How New Intravascular Imaging Techniques Will Change Clinical Practice in the Future

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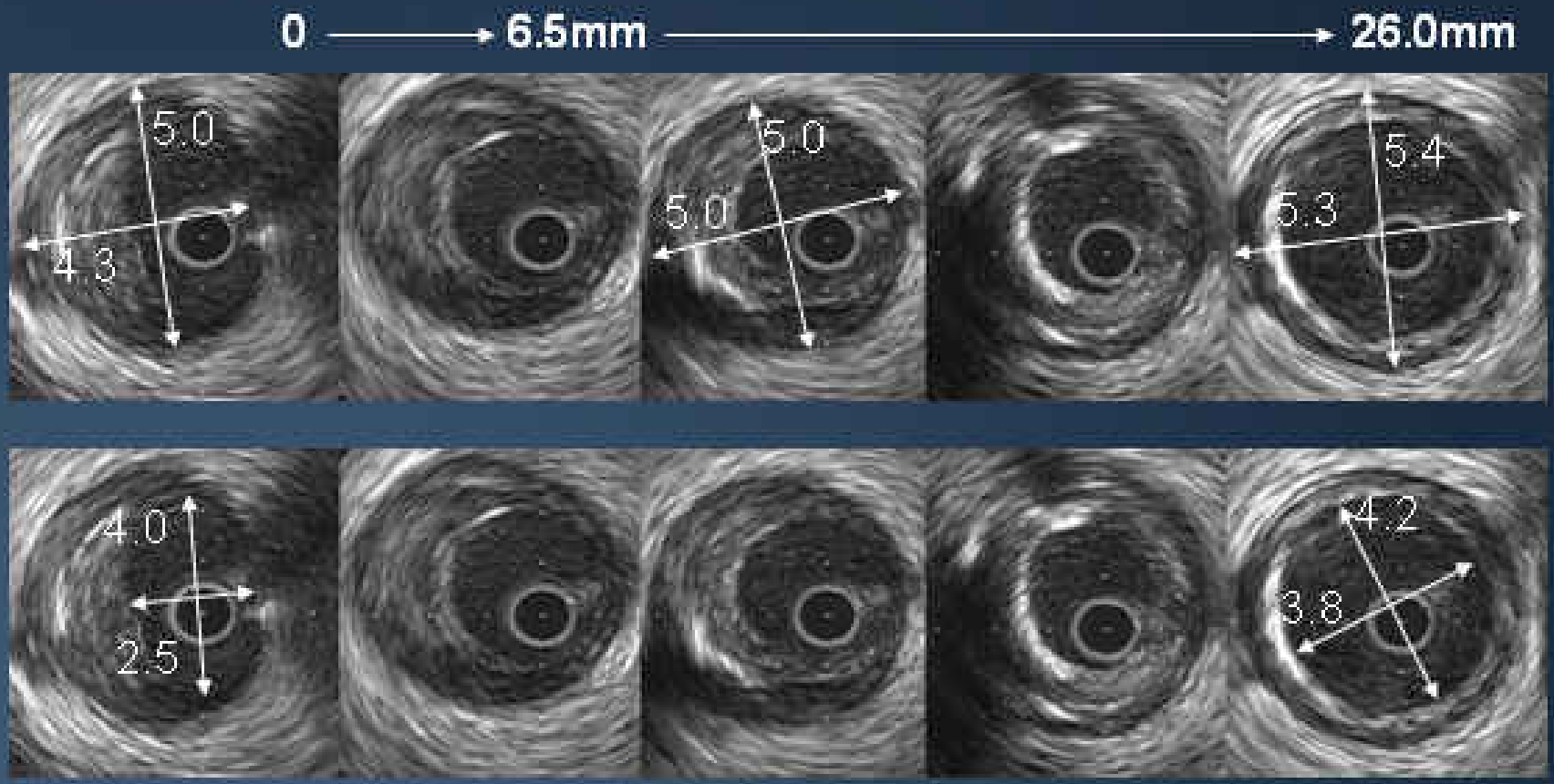
Greyscale IVUS

- Perhaps the best all around technique for assessing overall atherosclerosis burden and unusual lesions pre-intervention; guiding interventional procedures - including intermediate lesion and LM disease assessment; stent and length sizing and stent optimization; and assessing stents at follow-up
- Limited usefulness in assessing plaque composition (except calcium), thrombus formation, vulnerable plaques
- The workhorse in the cath lab

Will OCT replace IVUS? Three opinions

- Despite its high resolution and seductive images, OCT has limitations (penetration, true vessel sizing, assessment of plaque burden, etc) and really does not add ***important*** information. A good IVUS study provides all of the information needed to optimize the DES-PCI procedure. OCT only will have a niche role.
- (Almost) anything IVUS can do, OCT can do better. It is IVUS that will be relegated to a niche role (ostial lesions, large vessels, etc).
- There is simply not enough data or experience to say for sure.



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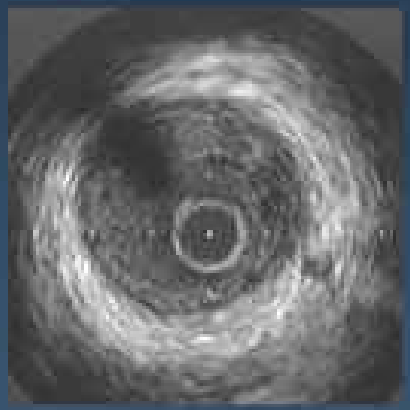
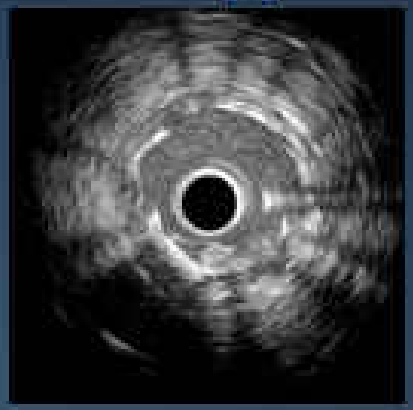
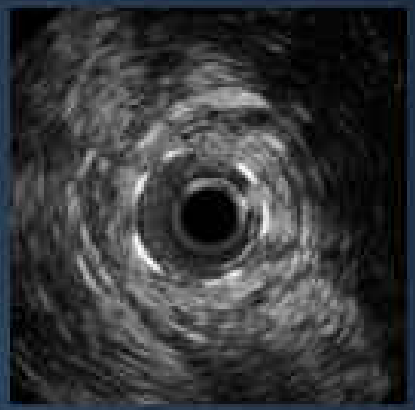
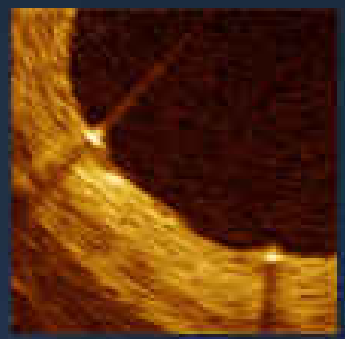
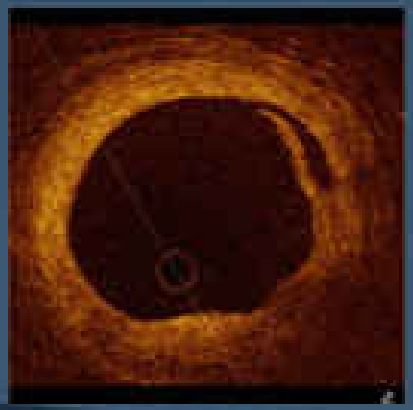
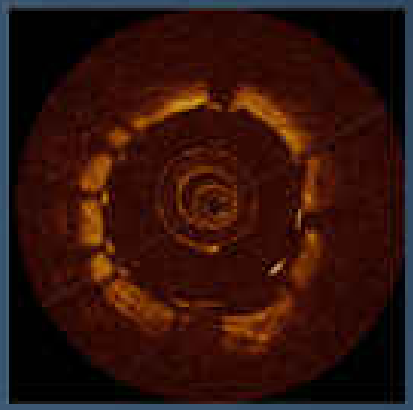
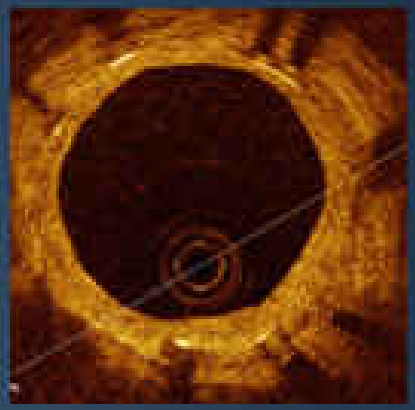
Predictors of DES Thrombosis & Restenosis

	DES Thrombosis	DES Restenosis
Underexpansion	•*Fujii et al. J Am Coll Cardiol 2005;45:995-8)* •*Okabe et al., Am J Cardiol. 2007;100:615-20* •*Liu et al. JACC Interventions, in press*	•*Sonoda et al. J Am Coll Cardiol 2004;43:1959-63* •*Hong et al. Eur Heart J 2006;27:1305-10* •*TAXUS IV, V, VI and ATLAS WH, LL, DS meta-analysis* •*Fujii et al. Circulation 2004;109:1085-1088* •*Sakurai et al. Am J Cardiol 2005;96:1251-3.*
Edge problems (geographic miss, secondary lesions, large plaque burden, etc)	•*Fujii et al. J Am Coll Cardiol 2005;45:995-8)* •*Okabe et al., Am J Cardiol. 2007;100:615-20* •*Liu et al. JACC Interventions, in press*	•*Liu et al.Am J Cardiol 2009;103:501-6* •*Costa et al, Am J Cardiol, 2008;101:1704-11*

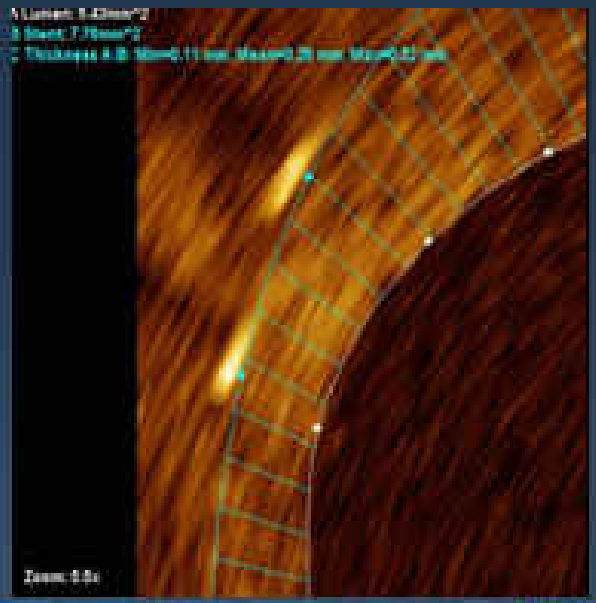
The superior resolution of OCT compared to IVUS only improves on the identification of small, clinically unimportant edge dissections, stent malapposition, etc.

Dissections **Stent Malapposition In-stent Restenosis**

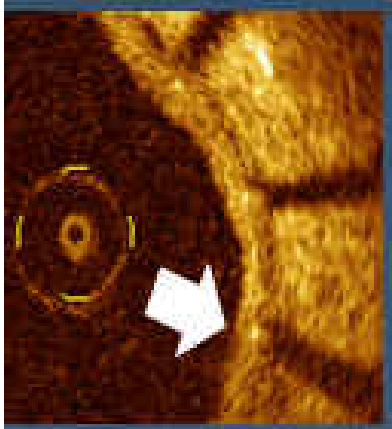








And re-endothelialization is still below the resolution of even OCT

Lumen Area, Stent Area, Strut-Lumen Distance



Strut-Vessel Wall Distance



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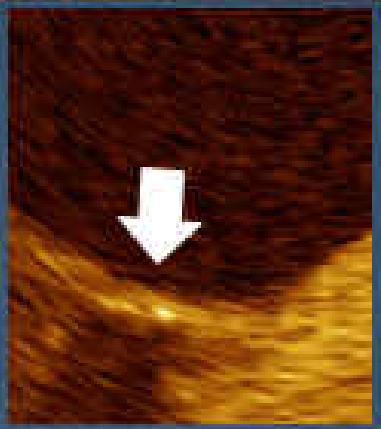
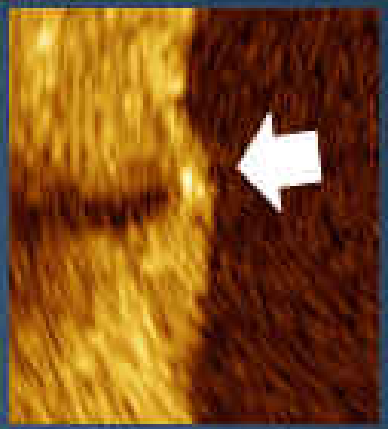
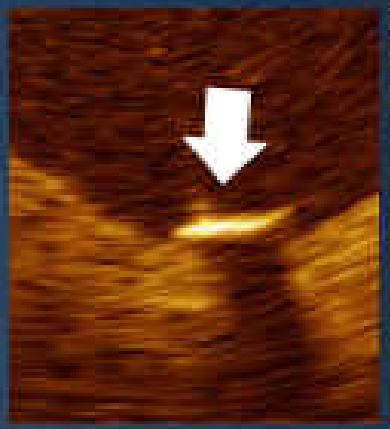
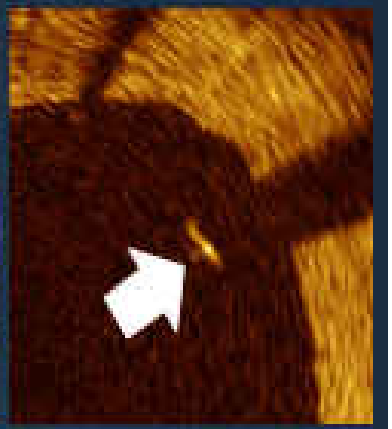




Protruding Covered



Protruding Uncovered



Malapposed

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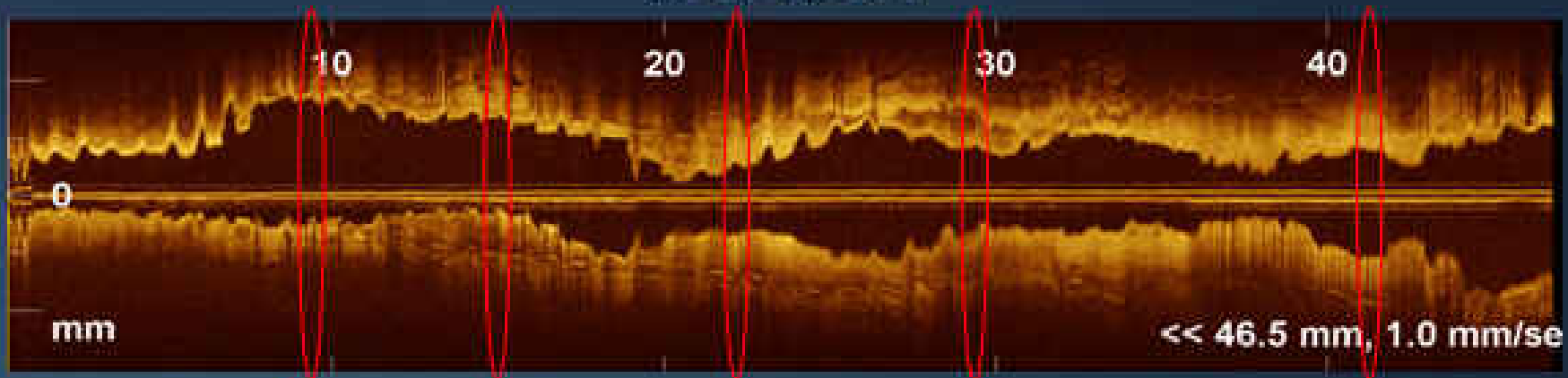
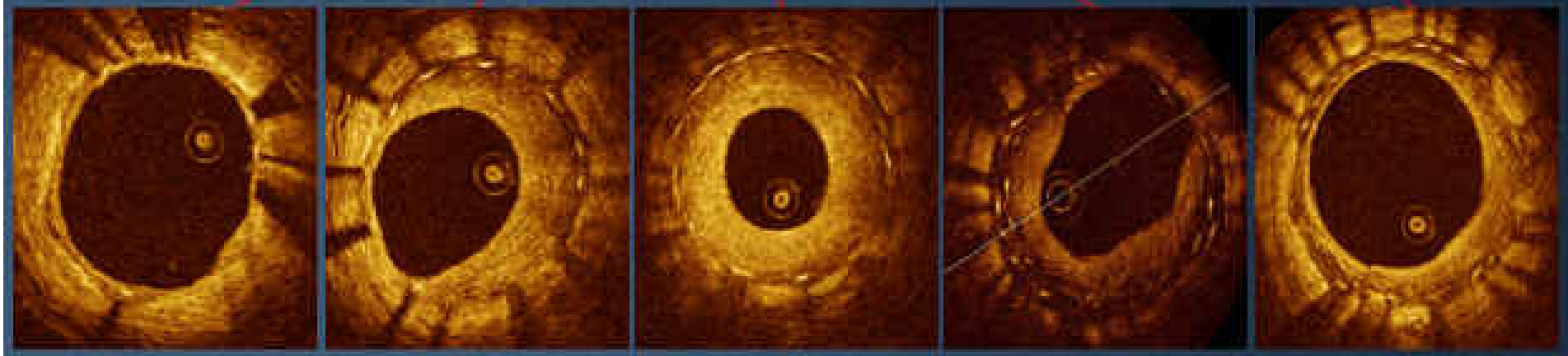
OCT Analysis in HORIZONS and ODESSA

Stents analyzed every 0.3 mm - 7,748 cross-sections or 43,884 struts in 117 pts in HORIZONS and 6,968 cross-sections or 53.047 struts in 77 pts in ODESSA





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Guagliumi et al TCT 2008 and AHA 2008

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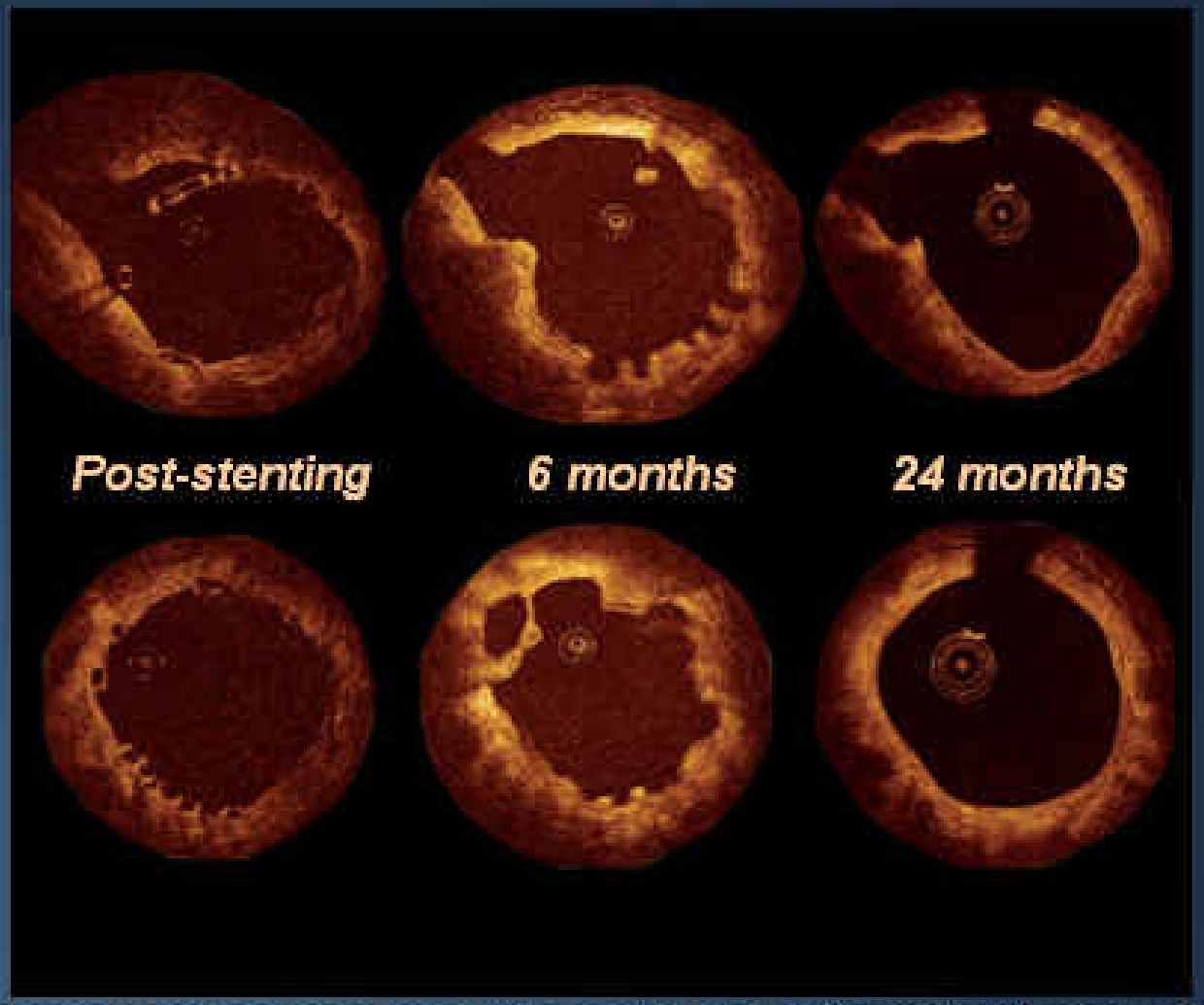
Serial assessment of bioabsorbable stent struts by OCT



At 2 years, there is smooth appearance of the endoluminal lining without strut malapposition since struts have been absorbed along with smooth endothelial lining with almost circular cross section and no longer discernible struts.

(Serruys et al. Lancet 2009;373:897–910)

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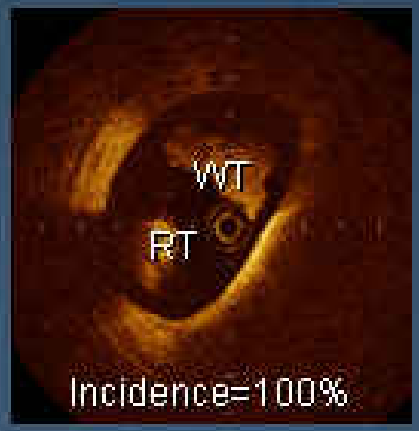
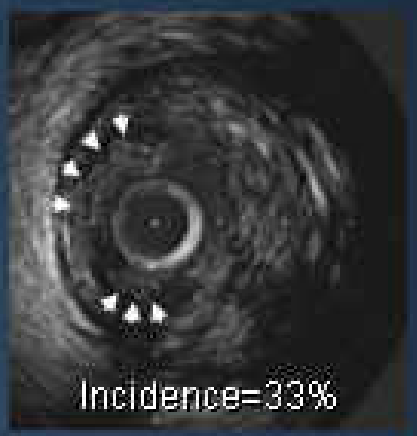
Why Will OCT Succeed?

- Interventionalists who have rejected IVUS (or who could/would never learn to interpret the IVUS images) will give intravascular imaging a second chance.
- The images are exquisite and seductive.
- OCT does provide additional information in specific situations . . .
- ?Follow-up imaging – especially in clinical trials – may belong to OCT
- *However, for OCT to replace IVUS as the workhorse in the cath lab, we will have to develop new paradigms for its use. . . just as we did when IVUS was "new."*

Angioscopy

- Perhaps the best technique for assessing thrombus
- Also useful for assessing stent neointimal coverage



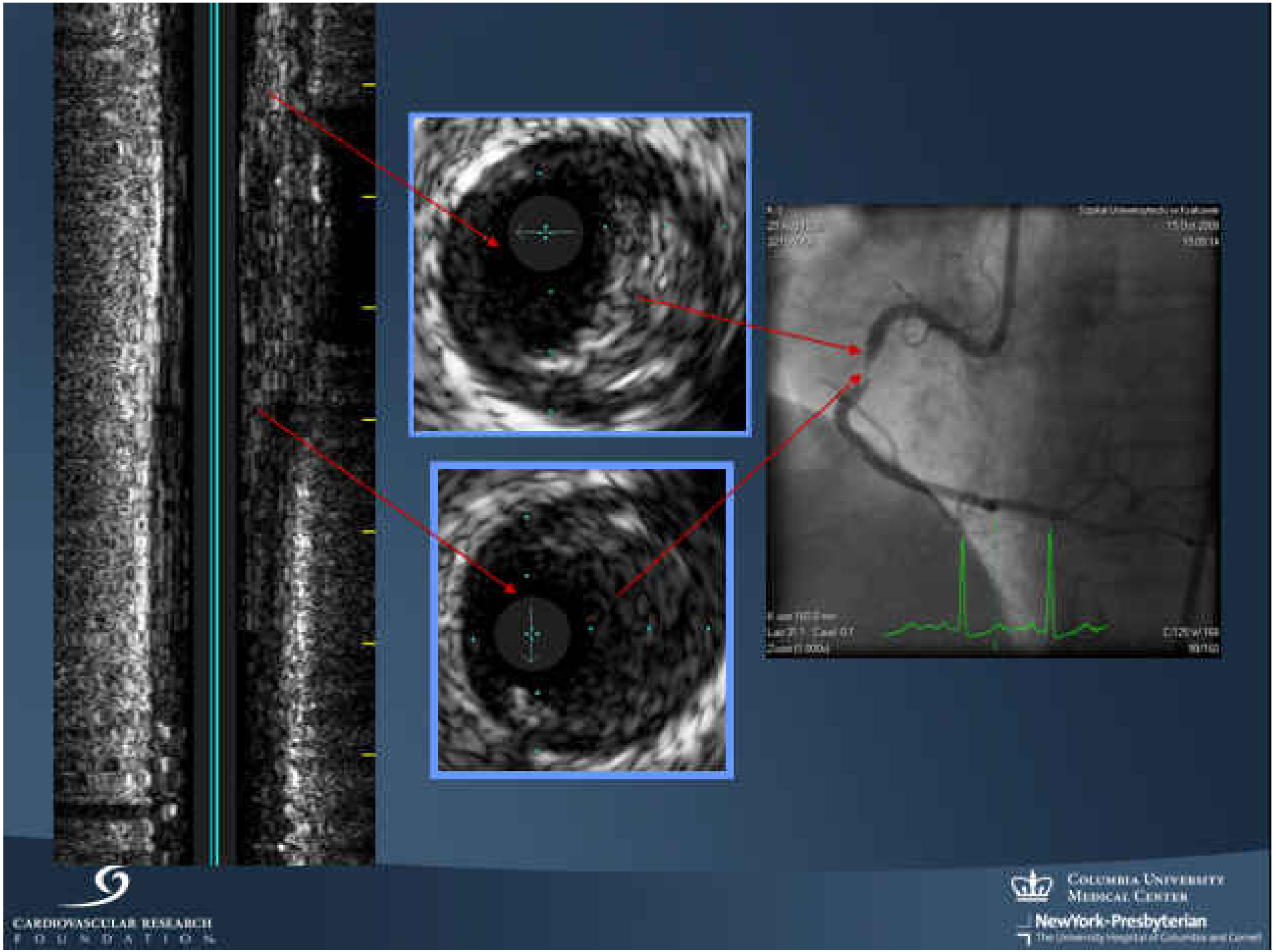


(Kubo et al. J Am Coll Cardiol 2007;50:933-9)
(Kubo et al. J Am Coll Cardiol 2007;50:933-9)

Recent angioscopy and OCT studies from Japan have shown the frequent presence of thrombi within DES at follow-up. What does this mean?

		Imaging Modality	SES	PES
Awata, et al	Circulation 2008;118:S897	Angioscopy	26% (n=30)	57% (n=19)
Hara, et al	Circulation 2008;118:S735	Angioscopy	12% (n=42)	46% (n=37)
Otake, et al	Circulation 2008;118:S896	OCT	18% (n=35)	
Ozaki et al	Circulation J 2009;73:Suppl 1-387	Angioscopy	42% (n=20)	72% (n=18)
Uemura	Circulation J 2009;73:Suppl 1-27	OCT	6.3% (n=48)	

What about vulnerable plaques?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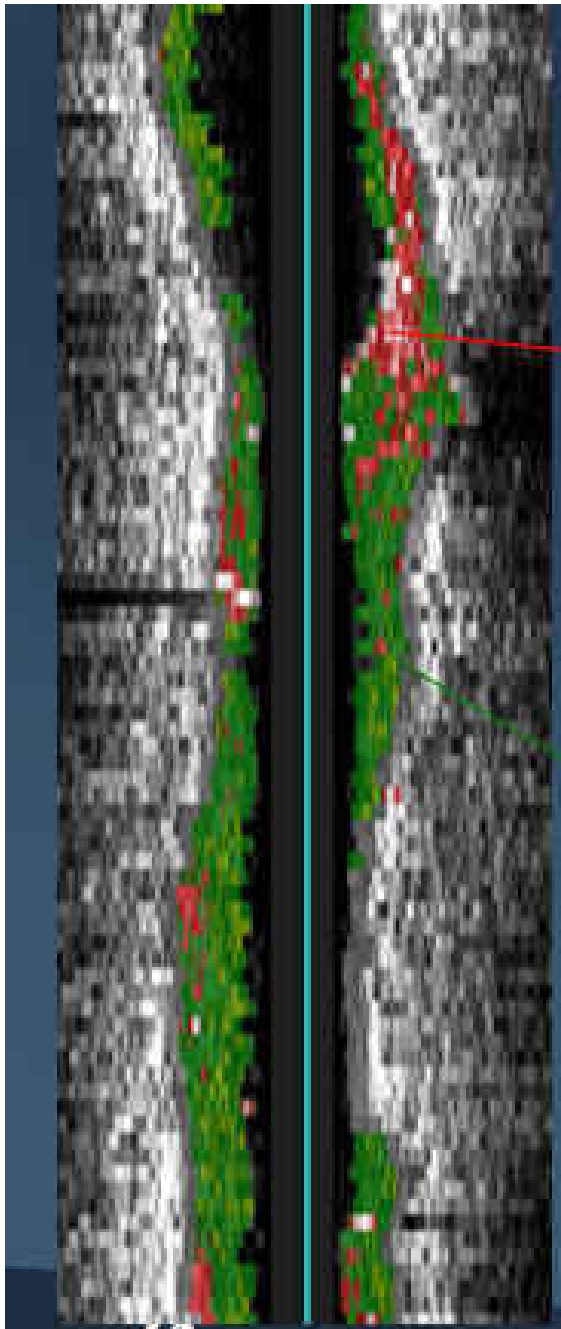
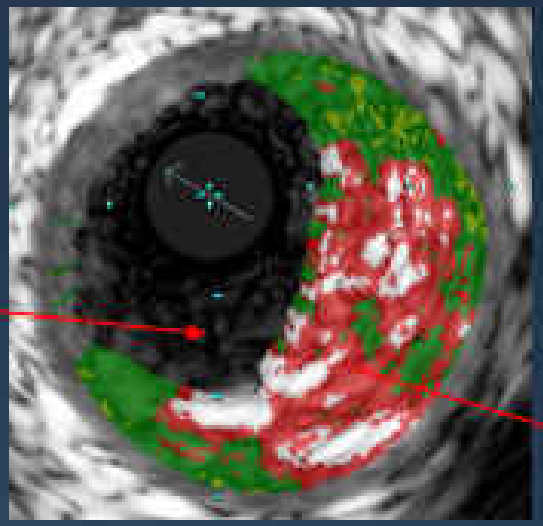
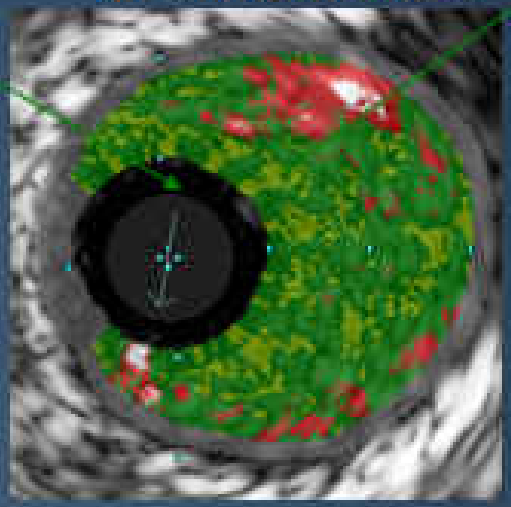
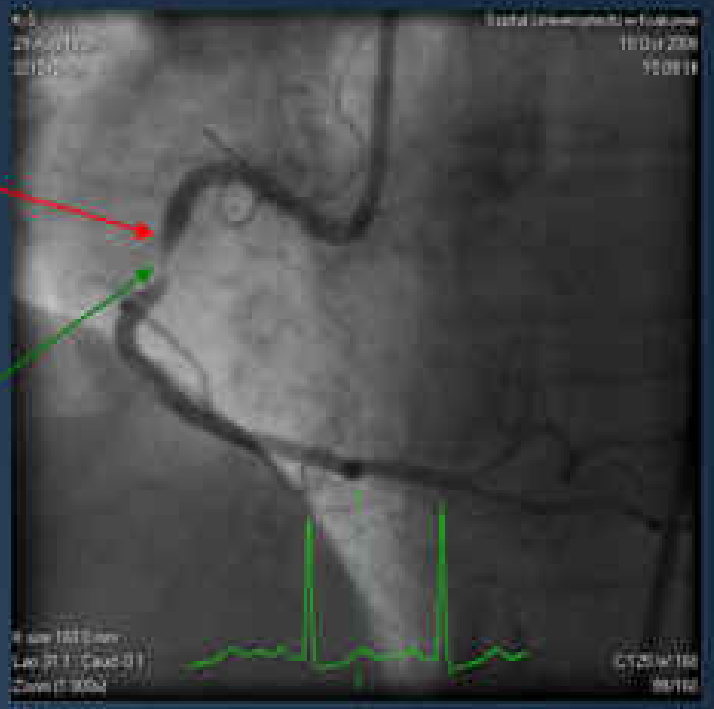




culprit of the culprit
proximal to MLA



MLA



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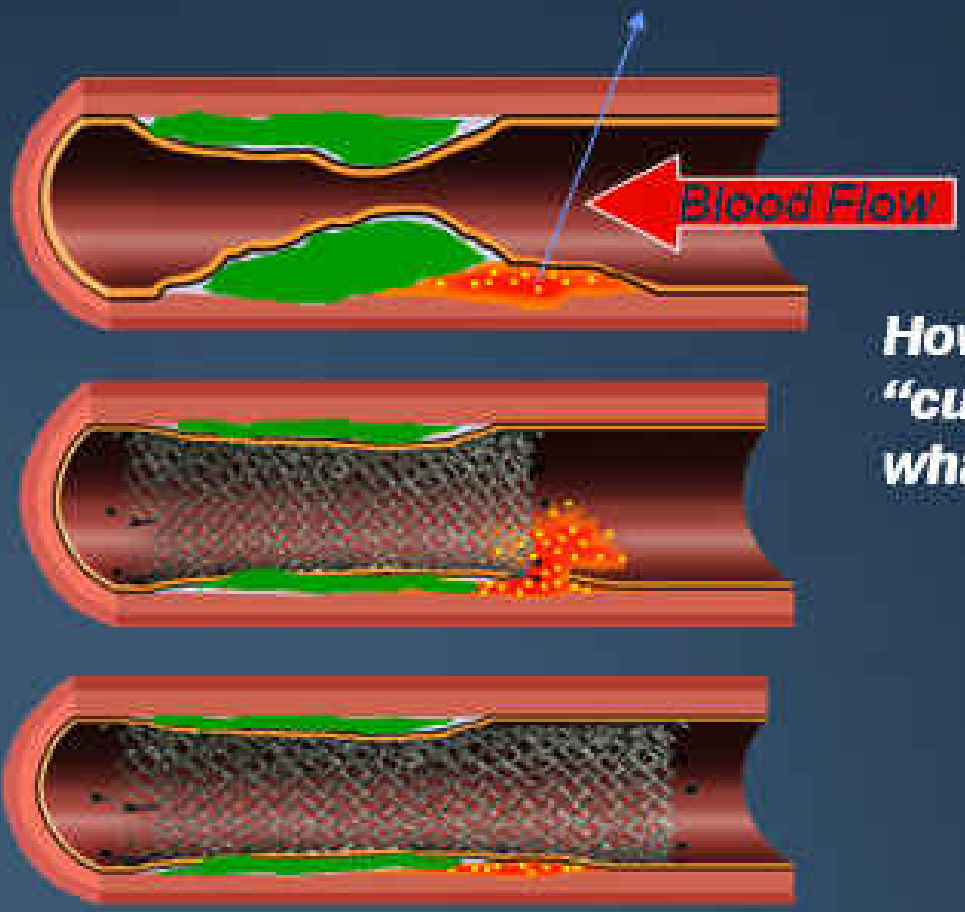
Possible Stent Positioning in Culprit Lesion PCI

NC, the "culprit of the culprit"



How often do we miss the "culprit of the culprit"? And what is the impact on

- **Distal embolization**
- **Stent thrombosis**
- **Restenosis**
- **Plaque progression**

444 ACS culprit lesions entirely imaged using post-stent VH-IVUS (The remaining patients were excluded because of incomplete imaging of stented lesion or unreliable pullback.)

TCFA behind stent (n=259)

No TCFA behind stent (n=185)

- No NC at all
- ThFA behind Stent
- Non-classifiable (Stent artifact)

TCFA behind stent with reference TCFA that was fully or partially uncovered (n=98)

Edge TCFA partially uncovered (n=20)

TCFA behind stent with fully uncovered reference TCFA (n=78)

TCFA only behind stent (n=161)

No TCFA behind stent with fully uncovered reference TCFA (n=33)

No TCFA behind stent w/o reference TCFA (n=152)

NC protruding through stent (n=63)

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Tsujita et al, AHA 2008

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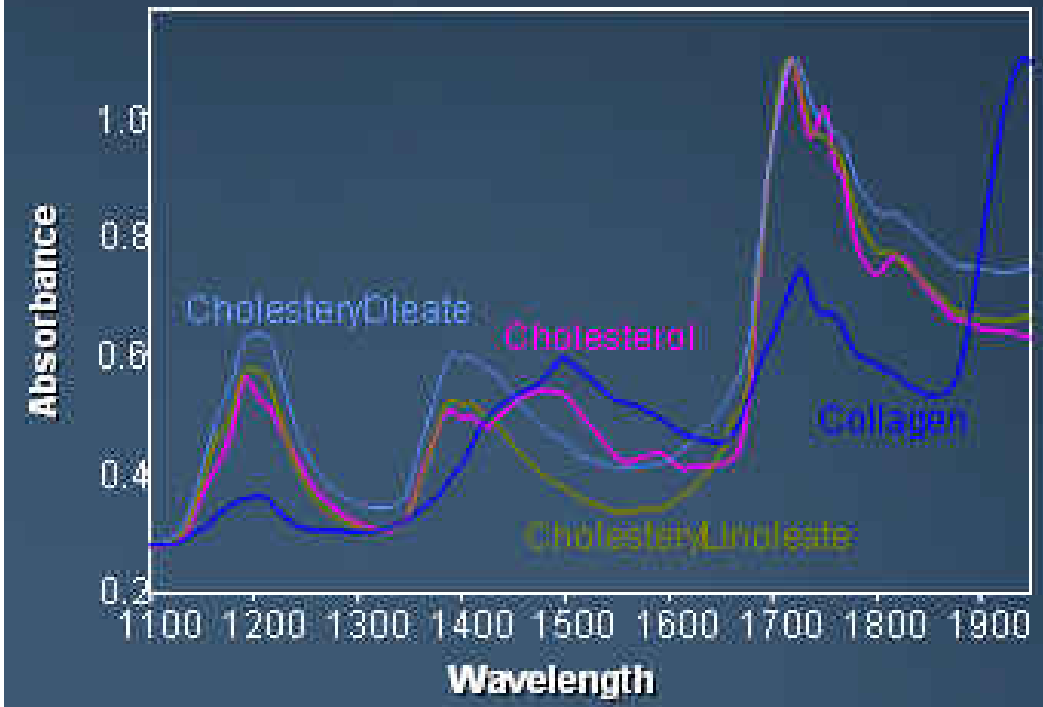
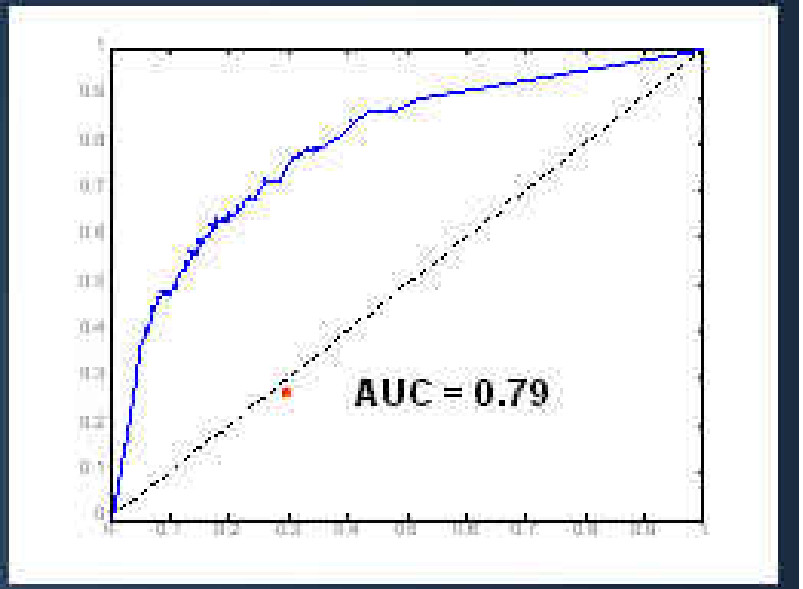
Numerous studies have shown a relationship between the maximum necrotic core and post-PCI distal embolization

- Kawaguchi et al. J Am Coll Cardiol. 2007;50:1641-6
  - ST re-elevation in 71 pts with STEMI
- Kawamoto et al. J Am Coll Cardiol. 2007;50:1635-40
  - Doppler FloWire high intensity transit signals in 44 pts undergoing elective stenting resulting in poor recovery of CVFR
- Park et al. VH Summit 2007 (unpublished)
  - Largest NC independent predictor of CK-MB release (n=332)
- Washington Hospital Center. Unpublished
  - Troponin post elective stenting
- Bose et al. Basic Res Cardiol 2008;103:587-97
  - CK and TnI in 55 pts undergoing direct stenting. Patients in the 4th quartile of NC volume had a particularly high increase in biomarkers.
- Higashikuni et al. Circ J 2008; 72: 1235-41
  - No reflow in 49 pts with ACS undergoing PCI
- Hong et al. Eur Heart J, in press
  - No reflow in 190 pts with ACS undergoing stenting

What about vulnerable plaques?

NIR Spectroscopy can identify the chemical composition of unknown substances and distinguish cholesterol from collagen. ROC Analysis of Validation of NIR Spectroscopy in 51 Autopsy Hearts (algorithm for detection of confluent [>0.2mm thick and >60°] and relatively superficial necrotic core [overlying mean fibrous cap thickness <0.45microns])





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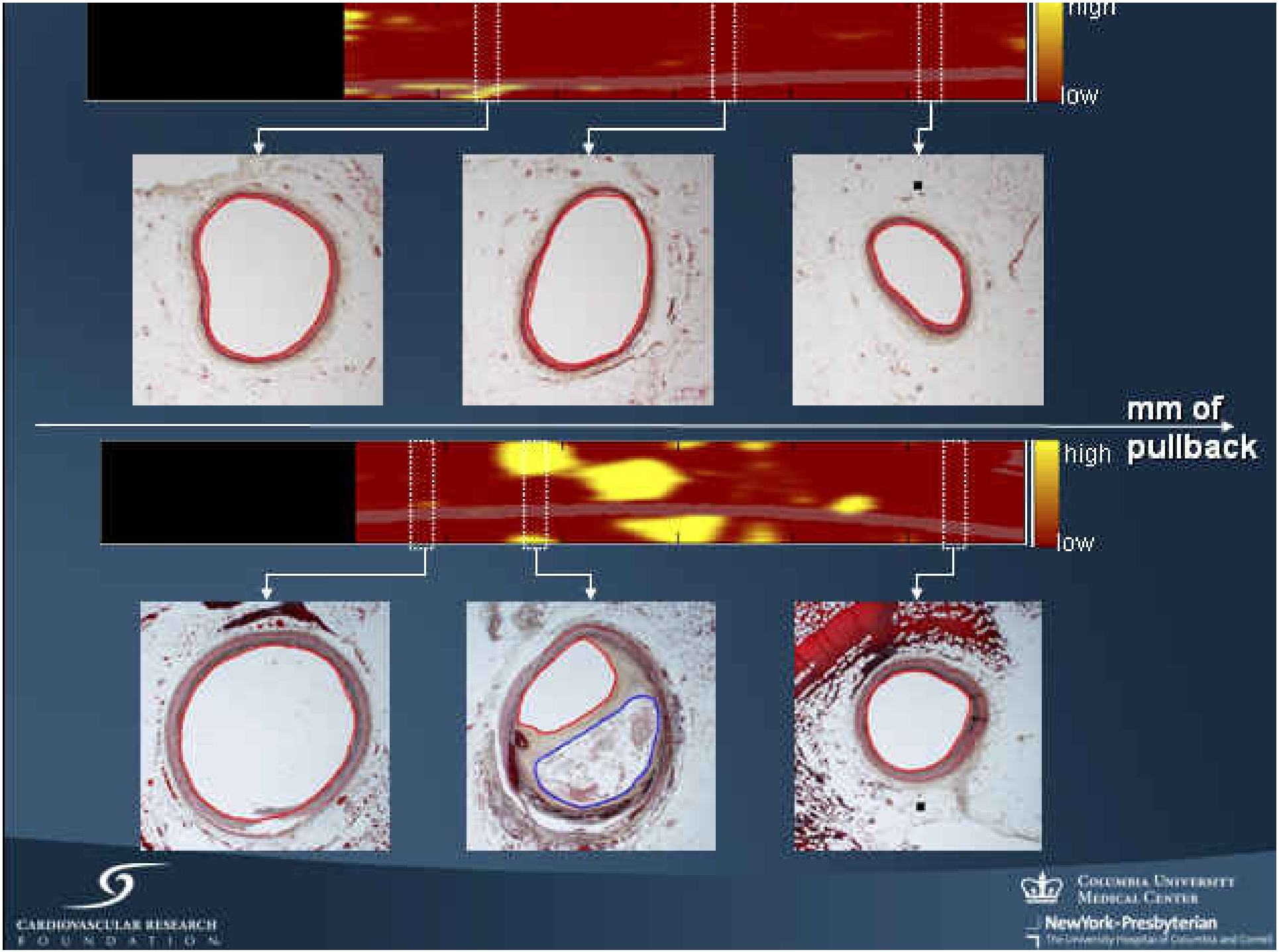
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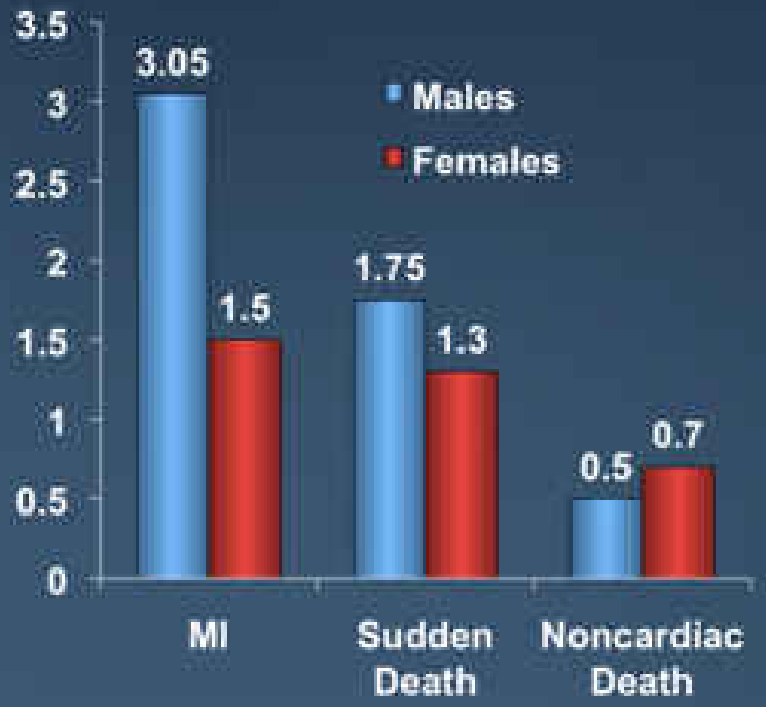
Number of thin-cap fibroatheromas in patients dying with MI, sudden death, or noncardiac causes and studied at necropsy

Cross-sectional analysis



Longitudinal analysis

	All pts	Pts with ≥1 ruptured plaque	Pts with ≥1 TCFA or ruptured plaque	Pts with CV death
# of patients	50	14	20	33
# of ruptured plaques	19 (0.38/pt)		19 (0.95/pt)	15 (0.45/pt)
#fibroatheromas	193			
# TCFAs	23 (0.46/pt)	15 (1.21/pt)	23 (1.15/pt)	18 (0.55/pt)

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(Burke et al. J Am Coll Cardiol 2003;41:1874-86)
(Cheruvu et al. J Am Coll Cardiol 2007;50:940-9)

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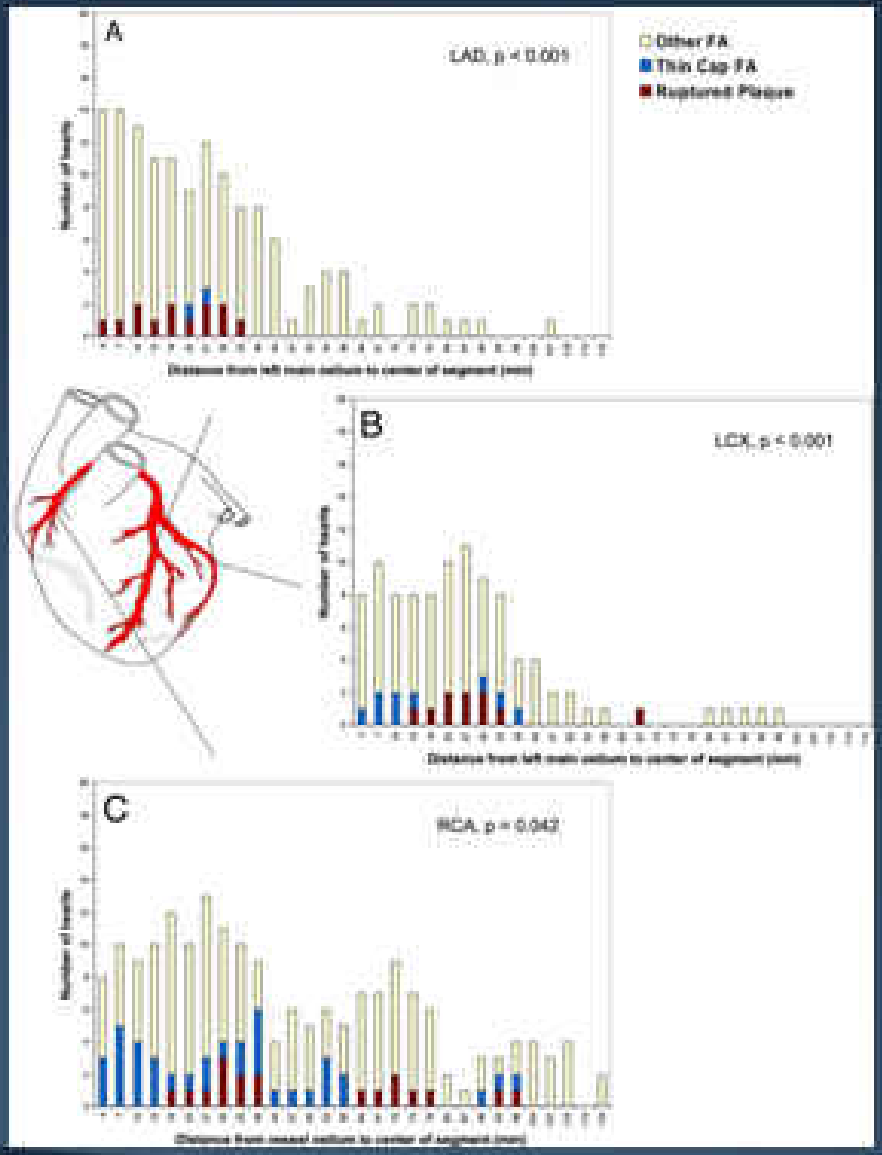
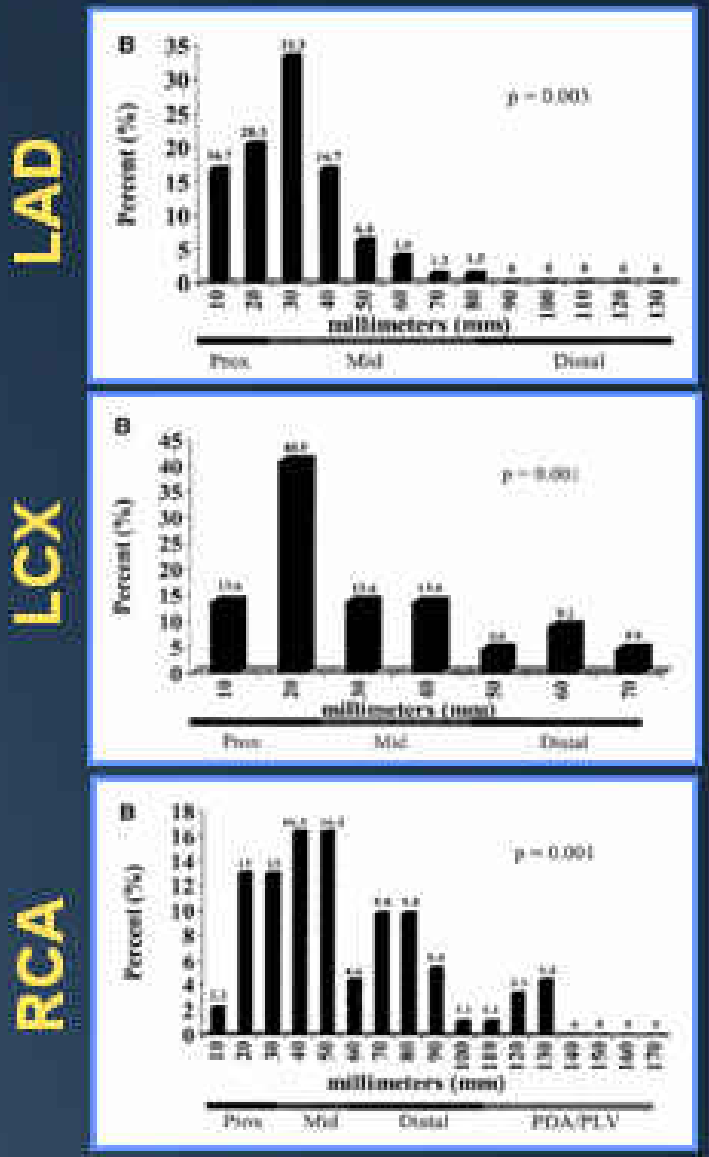
Pathology spatial Distribution of Advanced Coronary Lesions



Angiographic location of acute coronary occlusions



(Cheruvu et al. J Am Coll Cardiol 2007;50:940-9)

(Wang et al. Circulation 2004;110:278-84)

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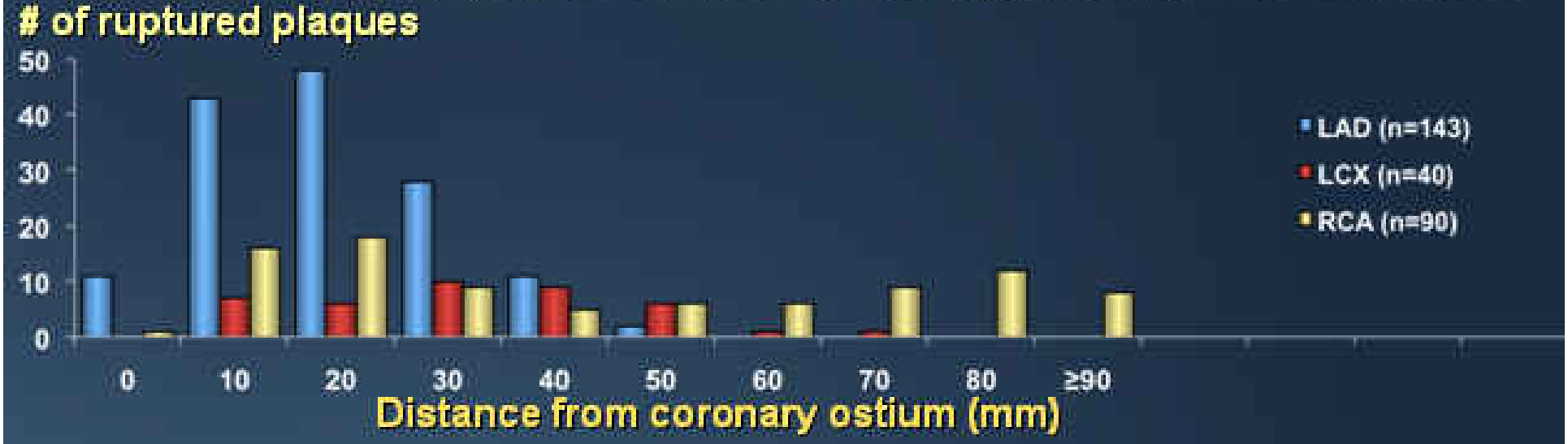
Location of 273 ruptured plaques in 158 pts with ACS and 48 pts with stable angina and 3-vessel IVUS



Location of 82 TCFAs in 34 pts with AMI and 17 pts with stable angina and 3-vessel OCT

(Hong et al J Am Coll Card 2005;46:261-5)
(Fujii et al J Am Coll Cardiol. 2008;52:787-8)

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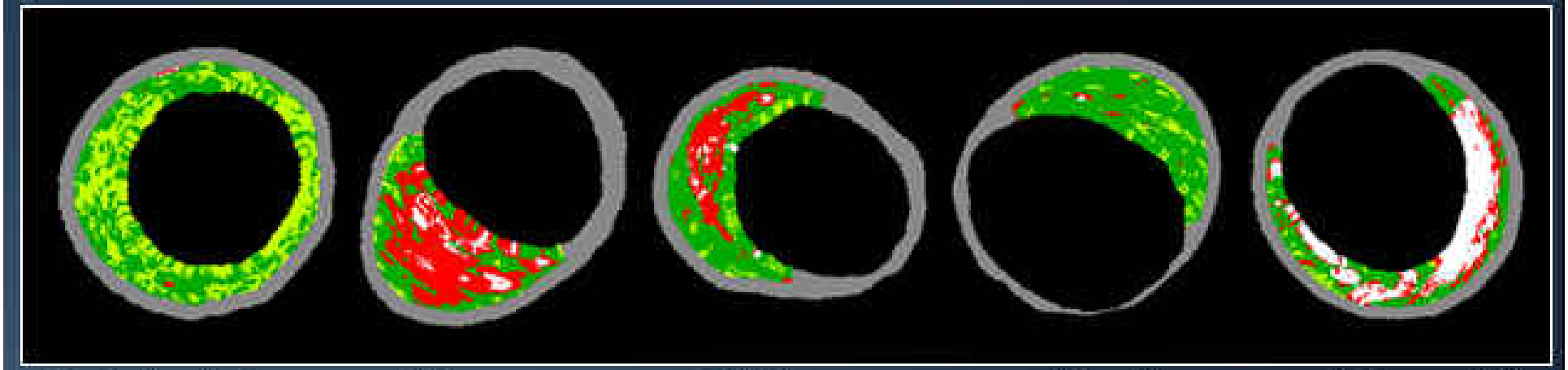
	IVUS	IB/VH-IVUS	Palpography	VV	OCT	NIR	Angioscopy
Major criteria							
Active inflammation			+		+		
Thin cap			?		+	+	
Large lipid/necrotic core		+			+	+	
Endothelial denudation							
Fissured plaque							
Plaque burden >90%	+	+					
Minor criteria							
Superficial calcific nodule	+	+			+		
Glistening yellow							+
Intraplaque hemorrhage or neovascularization				+			
Endothelial dysfunction							
Positive remodeling	+	+					

- Who should be studied?
  - All patients or just high risk paients?
  - Primary vs secondary prevention?
  - All arteries or just the PCI artery?
- What is the risk of multivessel invasive imaging?
- What is the cost?
- What is the impact of modern medical therapy: ASA, clopidogrel, statin?
- What is the temporal stability of TCFAs
  - How quickly do they form?
  - How often do they heal spontaneously?
  - How often do they rupture without causing events?

Change in non-culprit lesion phenotype in 106 pts (201 lesions) with plaque burden >40%) from the Global VH Registry with baseline and 8-month F/U



Pathological intimal thickening (PIT)

Thin-cap fibroatheroma (TCFA)

Thick-cap fibroatheroma (ThFA)

Fibrotic

Fibrocalcific

75% of TCFAs healed, 25% remained unchanged (mostly proximal in location), and 12 new TCFAs were noted; 6 late-developing TCFAs were PIT and 6 were ThFA at baseline.

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Kubo et al. AHA 2008

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The right tool for the right job

Ideal

Reality

In clinical medicine when faced with a diagnostic or therapeutic decision, we must always choose the right tool for the right job. Invasive imaging is no different. None of the available tools is a single, all-in-one solution. All of them are important in specific situations. Physicians must learn to interpret and use these techniques correctly. The issues with IVUS – time, cost, expertise, etc – apply to all of the new modalities.