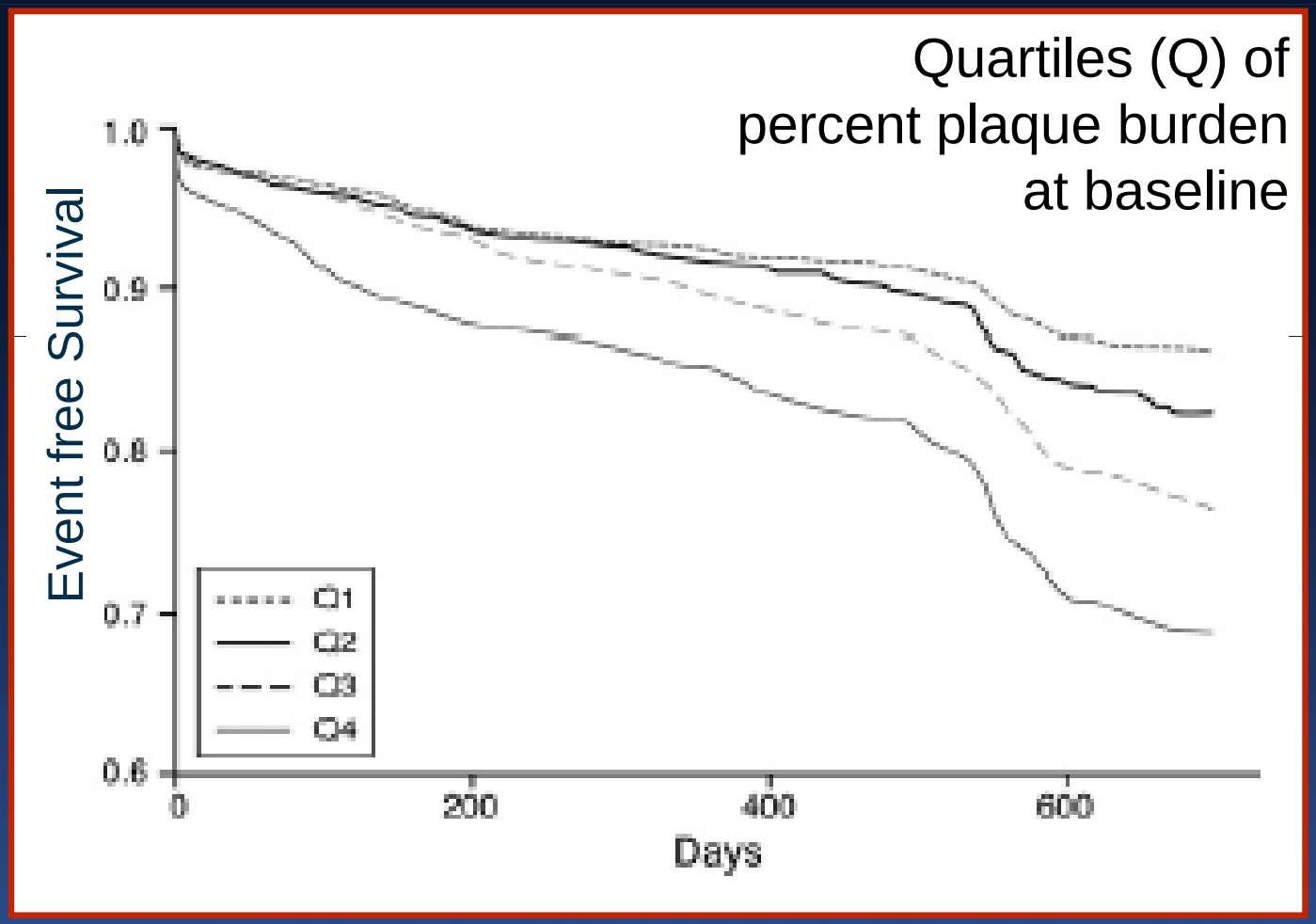
Mintz et al. J Am Coll Cardiol 1995;25:1479-85

Coronary Remodeling Hypothesis



Glagov et al N Eng J Med 1987;316:1371-5

Association Between Baseline Plaque Burden and Cardiovascular Events

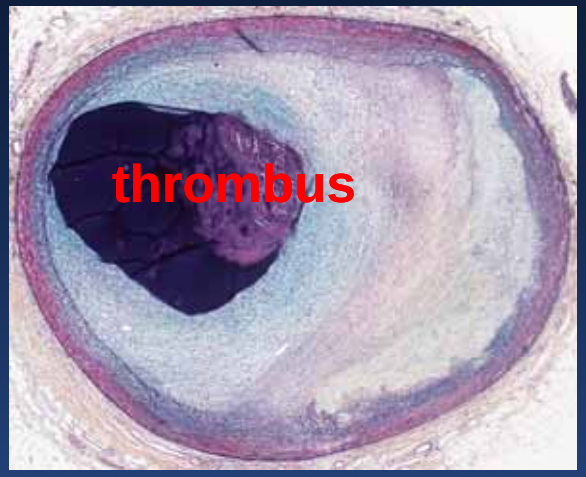


Nicholls et al. J Am Coll Cardiol 2010;55:2399-407

Causes of Intracoronary Thrombosis

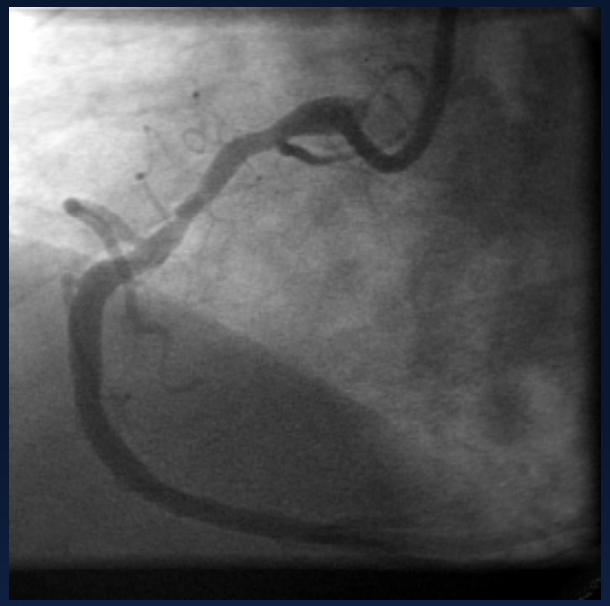
Plaque Rupture
80% (m) – 60% (f)

Plaque Erosion
20% (m) - 40% (f)

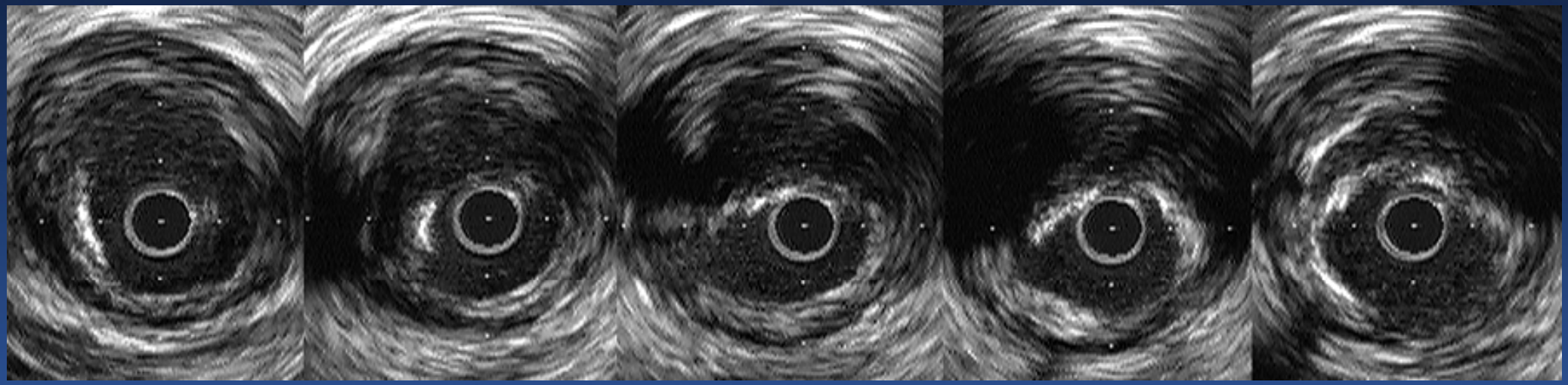


Calcified Nodule
2% - 7% (m/f)

Farb A et al. Circulation 1995;92:1701-9.



0 → 1mm → 4mm

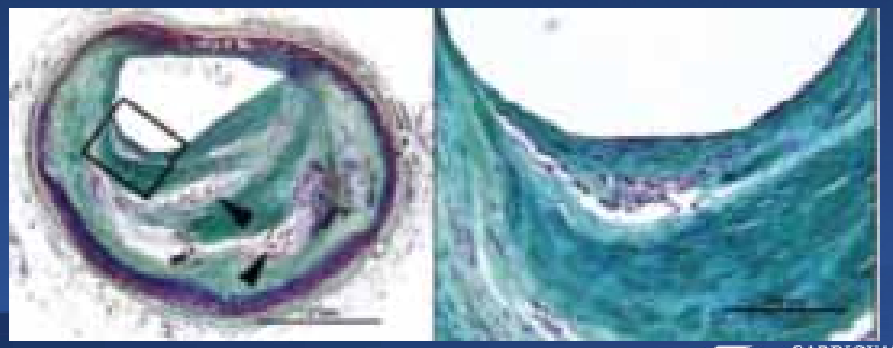


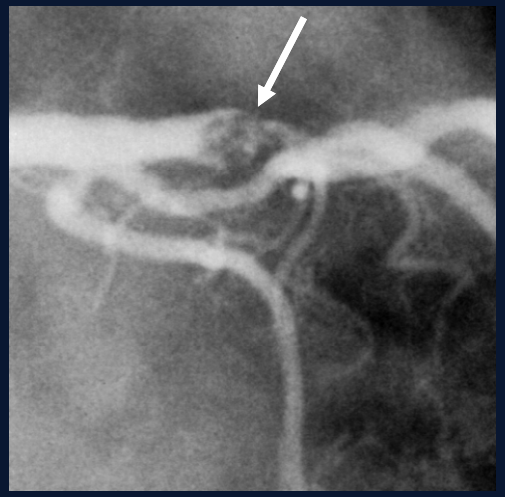
Culprit plaque ruptures in ACS/AMI studied with 3 vessel imaging

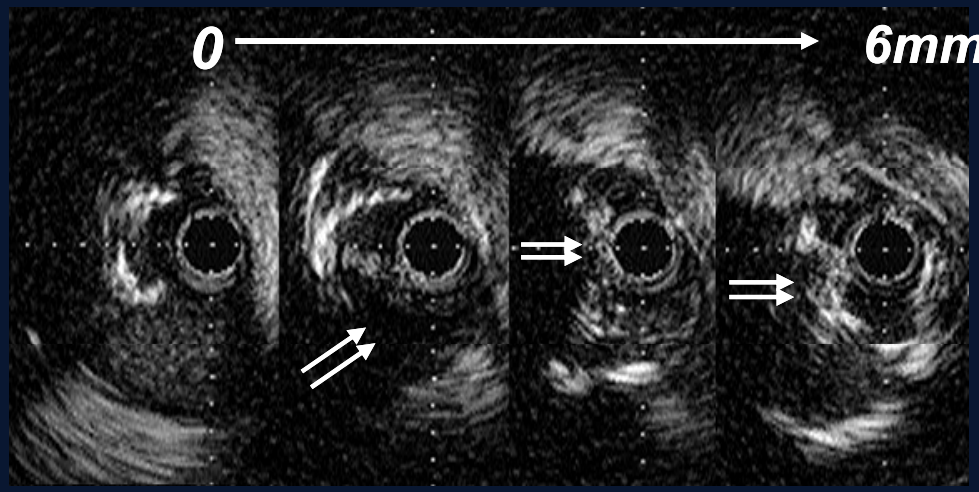
		# of pts	Culprit plaque ruptures	Secondary plaque ruptures
Riouful. Circulation 2002;106:804-8	IVUS	24	38%	79%
Hong. Circulation 2004;110:928-33	IVUS	122	66%	17%
Tanaka. J Am Coll Cardiol 2005;45:1594-9	IVUS	45	47%	24%
Fujii. J Am Coll Cardiol 2008;52:787-92	OCT	35	46%	31%
Tanaka. Am J Cardiol 2008;102:975-9	OCT	43	65%	12%
Kubo. Am J Cardiol 2012;105:318-22	OCT	26	77%	12%
Kukunaga. Eurointervention 2012;8:955-61	OCT	70	46%	31%
Xie. JACC Cardiovasc Imaging, in press	IVUS (20MHz)	660	N/A	14%
Average	IVUS		58%	17%
	OCT		55%	

Fate of secondary plaque ruptures

- 14 pts with 28 plaque ruptures with MLA >4.0mm^2 treated with statins and dual antiplatelet therapy. At 22±13 months, no clinical events and half had healed
  - (Rioful et al. Circulation 2004;110:2875-80)
- 28 pts with non-culprit plaque ruptures (half treated with statins) followed for 11.9±1.3 months. No TLR in statin-treated pts (p=0.11).
  - (Hong et al. Atherosclerosis. 2007;19:107-14)
- The event rate in 93 pts with at least 1 secondary plaque rupture was the same as in 567 pts with no secondary plaque ruptures.
  - (Xu et al. JACC Cardiovasc Imaging, in press)
- In 142 men with sudden cardiac death, there were 44 acute rupture sites - 9 with 1, 9 with 2, 9 with 3, and 6 with 4 healed previous rupture sites.
  - (Burke et al. Circulation 2001;103:934-40)







Volume 132, Number 2
American Heart Journal

Intravascular ultrasound identification of calcified intraluminal lesions misdiagnosed as thrombi by coronary angiography

Gaston R. Duissaillant, MD, Gary S. Mintz, MD, Augusto D. Pichard, MD, Kenneth M. Kent, MD, PhD, Lowell F. Satler, MD, Jeffrey J. Popma, MD, Jennifer Griffin, BS, and Martin B. Leon, MD
Washington, D.C.

Accurate identification of coronary atherosclerotic plaque composition is important for optimum patient management and transcatheter therapy. In particular, the presence of thrombus typically leads to protracted hospitalization, intravenous or intracoronary thrombolysis, prolonged systemic anticoagulation, and the use of transcatheter devices designed to remove thrombi. Furthermore, thrombi may be implicated in poor outcome after transcatheter therapy. Intraluminal filling defects are believed to be the most specific angiographic markers of thrombus. We report three patients with intracoronary filling defects initially diagnosed as thrombus; however, intravascular ultrasound (IVUS) imaging showed that these filling defects represented calcified nodules.

IVUS studies were performed with one of two commercially available systems. The first (Cardiovascular Imaging Systems Inc./InterTherapy Inc., Sunnyvale, Calif.) incorporated a single element 25-MHz transducer and an angled mirror mounted on the tip of a flexible shaft that was rotated at 1800 rpm within a 3.9F short monorail polyethylene imaging sheath to form planar cross-sectional images in real time. The second (Cardiovascular Imaging Systems) incorporated a single element 30-MHz beveled transducer within either a 2.9F long monorail imaging catheter having a common distal lumen design (the distal lumen alternatively accommodates the imaging core or the guide wire, but not both) or within a 3.2F short monorail imaging catheter. With both systems the imaging catheter was advanced 5 to 10 mm beyond the target lesion and the transducer was withdrawn automatically at 0.5 mm/sec within the imaging sheath to perform the imaging sequence. IVUS studies were recorded on ½-inch high resolution s-VHS tape for offline analysis.

Patient 1. A 78-year-old white man with a history of pneumoconiosis, gastrectomy for stomach cancer, congestive heart failure from dilated cardiomyopathy, and coronary artery disease was seen for progressive angina. A coronary angiogram showed a 95% ostial right coronary artery lesion with a round filling defect highly suggestive of thrombus. He was treated with heparin and sent home taking coumadin to dissolve the thrombus. However, he returned with worsening angina; coronary angiography still showed a large filling defect almost completely occluding the right coronary artery (Fig. 1). He was initially treated with an intracoronary urokinase infusion of 250,000 U followed by the use of a 2.2-mm transluminal extraction atherectomy cutter (Interventional Technologies, Inc., San Diego, Calif.) and 3.5-mm adjunct balloon

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Supported in part by the Cardiology Research Foundation, Washington, D. C.
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Am Heart J 1996;132:687-9.

0002-8703/96/$5.00 + 0 4/4/71990

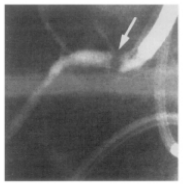


Fig. 1. Proximal right coronary artery filling defect *(arrow)* was initially thought to represent intracoronary thrombus. Patient was treated with prolonged system anticoagulation, intracoronary thrombolysis, and extraction atherectomy. IVUS imaging showed that filling defect was, in fact, calcified lesion protruding into lumen.

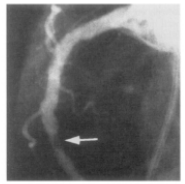


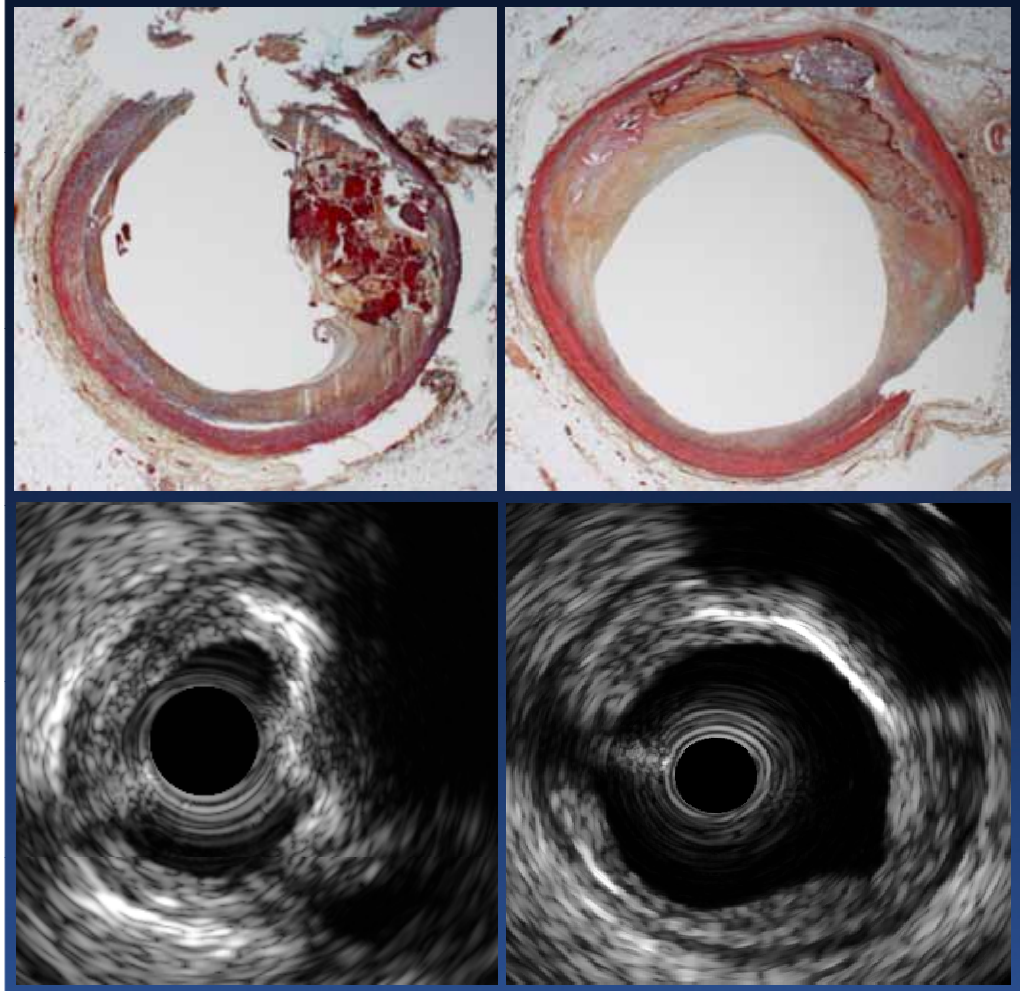
Fig. 2. Mid-right coronary artery filling defect *(arrow)* was initially thought to represent intracoronary thrombus. Patient was also treated with prolonged systemic anticoagulation. IVUS imaging showed that filling defect was, in fact, calcified lesion protruding into the lumen. Because of limited device availability, patient was referred for bypass graft surgery.

"We present three patients with classical angiographic features of intracoronary thrombus in whom IVUS imaging showed that the filling defects were not thrombi, but calcified (presumably atherosclerotic) masses."

Dussaillant et al. Am Heart J 1996;132:687-9

IVUS Validation Study

Calcific nodule	Non-nodular calcium



Compared to non-nodular calcium, IVUS characteristics of calcific nodules included

- protrusion with a convex shape of the luminal surface (94.1% in calcific nodules vs 9.7% in non-nodular calcium, p<0.001)
- convex shape of the luminal surface of calcium (100% vs 16.0%, p<0.001)
- irregular luminal surface (64.7% vs 11.6%, p<0.001)
- irregular leading edge of calcium (88.2% vs 19.0%, p<0.001).

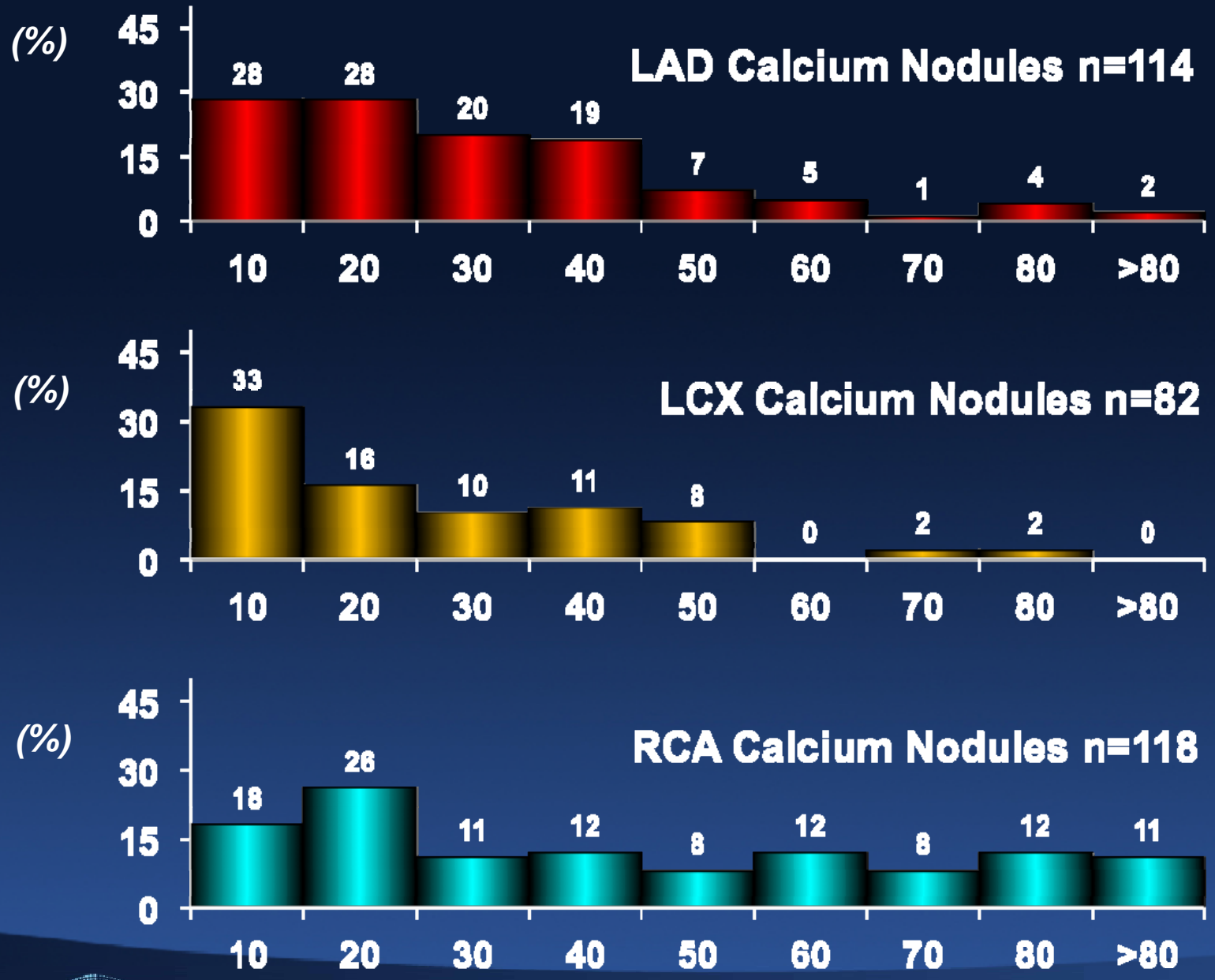
Lee et al. Am J Cardiol 2011;108:1547-51

Calcified Nodules in PROSPECT

- At least one calcified nodule was present in 16% of arteries and 30% of patients.
- Two or more calcified nodules were detected in 48 coronary arteries (3%) in 76 patients (12%).
- The angiographic appearance was severe calcium in 3, moderate calcium in 35, hazy in 19, and normal in 257
- The VH-IVUS appearance was a fibroatheroma in 42% (116 of 276) , but only a VH-TCFA in 5.

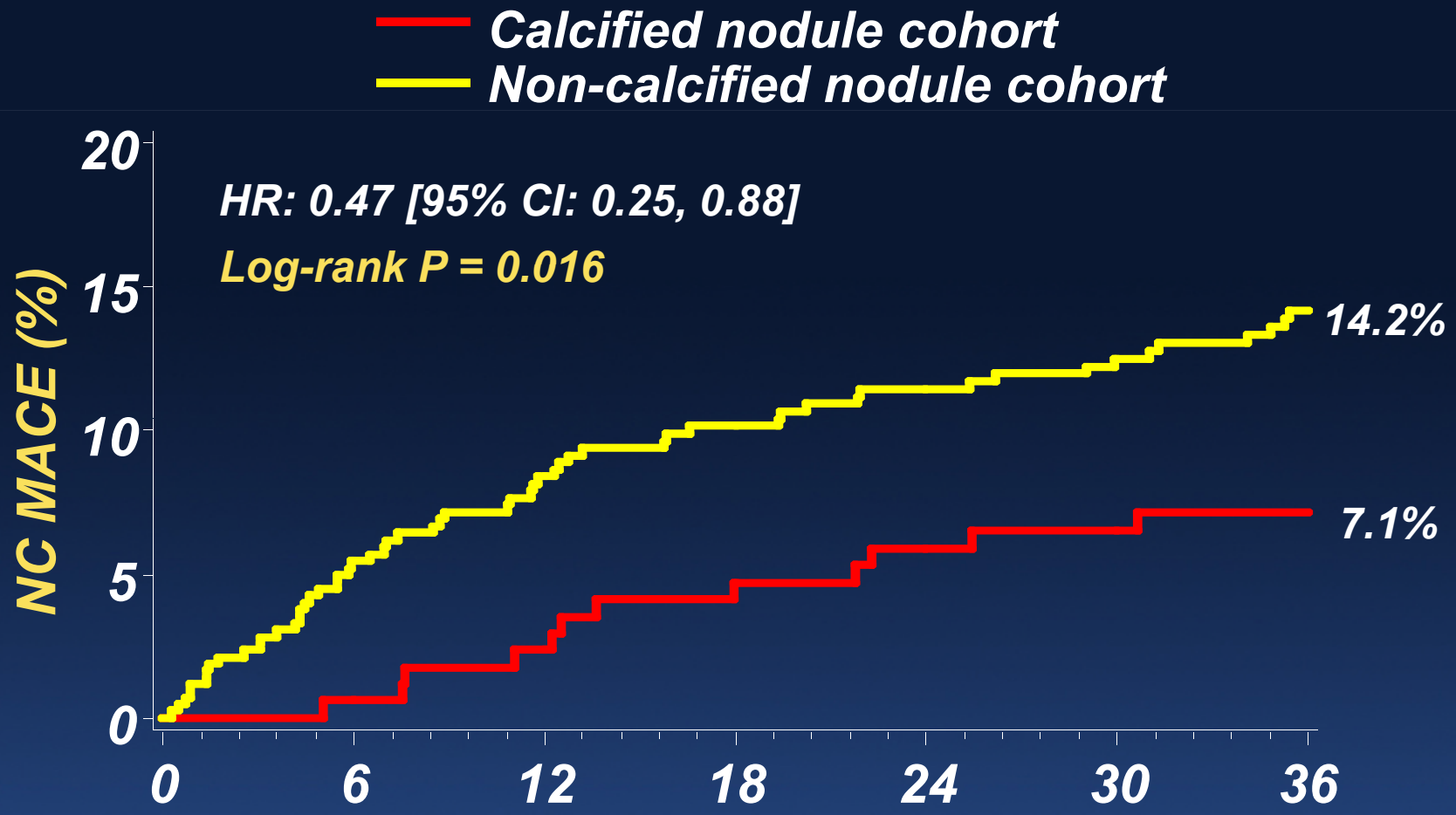
Xu et al. Circulation 2012;126:537-45

Longitudinal Distribution of 314 Calcified Nodules in PROSPECT



Xu et al. Circulation 2012;126:537-45

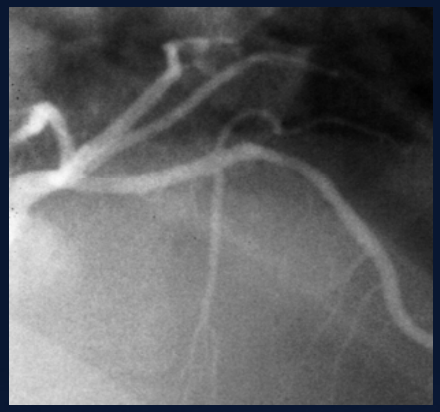
Clinical Outcome of Patients with Calcified Nodules in PROSPECT

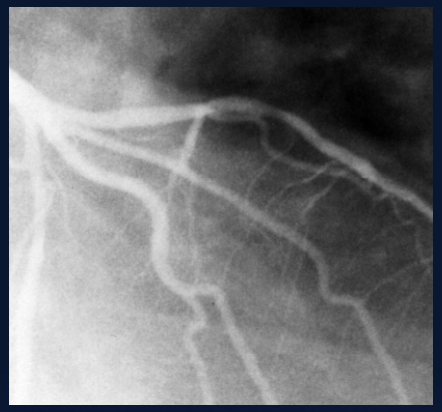


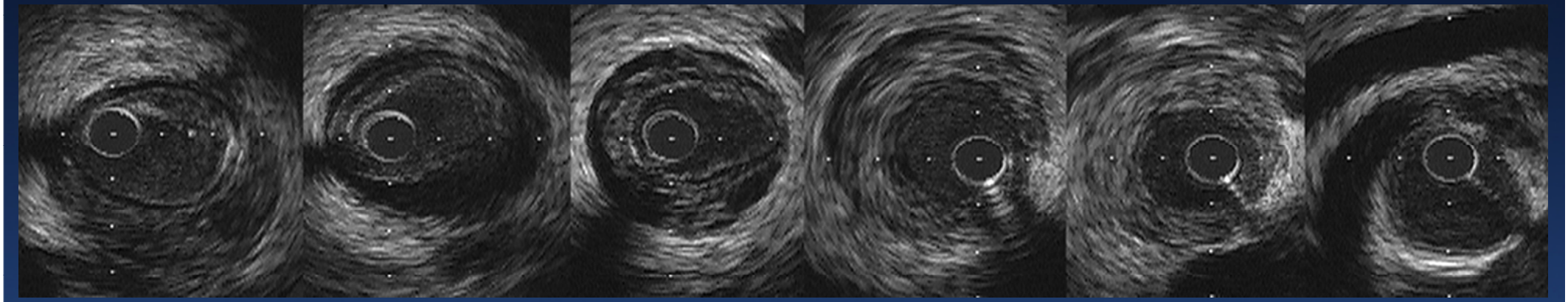
Number at risk

CN cohort	185	170	166	162	156	150	90
Non-CN cohort	438	389	370	352	338	324	190

Xu et al. Circulation 2012;126:537-45







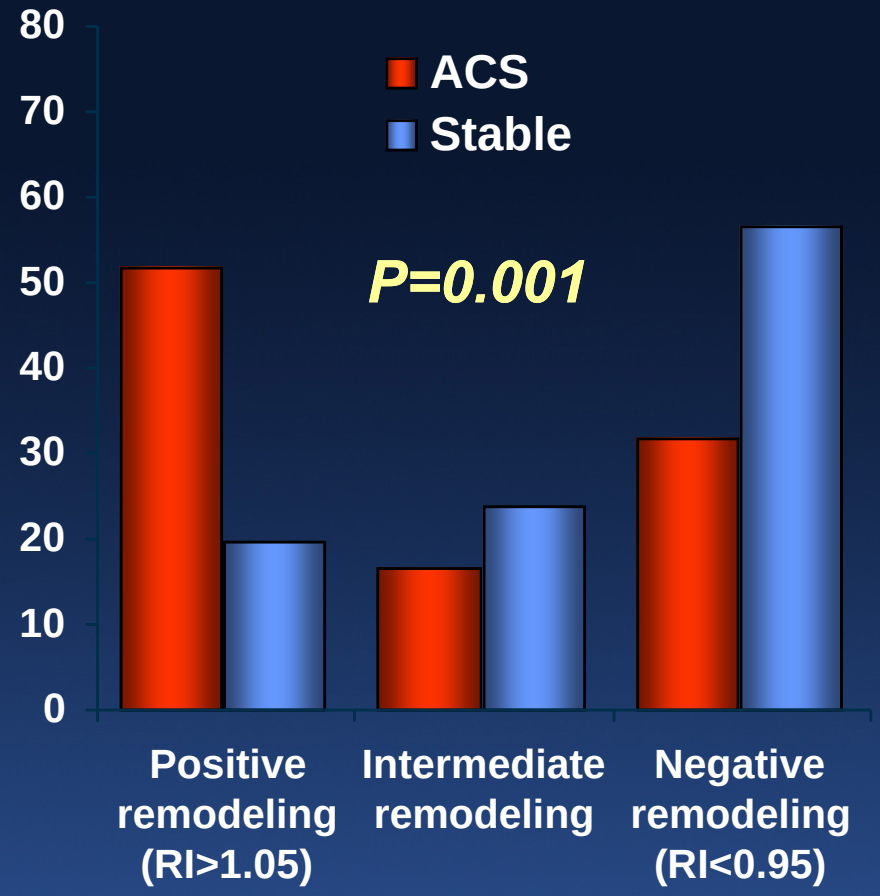
0 → 8 → 40mm

Spontaneous Coronary Artery Dissection (SCAD)

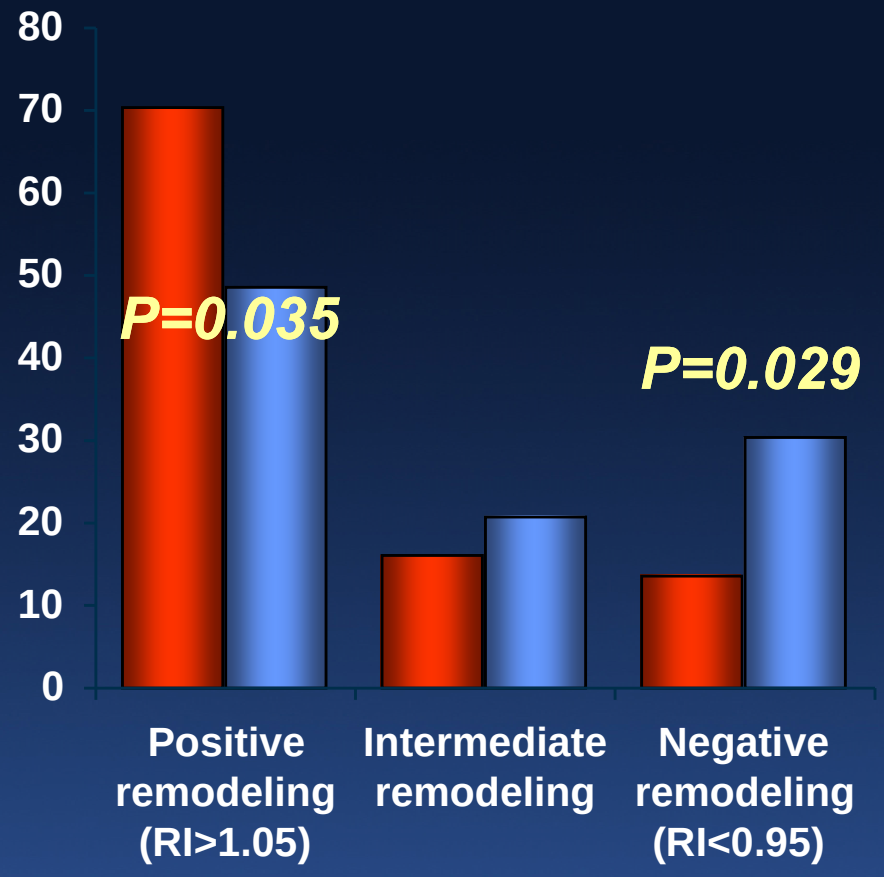
- SCAD affects a young, predominantly female population, frequently presenting as STEMI.
- In-hospital mortality is low regardless of initial treatment, and in most patients prognosis appears favorable after initial stabilization.
  - Revascularization appears to be required in 35-45% of patients, remains challenging and is associated with a high rate of complications (especially PCI), and should be restricted to patients with ongoing/recurrent ischemia and suitable anatomy.
- Long-term prognosis is generally favorable regardless of treatment although the risks of SCAD recurrence and MACE emphasize the need for close follow-up.

Ito et al. Am J Cardiol 2011;107:1590-6
Tweet et al. Circulation 2012;126:579-88
Alfonso. Circulation 2012;126:667-70
Alfonso et al. JACC Cardiovasc Interv 2012;5:1062-70

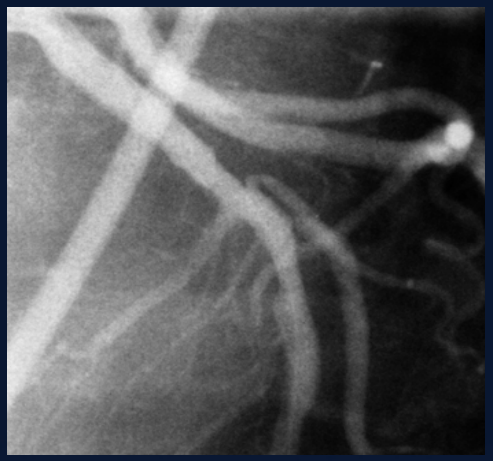
Despite the fact that more than a dozen histopathologic and IVUS studies showed a relationship between positive remodeling and unstable lesion morphology, neither the remodeling index nor positive remodeling were predictive of events in PROSPECT

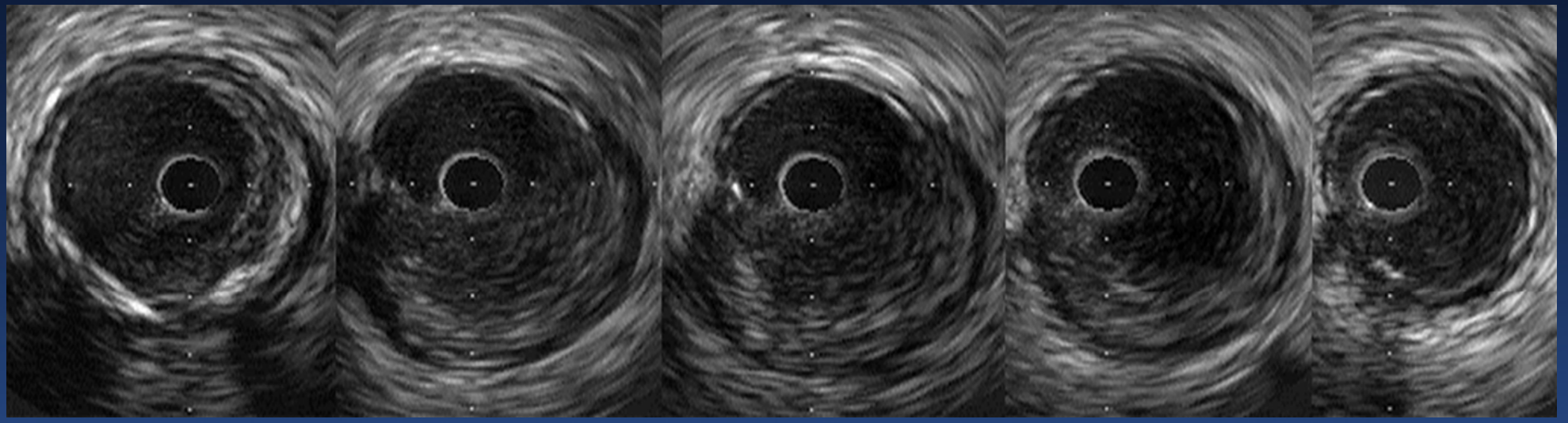


(Schoenhagen et al. Circulation 2000;101:598-603)

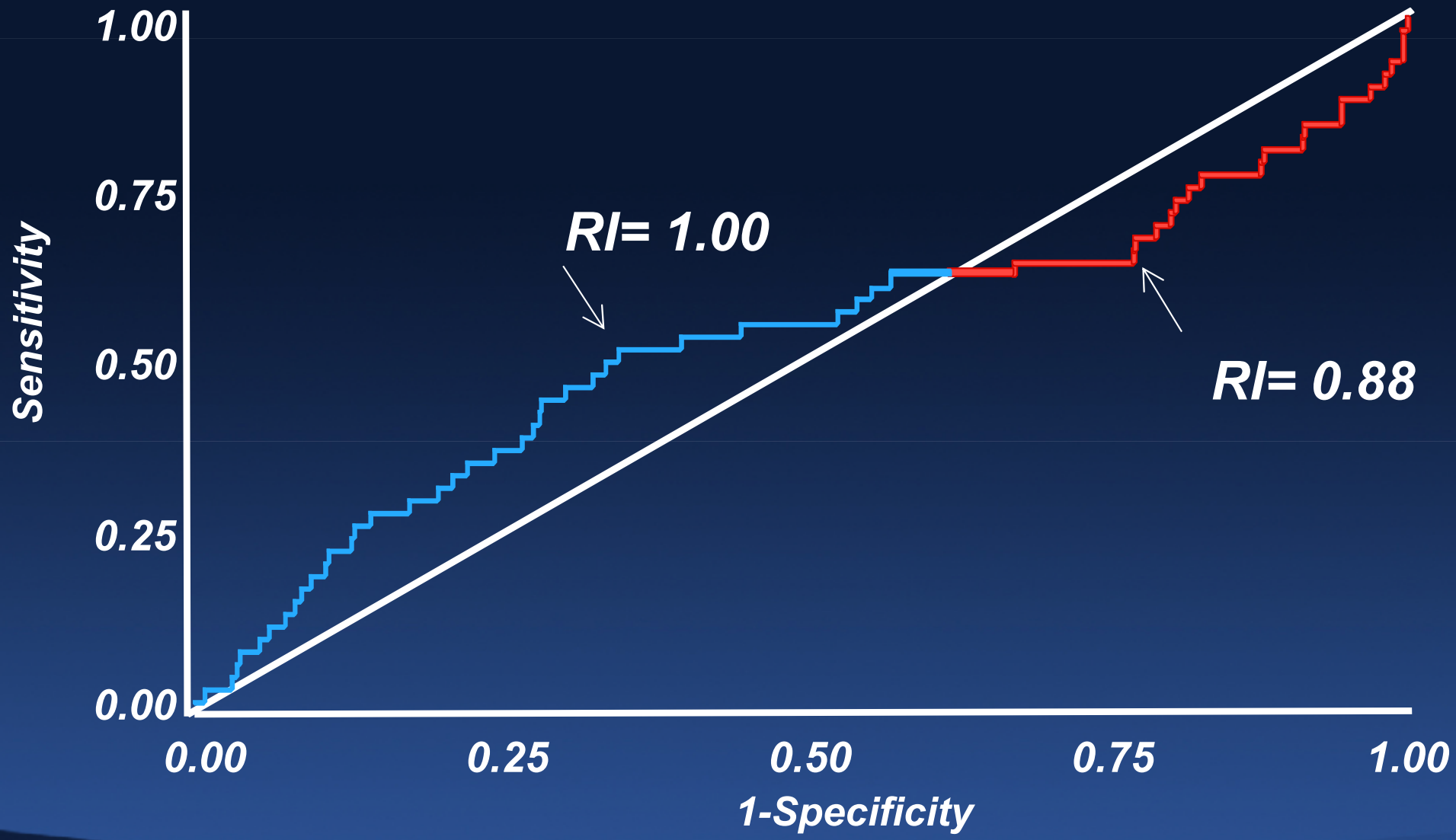


(Prati et al. Circulation 2003;107:2320-5)



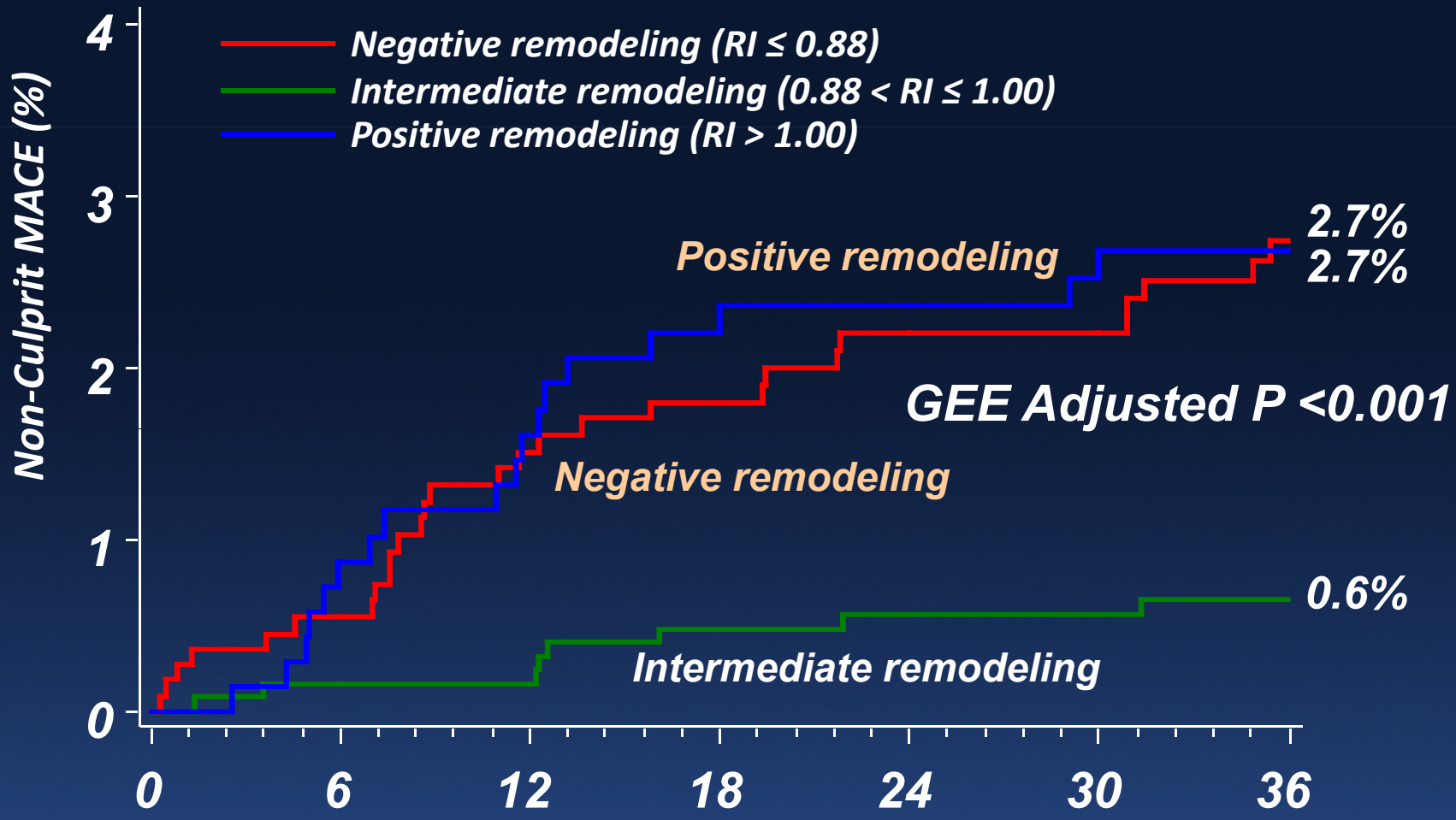

0
3
12mm

Remodeling index that predicted MACE in PROSPECT (Training set: 1041 lesions in 214 pts)



Inaba et al. JACC Cardiovasc Imaging, in press

Impact of Positive and Negative Remodeling in PROSPECT (Test set: 2182 lesions is 443 pts)



Number at risk							
Negative Remodeling	1137	1041	1019	996	970	940	598
Intermediate Remodeling	1355	1262	1244	1209	1186	1154	712
Positive Remodeling	731	675	666	640	622	601	385

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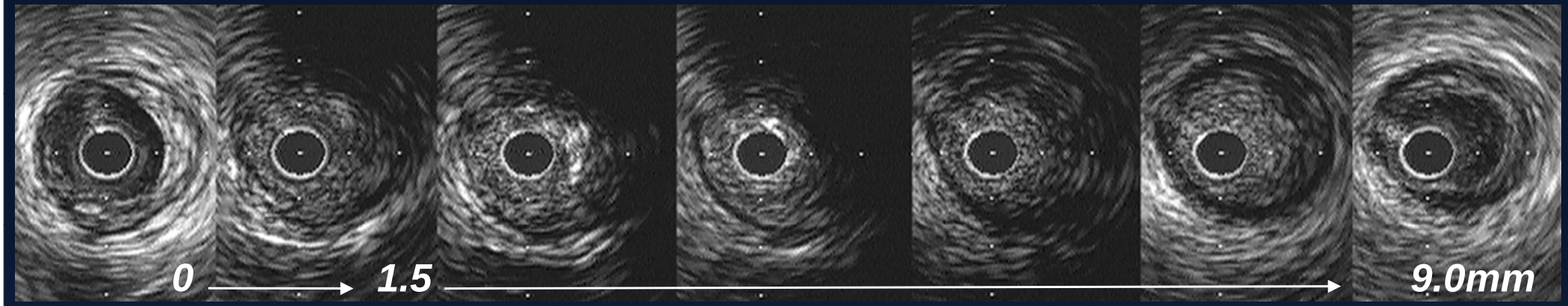
Inaba et al. JACC Cardiovasc Imaging, in press

CARDIOVASCULAR RESEARCH FOUNDATION At the heart of innovation

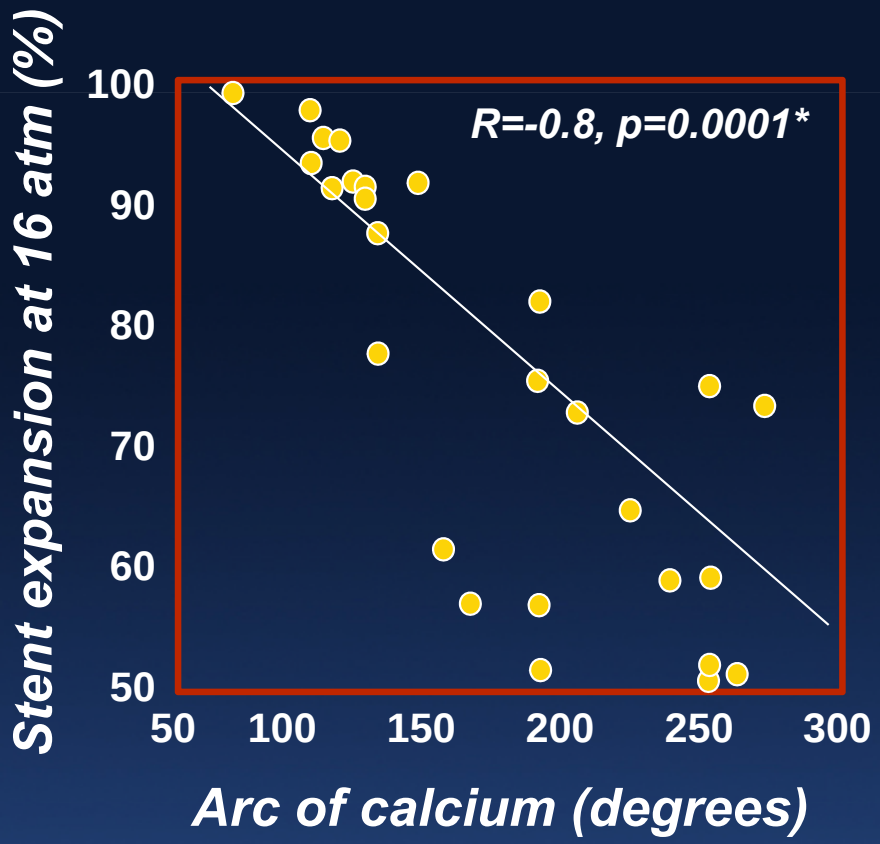
Attenuated Plaque

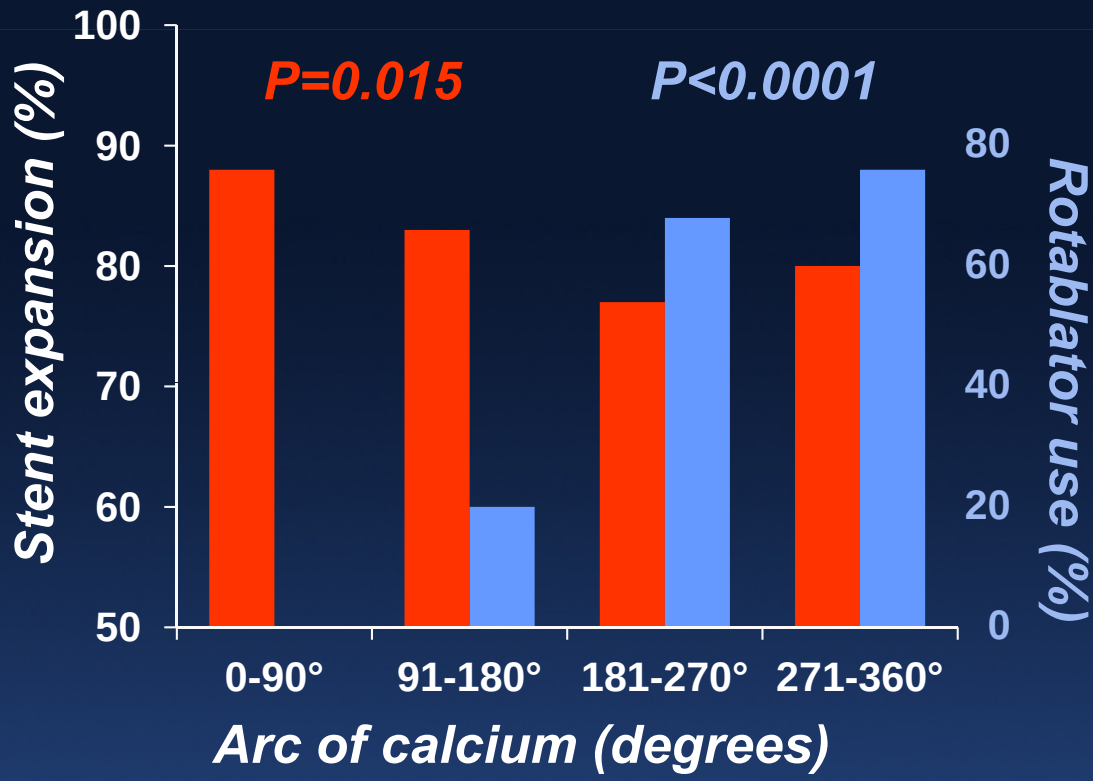


- *Attenuated plaques were seen in 39.6-78.0% of STEMI, 17.6% of NSTEMI, and 0% of stable angina.*
- *Attenuate plaques were associated with more fibroatheromas and a larger necrotic core (on VH-IVUS).*
- *In ACS or MI pts with attenuated plaques (1) the level of CRP was higher, (2) angiographic thrombus and initial coronary flow <TIMI 2 were more common, and (3) no-reflow or flow deterioration post-PCI was also more common.*
- *In STEMI patients with attenuated plaques, the amount, not the presence, of attenuated plaque predicted no-reflow or MRI-derived microvascular obstruction post stent implantation*
- *Attenuated plaque was associated with the presence of TCFA, ruptured plaques, thrombus, and greater lipid content*
- *Attenuated plaques contained the highest NIRS probability of lipid core, and by VH-IVUS, 93.5% of attenuated plaques contained confluent necrotic core and were classified as fibroatheromas*

(Lee et al. JACC Cardiovasc Interv. 2009;2:65-72)
(Wu et al, Am J Cardiol 2010;105:48-53)
(Okura et al, Circ J 2007;71:648-53)
(Wu et al. JACC Cardiovasc Interv 2011;4:495-502)
(Lee et al JACC Cardiovasc Interv. 2011;4:483-91)
(Kubo et al. Cardiol Res Pract. 2011;687515)
(Pu et al. Eur Heart J 2012;33:372-83)
(Shiono et al, JACC Cardiovasc Interv 2013;6:847-53)

Stent Expansion in Calcified Lesions





**There was a similar, albeit less strong, correlation after 20 atm inflation (r=-0.58, p=0.0007)*

Vavarunakis et al. Catheter Cardiovasc Interv 2001;52:164-172
Hoffmann et al. Eur Heart J 1998;19:1224-31

We all have cases like this. . .

Catheterization and Cardiovascular Interventions 52:208–211 (2001)

Case Reports

Rotational Atherectomy (Stentablation) in a Lesion With Stent Underexpansion Due to Heavily Calcified Plaque

Yoshio Kobayashi,[1] MD, Paul S. Teirstein,[2] MD, Thomas J. Linnemeier,[3] MD, Gregg W. Stone,[1] MD, Martin B. Leon,[1] MD, and Jeffrey W. Moses,[1*] MD

We report treatment of a lesion with coronary stent underexpansion due to heavily calcified plaque. Conventional balloon angioplasty was attempted for in-stent restenosis, but the lesion was undilatable despite 25-atm inflation pressure. Intravascular ultrasound (IVUS) revealed stent underexpansion due to heavily calcified plaque. Rotational atherectomy was performed using a stepped burr approach, after which repeat IVUS revealed marked ablation of the stent-calcium complex. Adjunctive balloon angioplasty then easily resulted in full balloon and stent expansion, with an excellent angiographic and IVUS result. The patient's hospital course was uneventful. *Cathet Cardiovasc Intervent 2001; 52:208–211.* © 2001 Wiley-Liss, Inc.

Key words: restenosis; angioplasty; coronary artery disease

INTRODUCTION

Although previous randomized trials [1] have demonstrated lower restenosis rates in selected lesions with coronary stents compared to conventional balloon angioplasty, in-stent restenosis remains an important clinical problem [2]. Intimal hyperplasia is the predominant cause of in-stent restenosis [3]. However, in-stent restenosis predominantly due to stent underexpansion (pseudo–in-stent restenosis) is sometimes observed, especially in heavily calcified lesions [4]. While conventional balloon angioplasty can be attempted, rigid calcified plaque may prevent full stent expansion. This case report describes successful treatment of a lesion with stent underexpansion due to heavily calcified plaque using rotational atherectomy.

CASE REPORT

A 69-year-old male status postcoronary bypass surgery with previous stent placement in the proximal left circumflex artery (LCx) was admitted due to exertional angina. Coronary angiography revealed in-stent restenosis of 90% severity in the proximal LCx (Fig. 1A). The left anterior descending coronary artery (LAD) and the right coronary artery (RCA) were totally occluded. A saphenous vein graft (SVG) to the LAD had a 70% stenosis at the distal anastomosis and an SVG to the RCA had a 70% stenosis at the distal segment. The moderate lesions in the SVG to the LAD and RCA were successfully treated with stents. Conventional balloon angioplasty was then performed to treat the in-stent restenosis in the proximal LCx. Although a 3.25-mm NC Ranger balloon catheter (Boston Scientific, Maple Grove, MN) was inflated up to 25 atm (Fig. 1B), it would not fully expand (Fig. 1C). The patient remained symptomatic and was referred to our institute for repeat attempted angioplasty of the lesion in the proximal LCx.

Procedure

Coronary angiography demonstrated a tandem lesion in the proximal LCx (Fig. 2A). Rotational atherectomy was planned for ablation of presumably densely fibrotic hyperplasia tissue. A 0.009″ RotaWire (Extra support) with 0.014″ tip (Boston Scientific) was placed across the lesion into the distal LCx. Intravascular ultrasound (IVUS) imaging was performed in the proximal LCx using a 30-MHz 3.2 Fr UltraCross catheter (Boston Sci-

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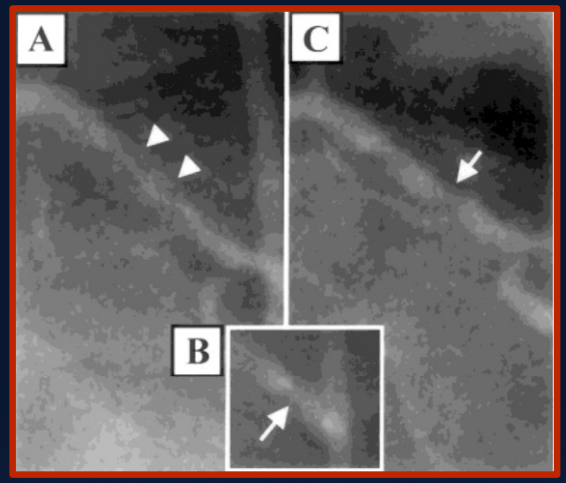
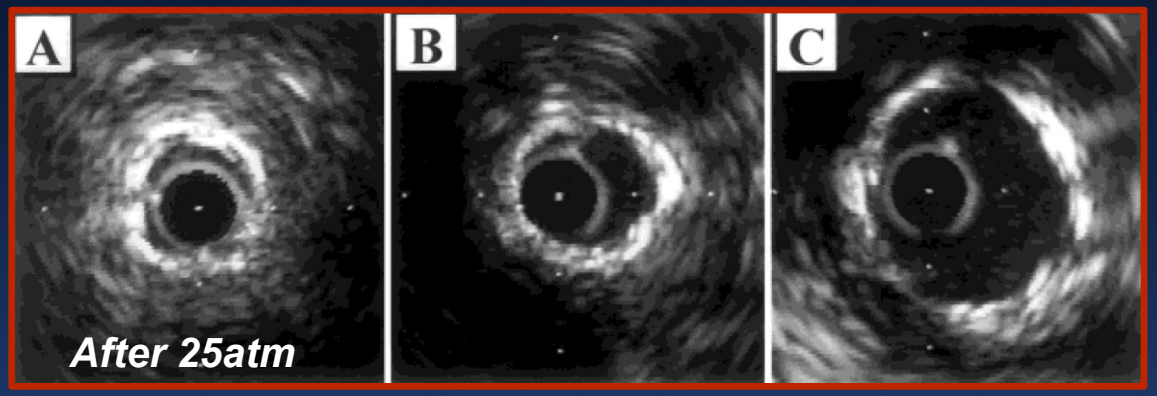
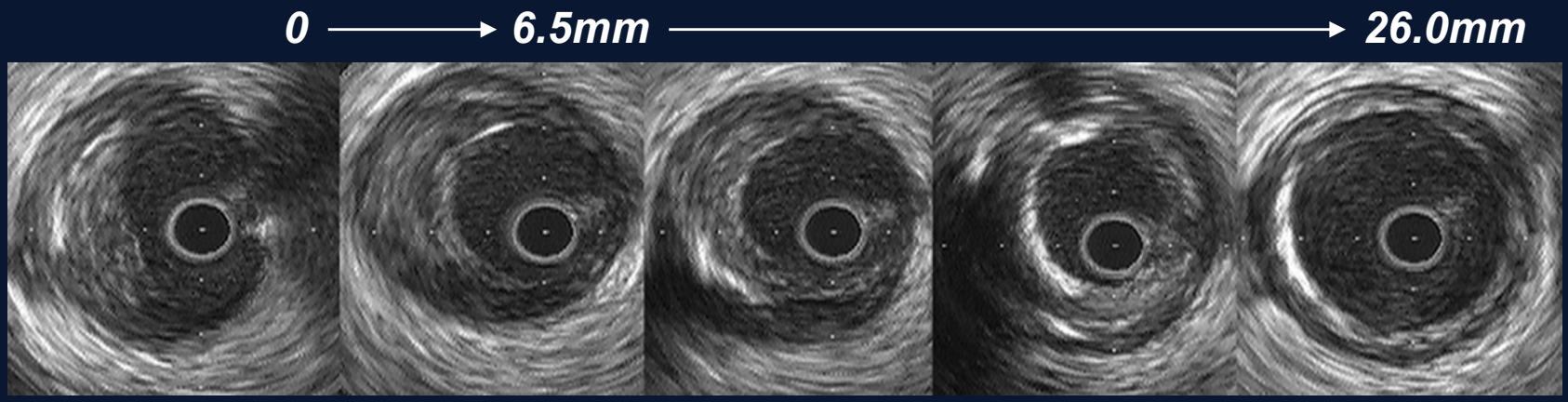
Received 16 June 2000; Revision accepted 24 September 2000





. . . however, it is hard to define a threshold of IVUS calcium where plaque modification is "necessary." The missing piece of information may be calcium thickness.

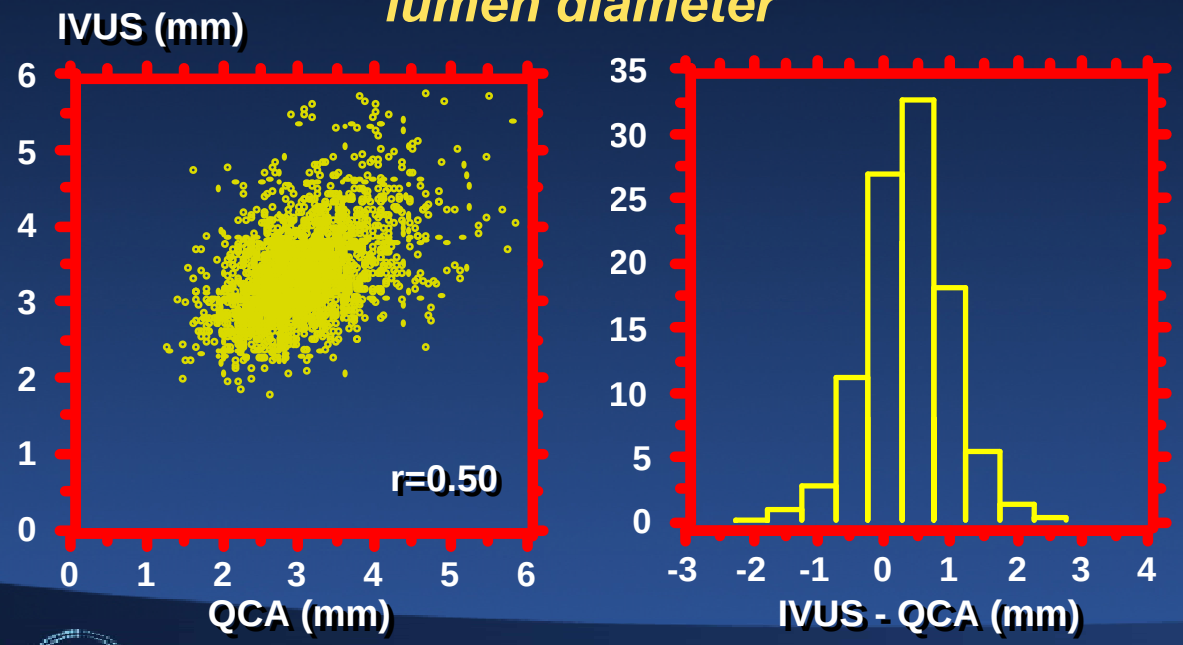
Kobayashi et al. Catheter Cardiovasc Interv 2001;52:208-11



Increasingly aggressive

- Largest reference lumen (prox or dist)
- Midwall
- Media-to-media (typically discounted)

IVUS vs QCA reference lumen diameter



IVUS vs QCA lesion length