

Beyond IVUS: OCT, NIRS, and Beyond

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Disclosure Statement of Financial Interest

Within the past 12 months, I or my spouse/partner have had a financial interest/arrangement or affiliation with the organization(s) listed below.

Affiliation/Financial Relationship

- Grant/Research Support
- Consulting Fees/Honoraria
- Speaker Fee

Company

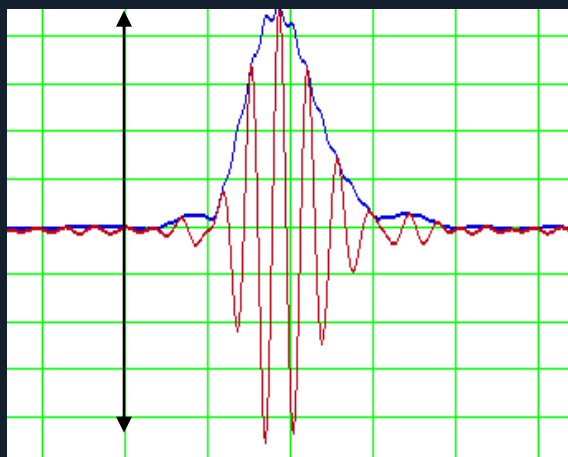
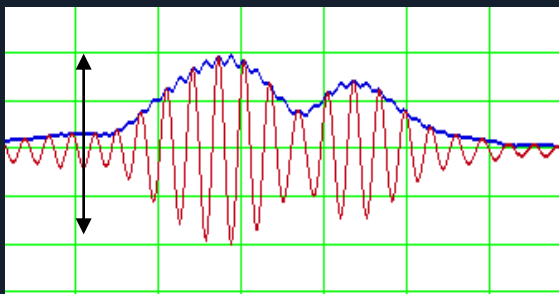
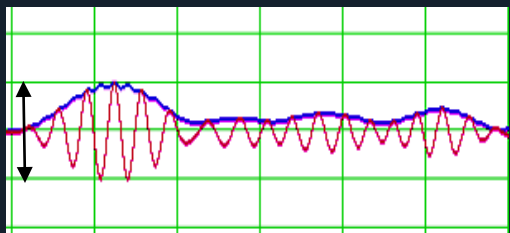
- Boston Scientific Corporation
- Boston Scientific Corporation, ACIST
- Volcano Corporation, St Jude Medical

Old Friend, Grayscale IVUS



Gray Scale IVUS Tissue Characterization

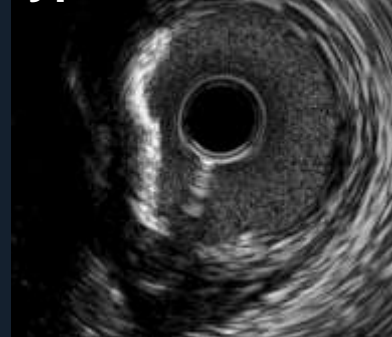
Ultrasound Wave



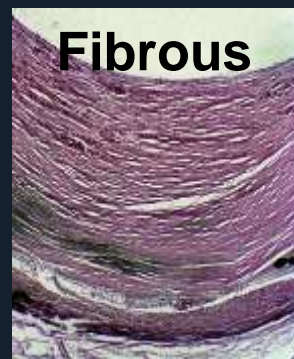
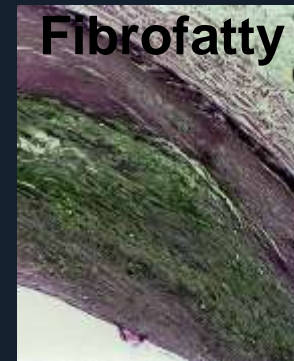
Grey Scale



Hyper with Shadow



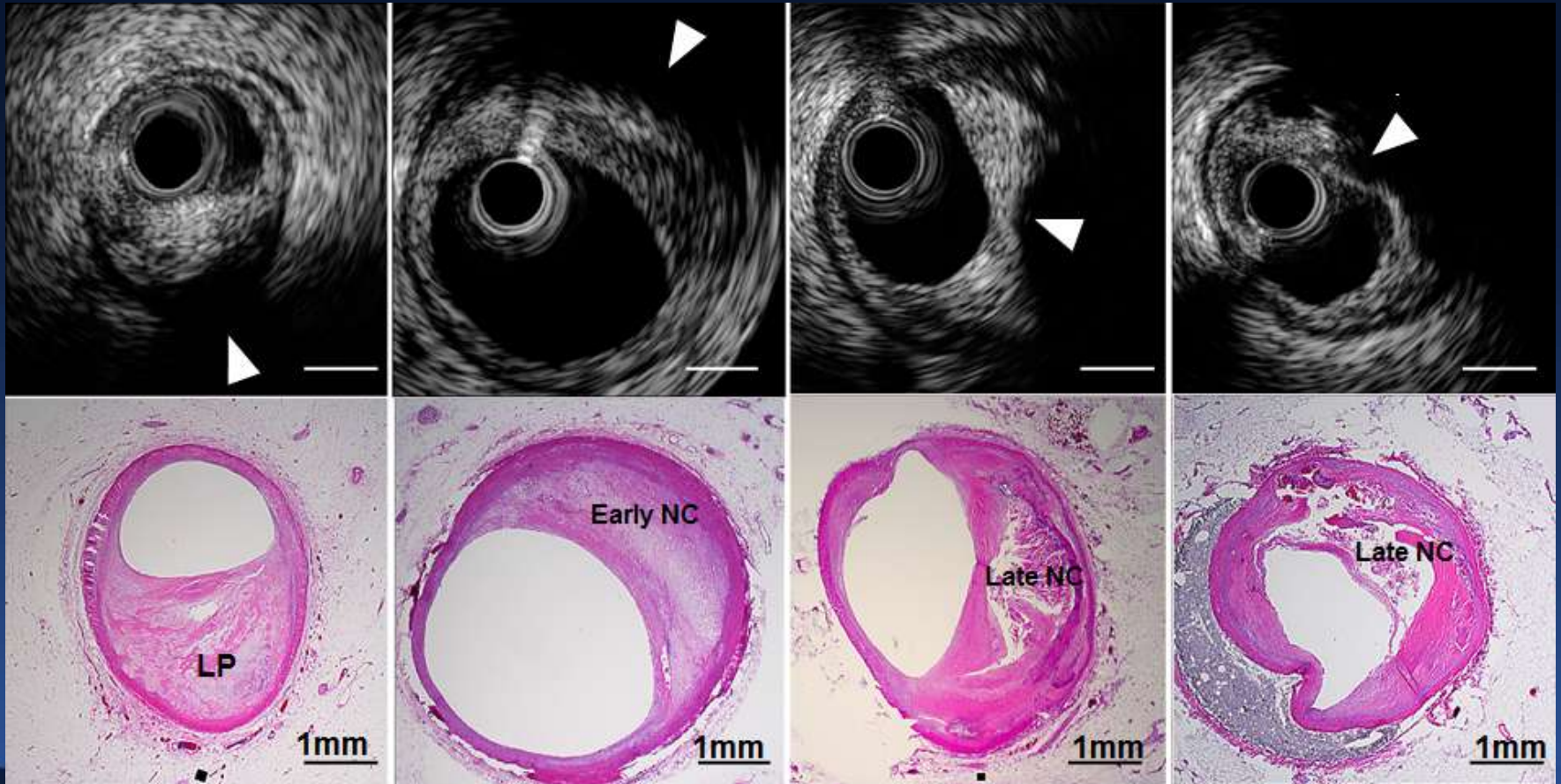
Pathology



Attenuated Plaque (Superficial, Deep)

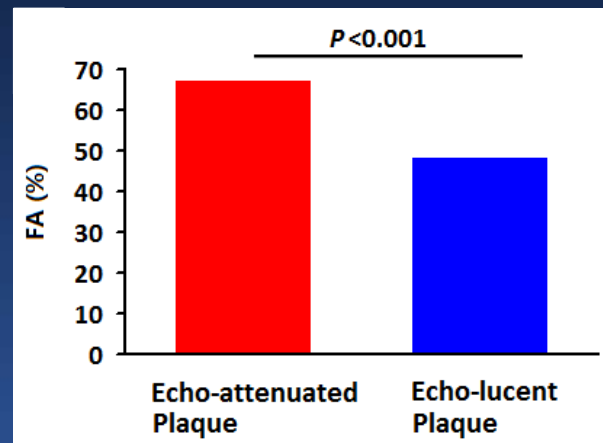
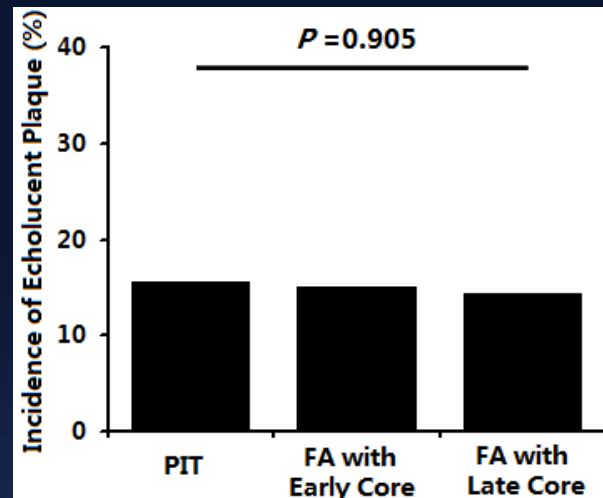
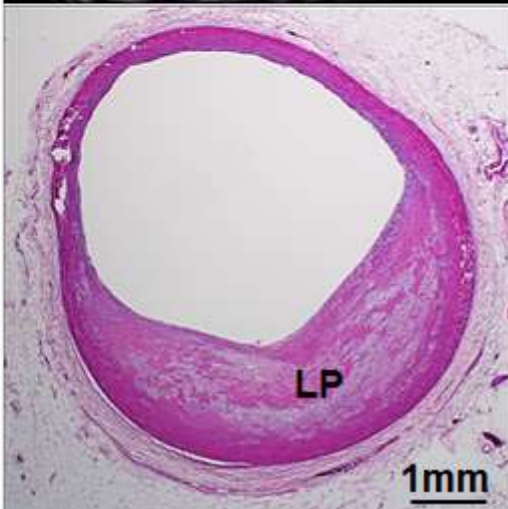
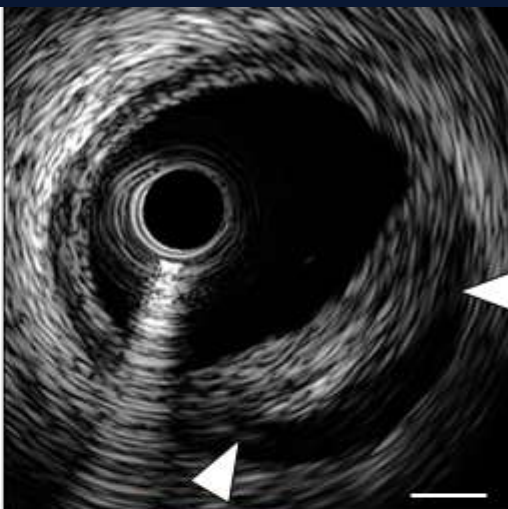
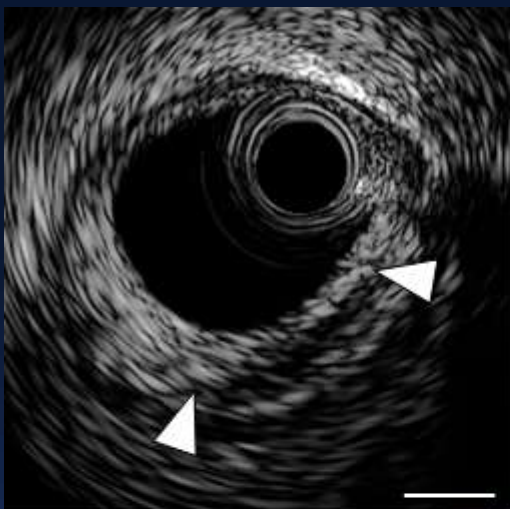
- 2,292 2-mm long segments from 151 coronary specimens in 62 autopsy hearts.
- Data obtained in the CDEV3 Study, Gardner et al, JACC Imaging, 2008, sponsored by InfraReDx, Inc.

Deep Attenuated Plaque Superficial Attenuated Plaque

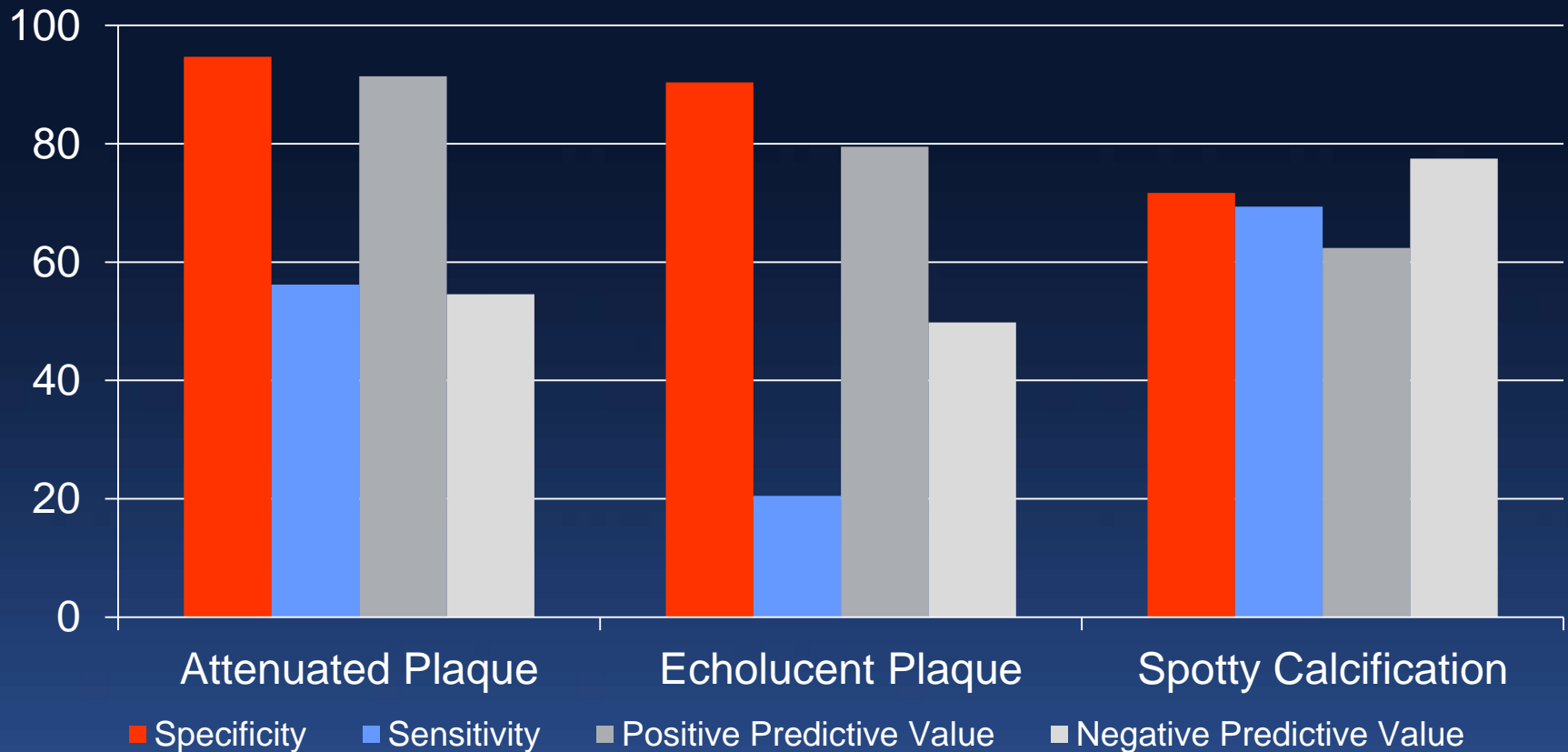


Pu J, et al. JACC *in press*

Echolucent Plaque (Superficial, Deep)

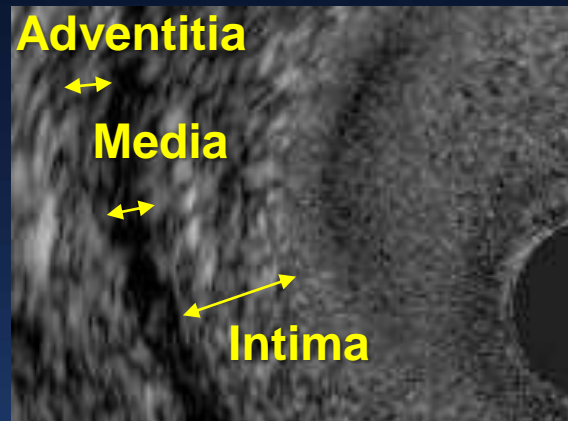
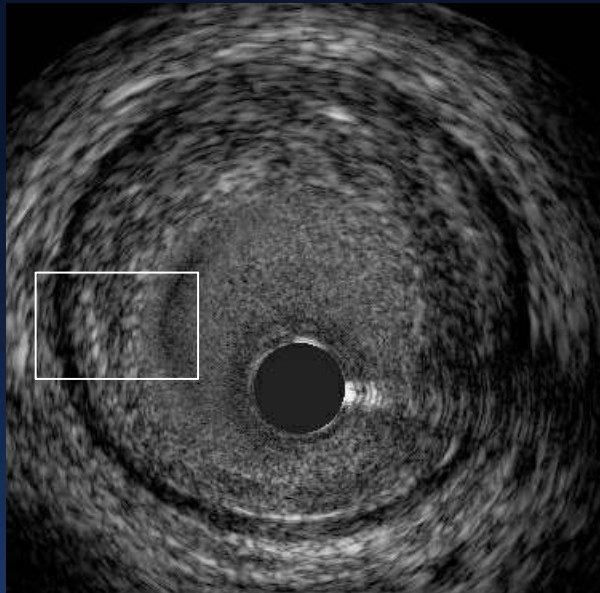


Diagnostic Summary of Large Necrotic Core/Lipid Pool



New Generation High Frequency IVUS

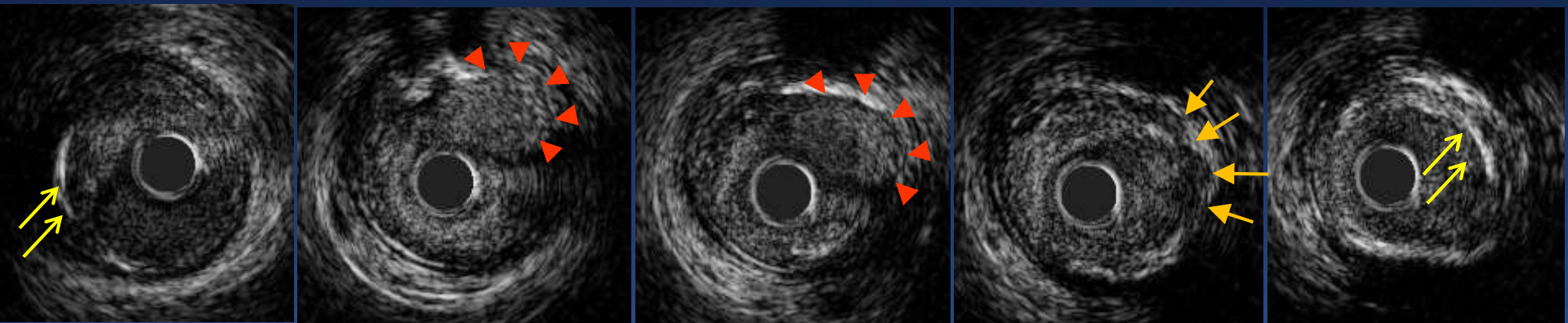
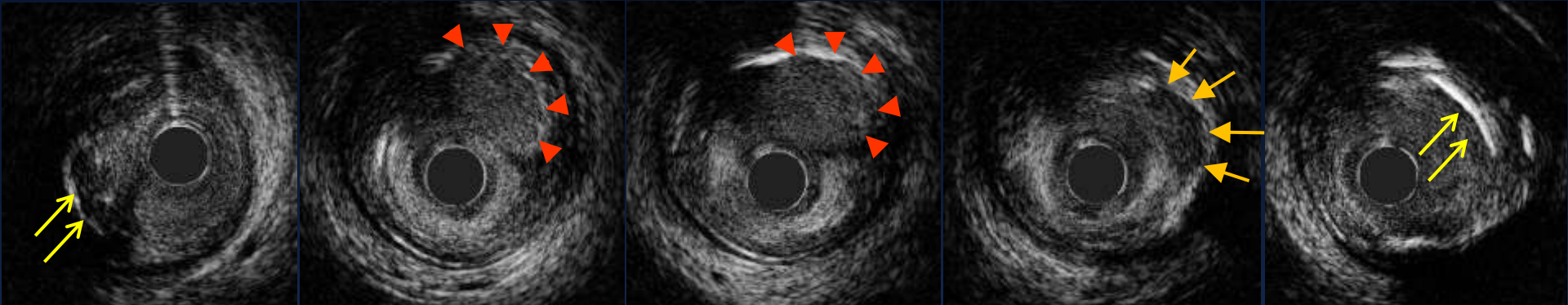
Three Layers Appearance



Axial Resolution: 40 μ m

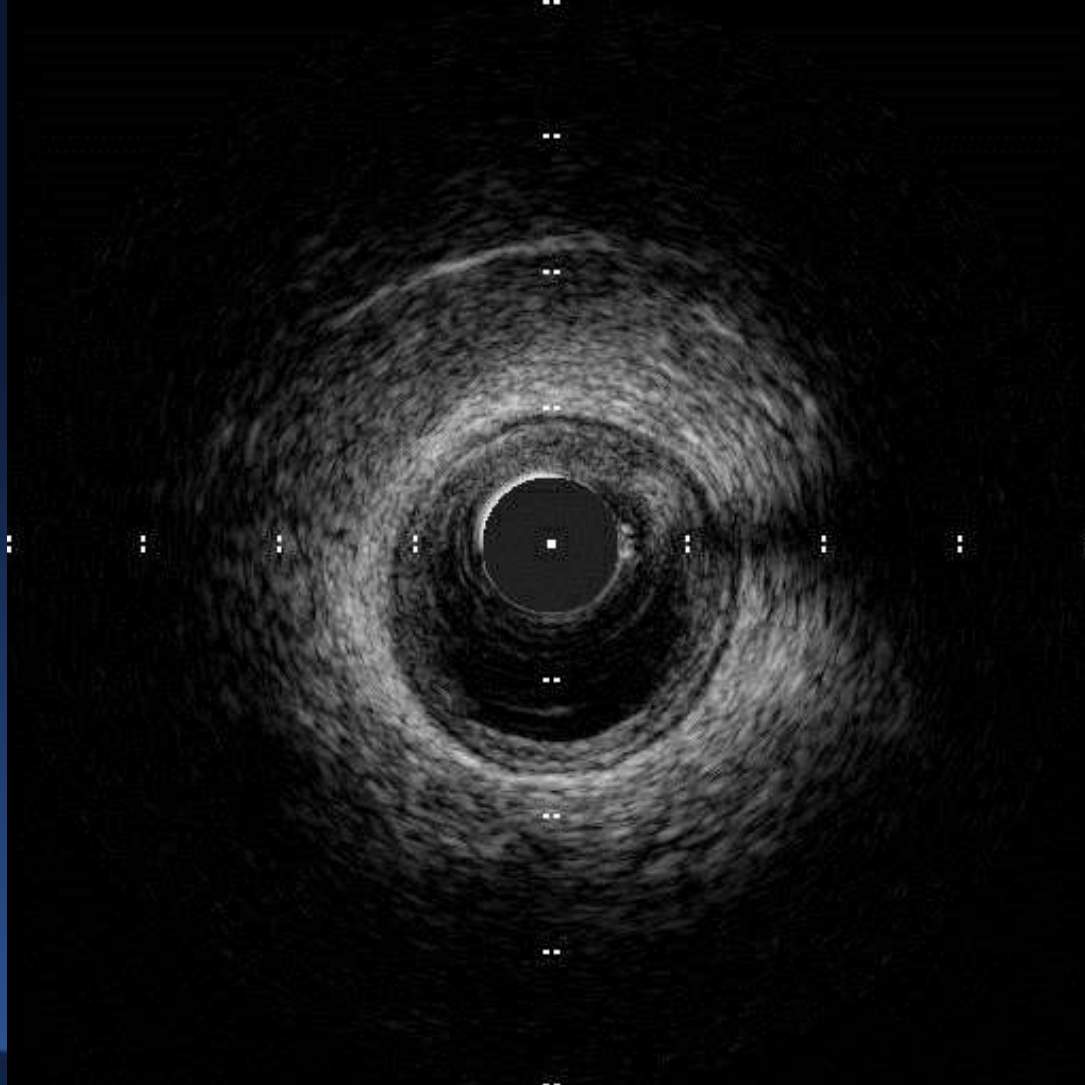
Ruptured Plaque

60MHz



40MHz

High Speed Pullback with Flush



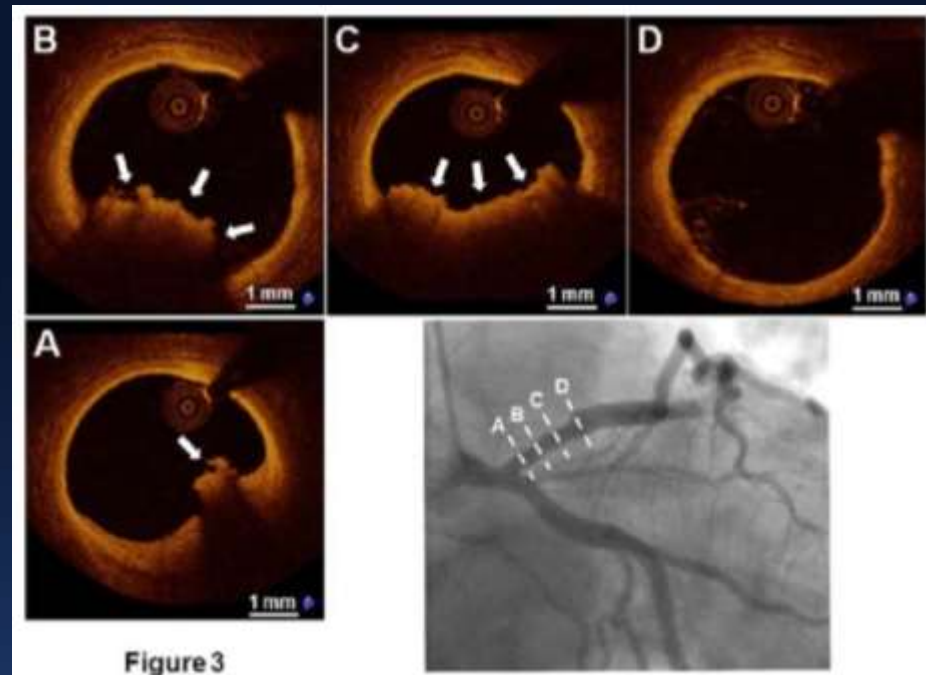
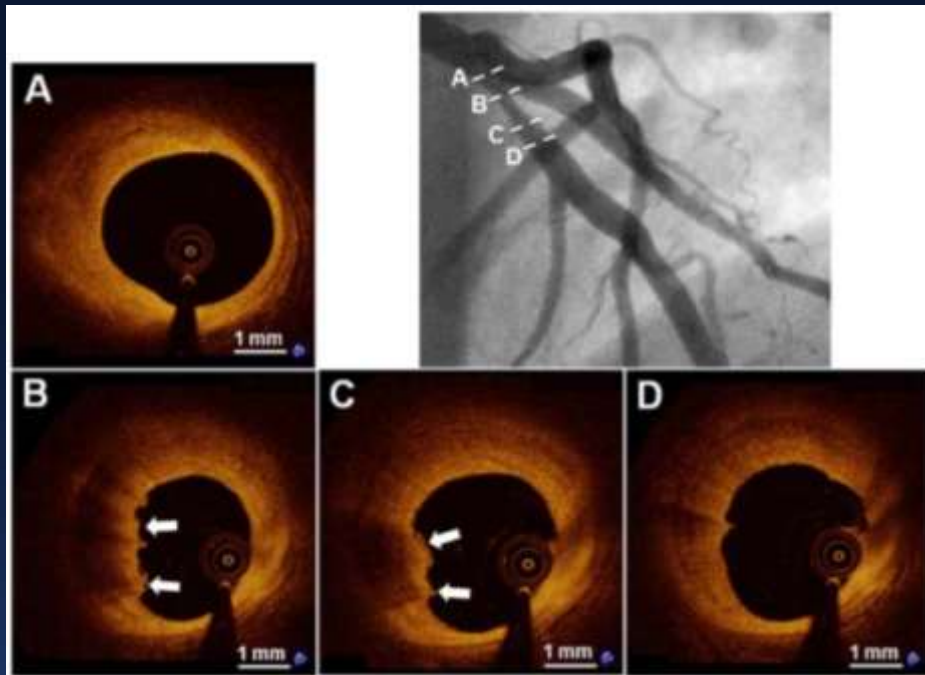
New Friend, OCT



MGH Multicenter OCT Registry (n=126)

Definite OCT-Erosion

Probable OCT-Erosion



Presence of attached thrombus overlying an intact and visualized plaque

- 1) Luminal surface irregularity without thrombus
- 2) Attenuation of underlying plaque by thrombus without superficial lipid or calcification immediately proximal or distal site

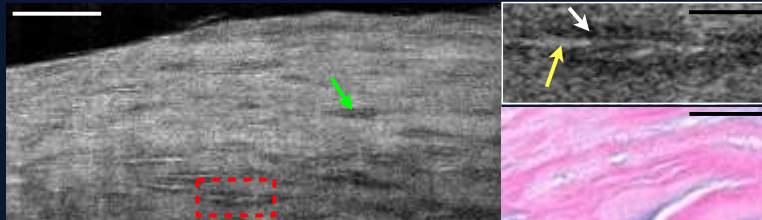
Multicenter OCT Registry

At median follow-up of 753 days, only one TLR with rupture.

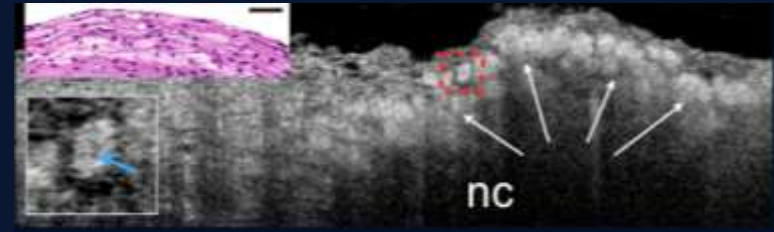
	Intact Fibrous Cap (n=12)	Ruptured Fibrous Cap (n=19)	P-value
Treatment	Thrombectomy only	Thrombectomy + Stent	
Age	52.2±12.0	62.6±10.8	0.019
Male	10 (83%)	16 (84%)	NA
Hypertension	1 (8%)	10 (53%)	0.033
Pre DS (%)	79±33	88±17	0.95
Post Aspiration DS (%)	27±19	32±35	0.48
Total Ischemic time (h)	3.5±3.0	3.6±2.3	0.82

μ OCT with <1 -2 Micron Resolution

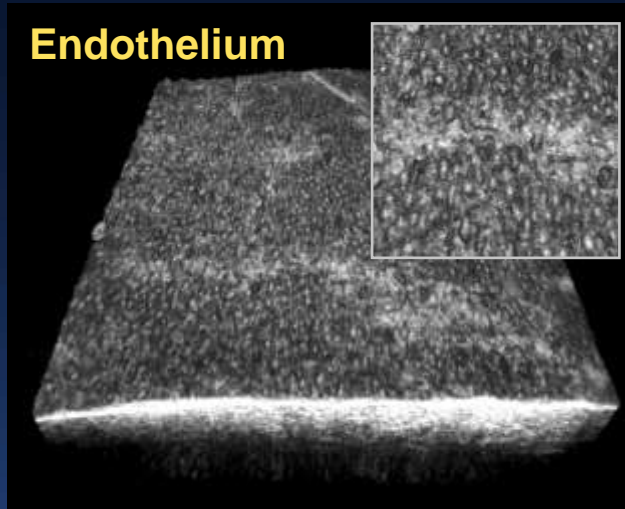
Smooth Muscle Cells



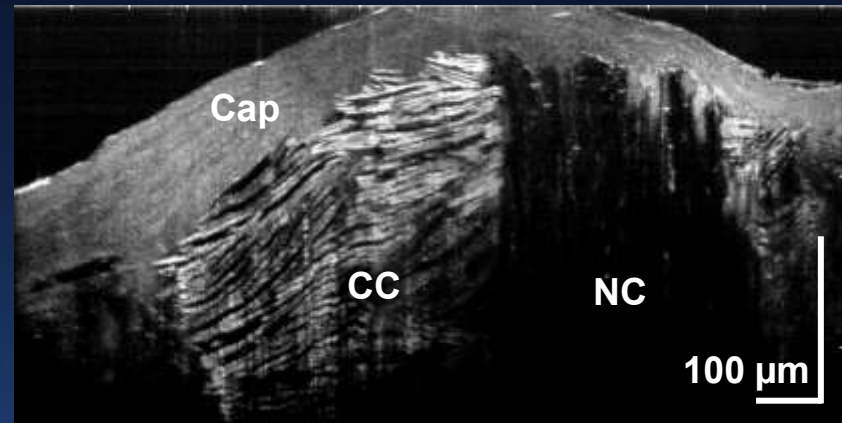
Macrophages



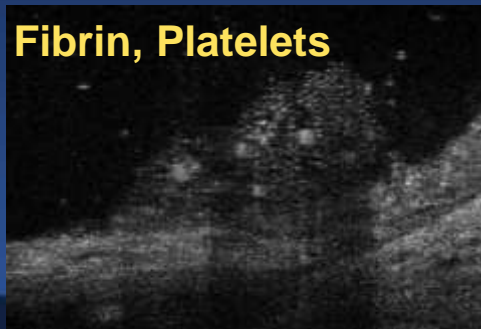
Endothelium



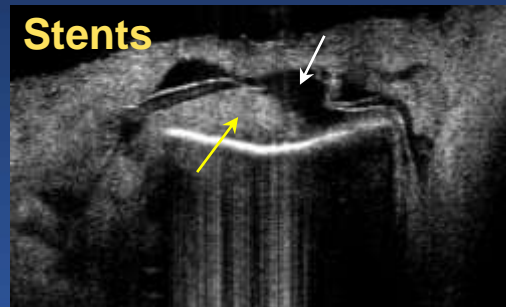
Necrotic Core Cholesterol Crystals



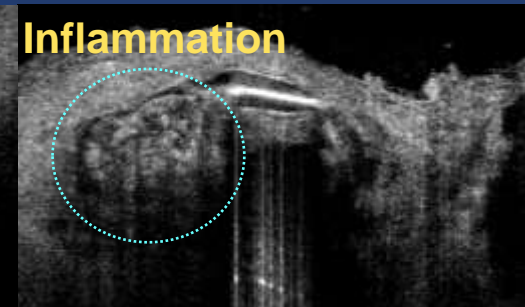
Fibrin, Platelets



Stents



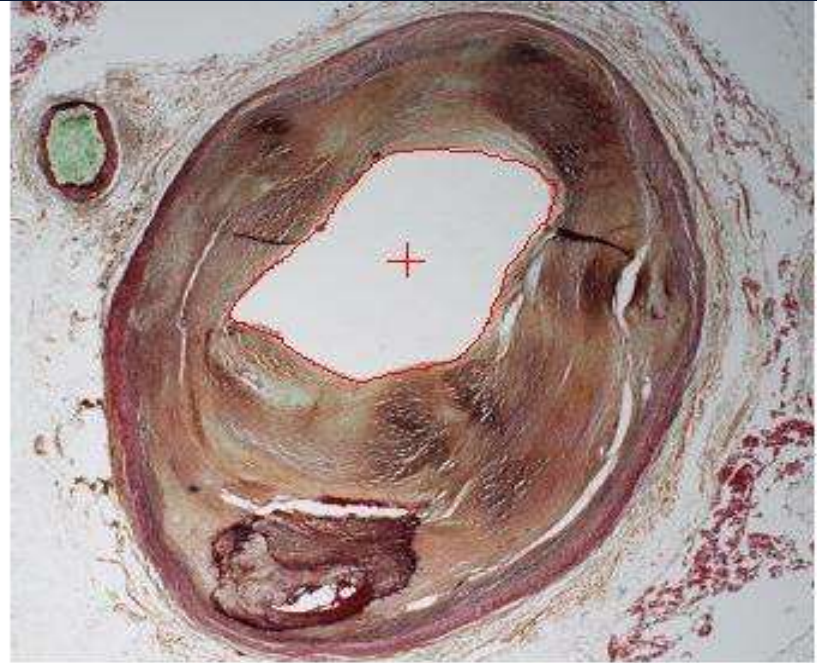
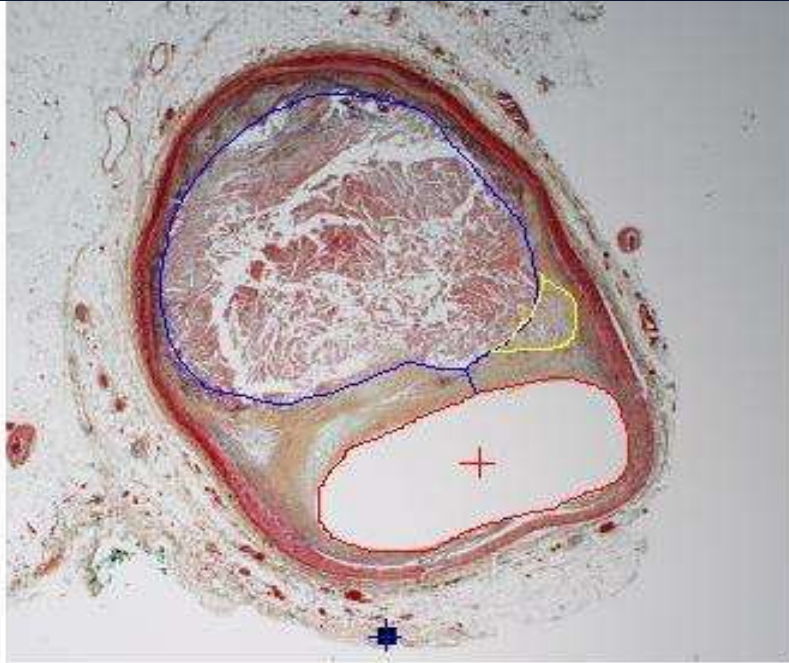
Inflammation



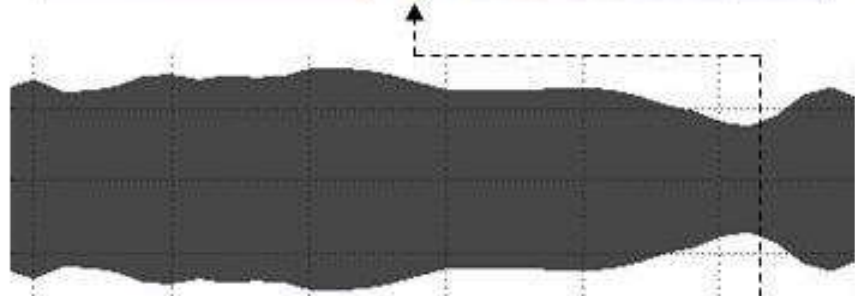
Powerful New Friend, NIRS



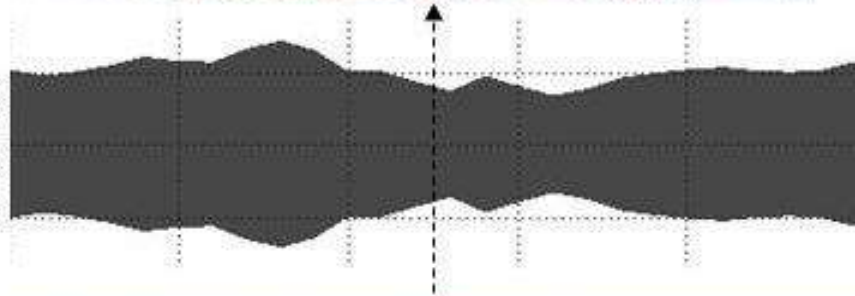
NIR can Distinguish Lipid-rich from Fibrotic Plaques



IVUS
DIAMETER



IVUS
DIAMETER

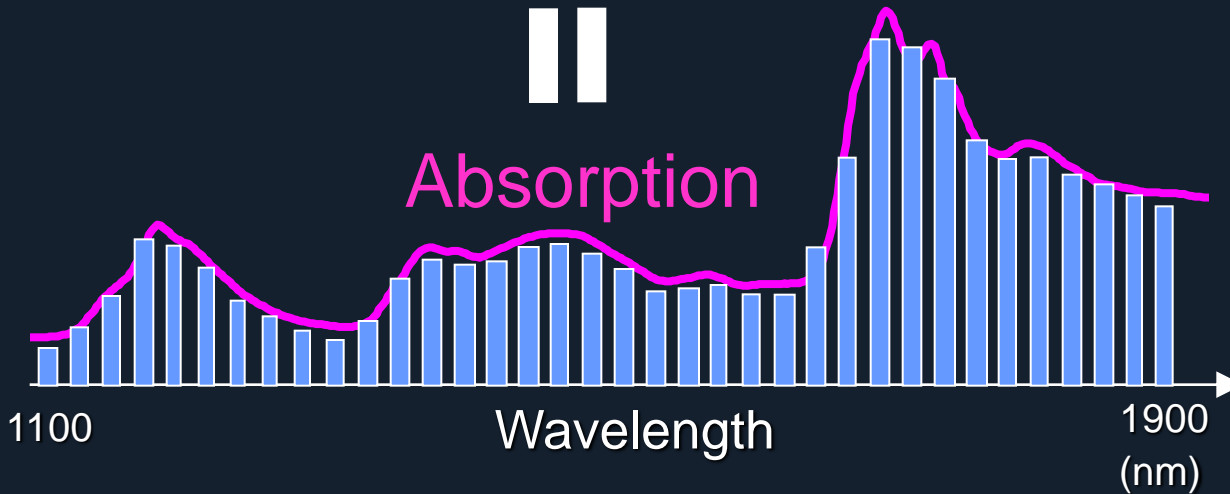
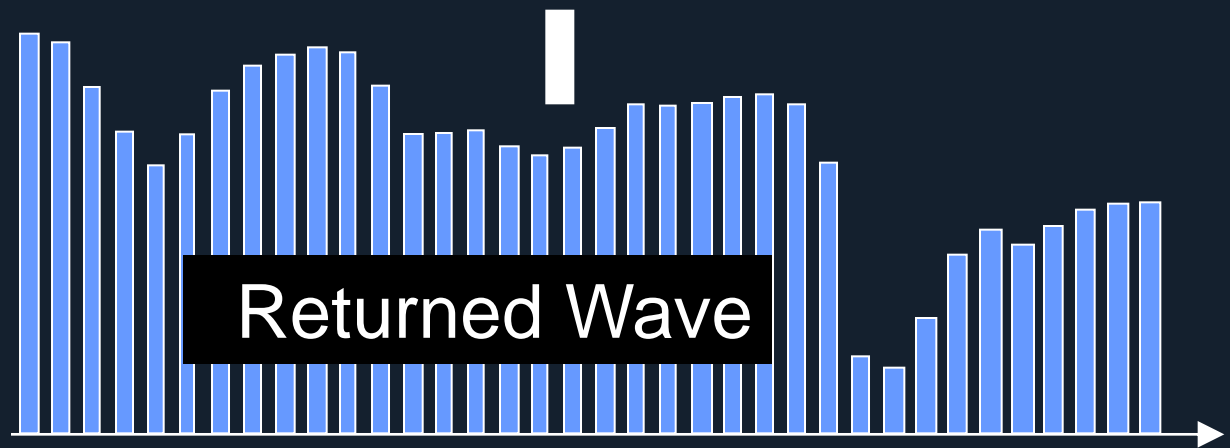
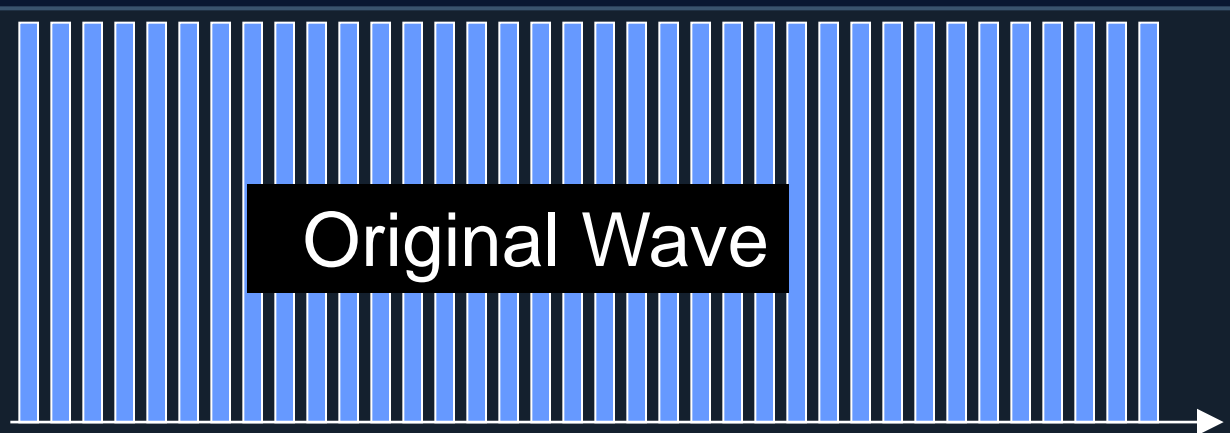
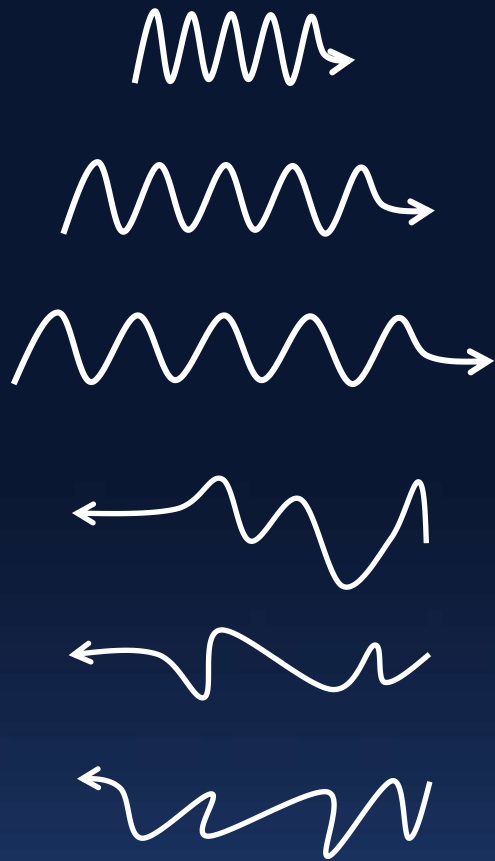


CHEMOGRAM



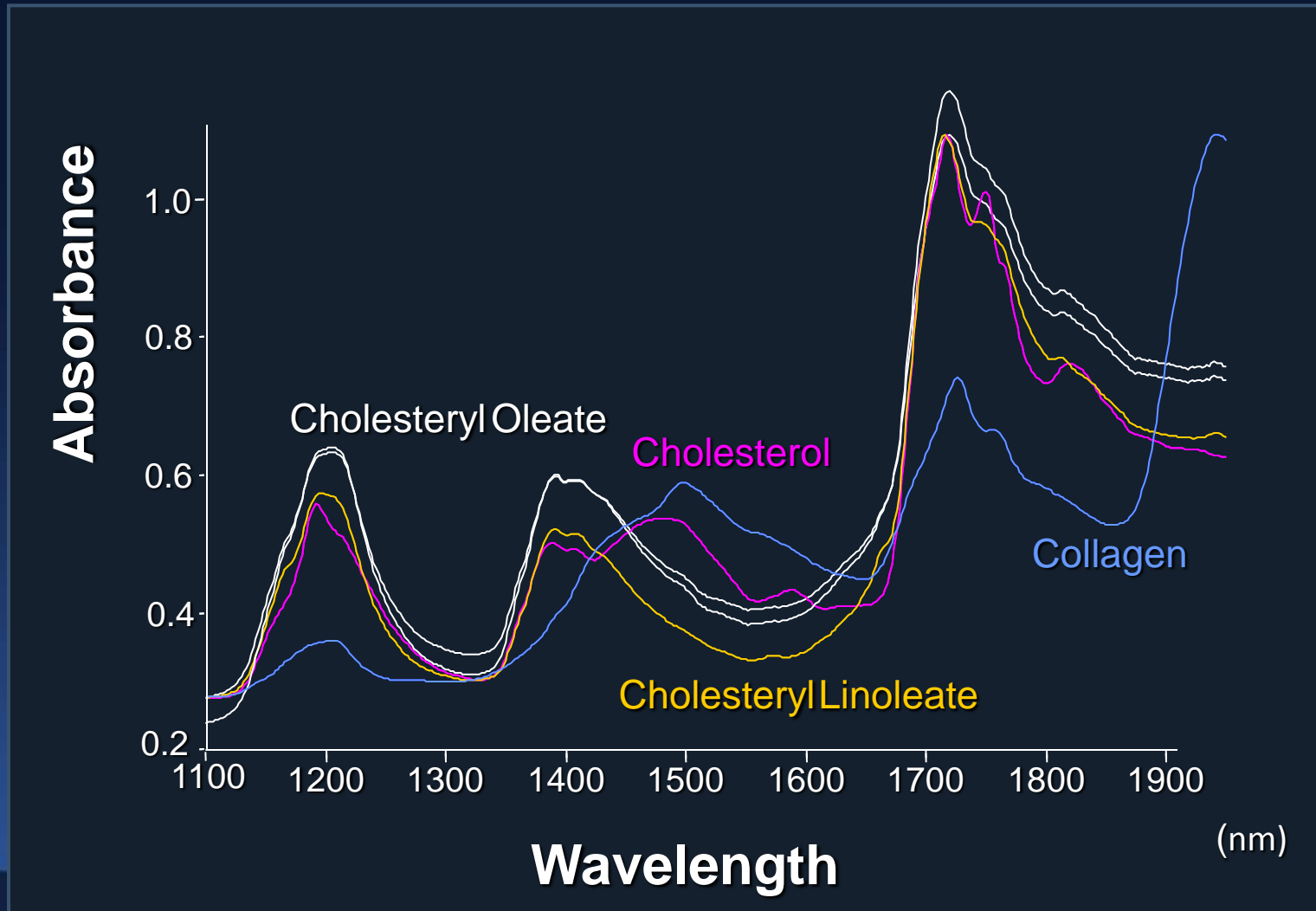
CHEMOGRAM



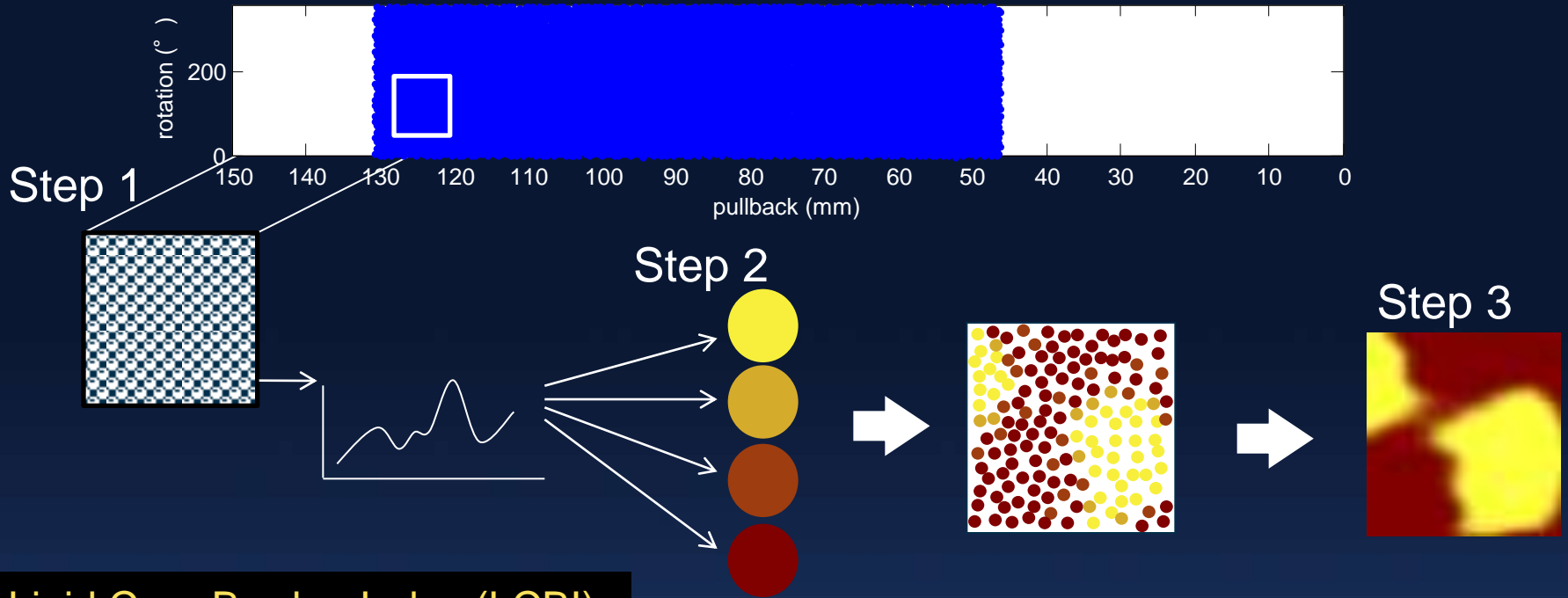


NIR Spectroscopy

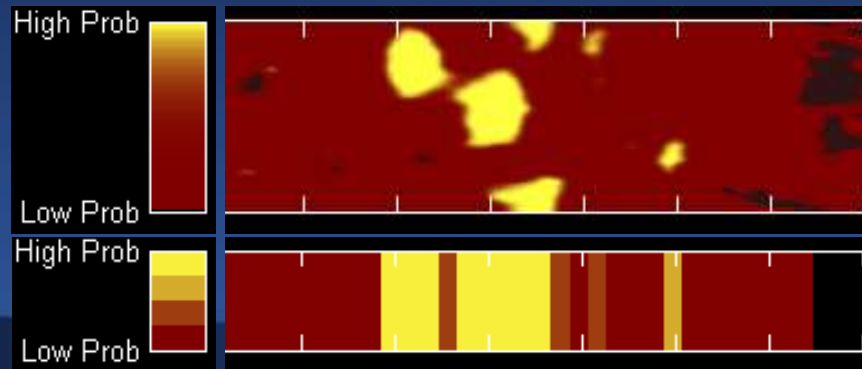
- Necrotic Core $>0.2\text{mm}$ thick, $>60^\circ$, Cap $<0.45\text{mm}$



Near Infrared Spectroscopy

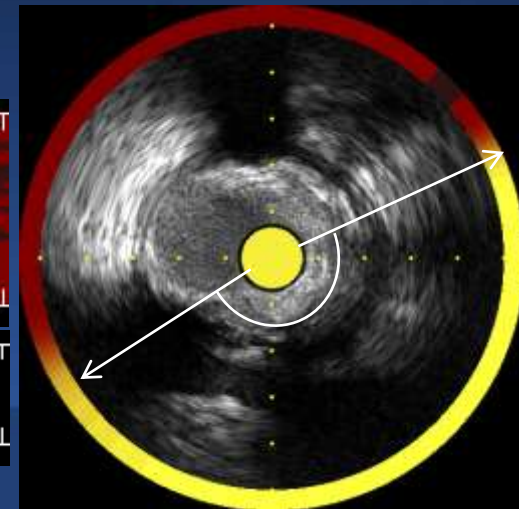


Lipid Core Burden Index (LCBI)
 = Yellow pixel / All variable pixel
 × 1000

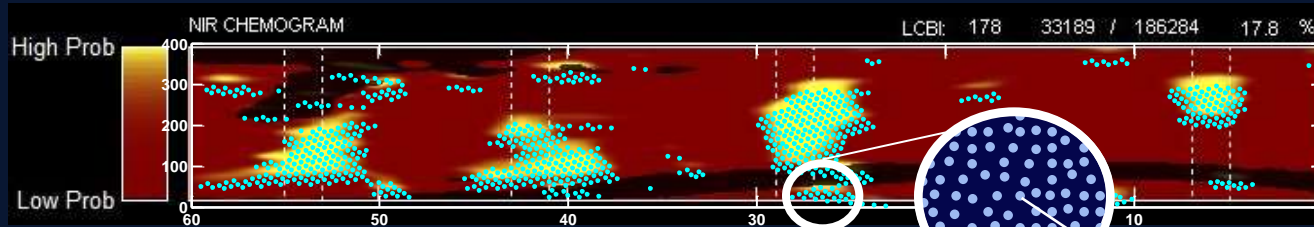


Proximal

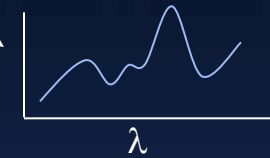
Distal



Formation of the Cap Thickness Prediction Image



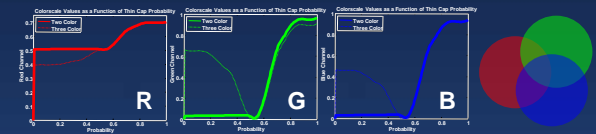
Spectra acquired at discrete pullback and rotation positions



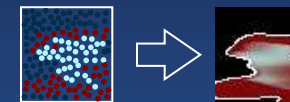
LCP Spectra transformed into posterior probability of thin cap presence



Probability mapped to a color

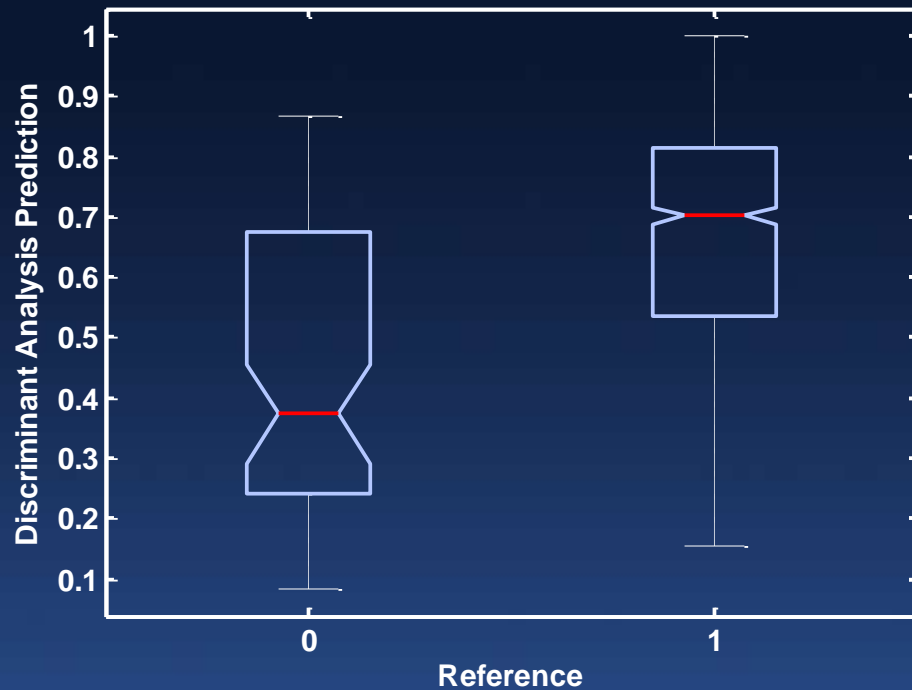


Pixels formed into an image

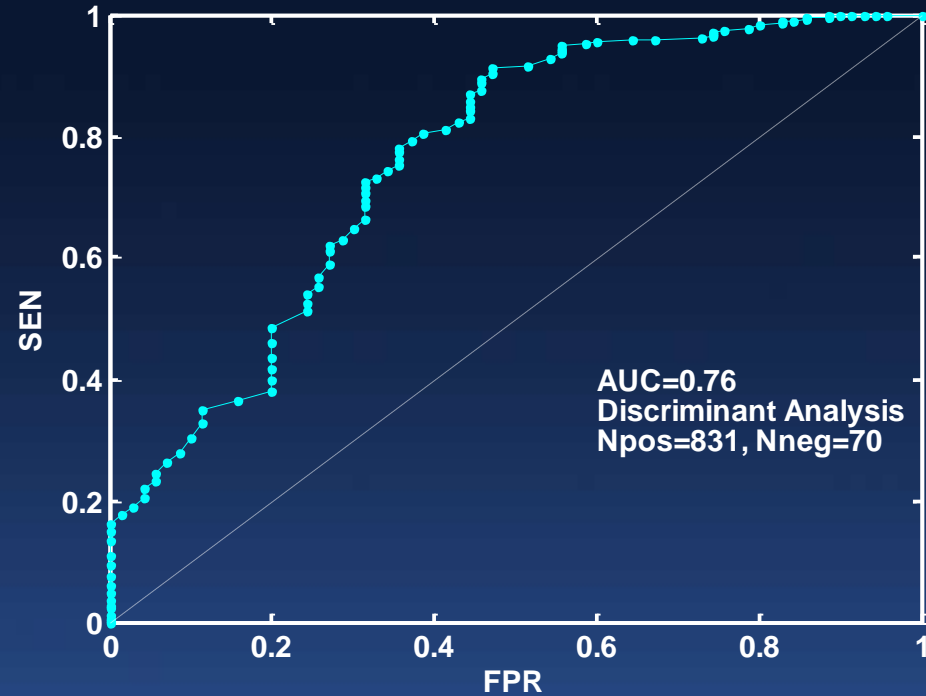


Ability to Predict Thin Cap (<0.065mm)

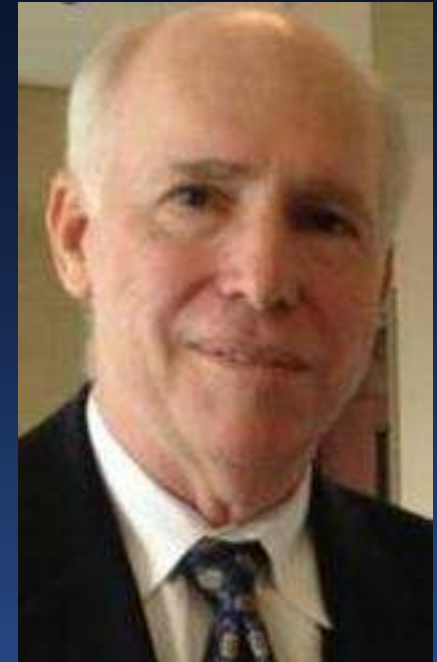
Capmeth=min, Neg=Cap<0.065mm,
Pos=Cap>0.065mm, CapTypes=[LCNCCC]



Capmeth=min, Neg=Cap<0.065mm,
Pos=Cap>0.065mm, CapTypes=[LCNCCC]

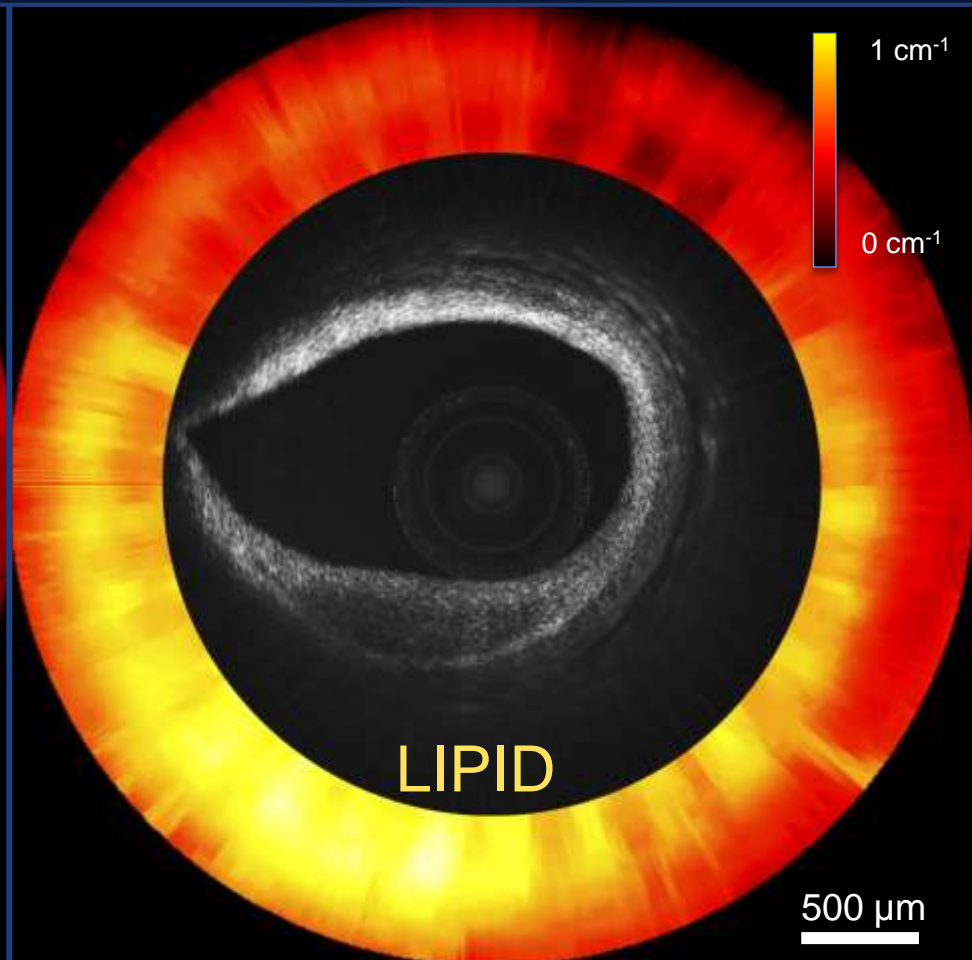
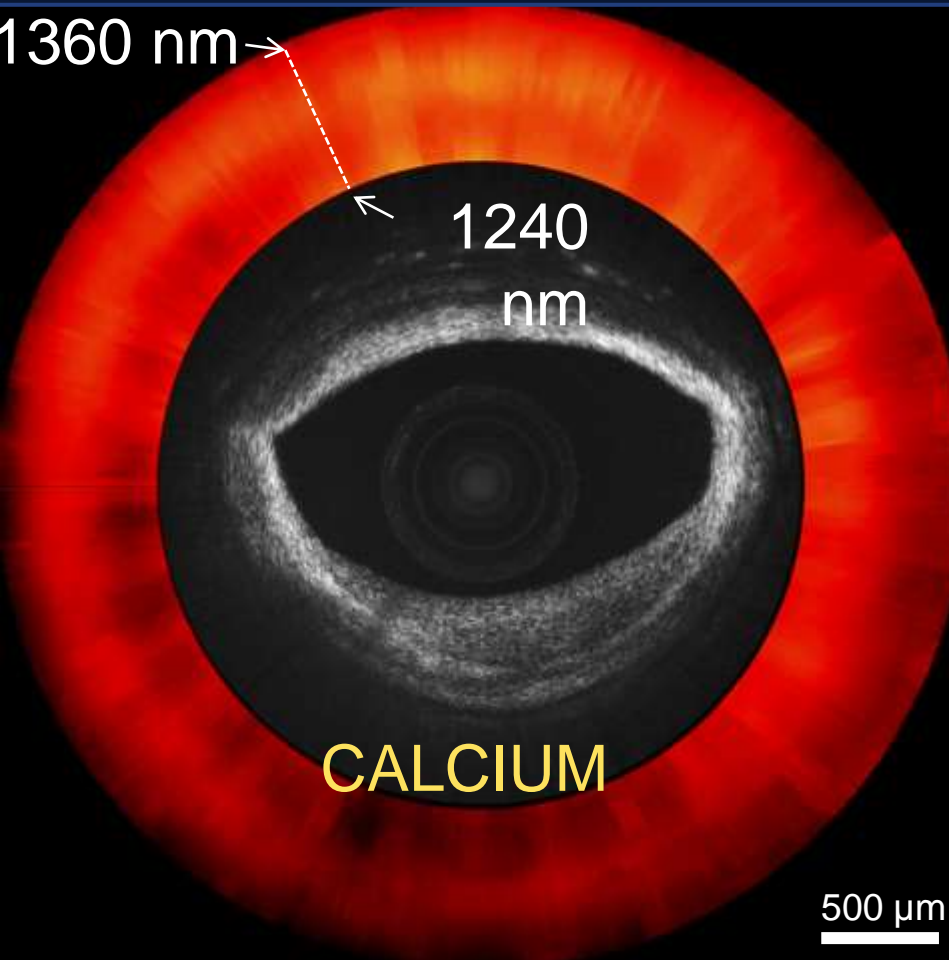


All Together

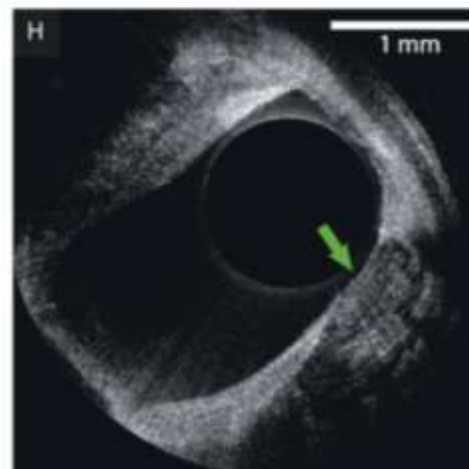
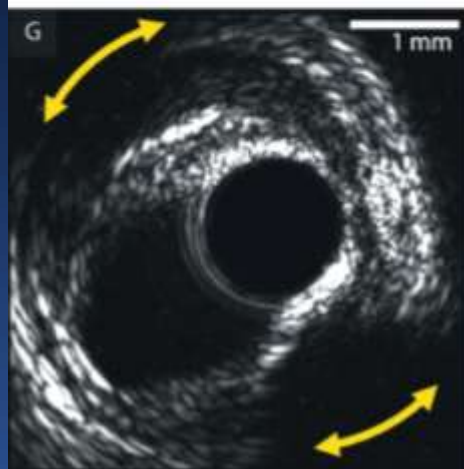
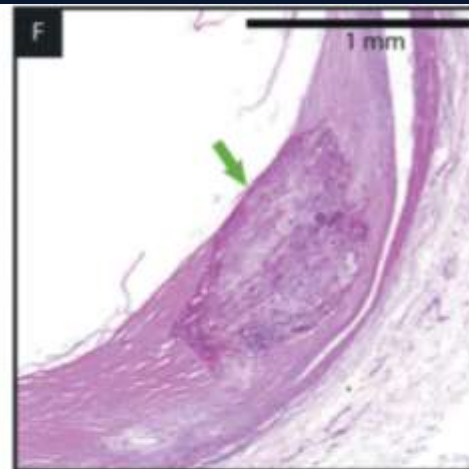
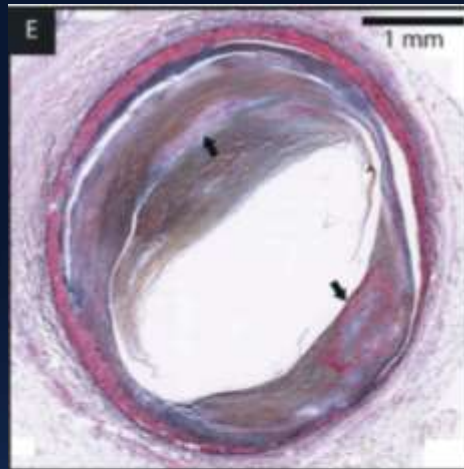
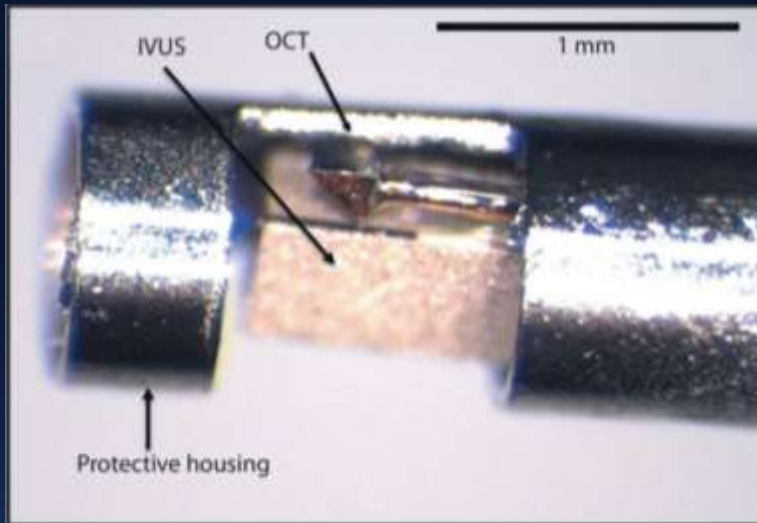


OCT-NIRS

Cadaver Coronary Plaques



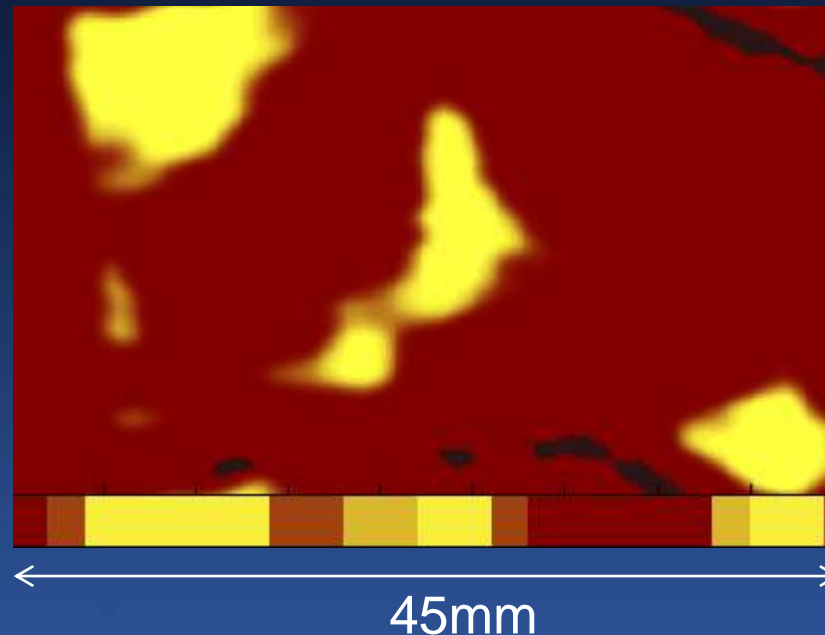
OCT and IVUS



Relationship between Lipid Rich Plaque detected by NIRS and Outcomes

- Prospective Single Center Study, 206 patients (ACS47%)
- Primary Endpoint: Composite of all-cause mortality, non-fatal ACS, stroke and unplanned PCI during one-year FU
- >40mm non culprit segment of NIRS

Lipid Core
Burden Index
(LCBI)=188

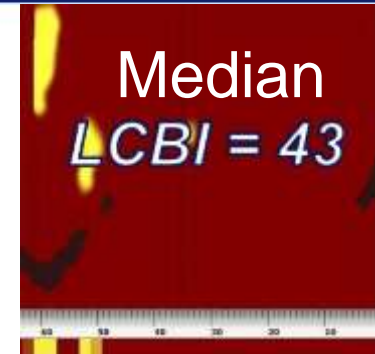
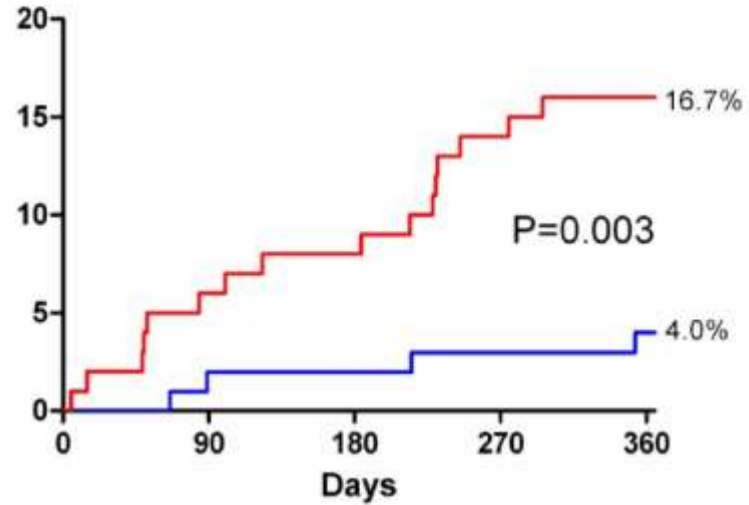


Relationship between Lipidic Plaque detected by NIRS and Outcomes

Primary endpoint



Cumulative Rate of
All-Cause Mortality, Stroke,
Non-fatal ACS or unplanned PCI
(excl. definite CLR events)



— LCBI ≥ median
— LCBI < median

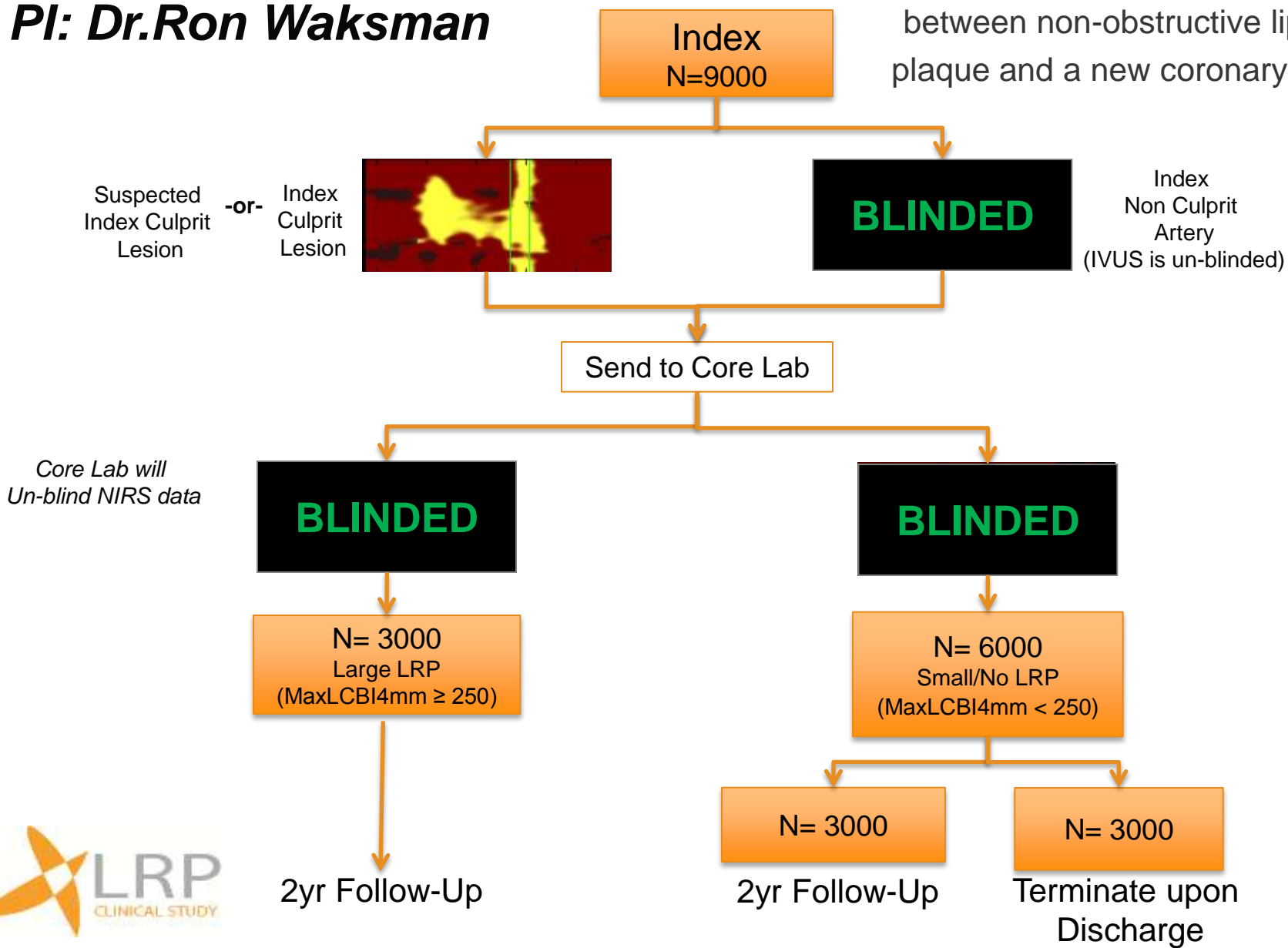
No. at Risk

LCBI < Median	101	99	99	97	91
LCBI ≥ Median	102	94	92	86	83

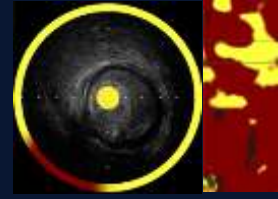
Adjusted HR: **4.04** 95% CI: 1.3-12.3 P=0.01

PI: Dr. Ron Waksman

“To evaluate the relationship between non-obstructive lipid-rich plaque and a new coronary events”



PROSPECT II Study



**900 pts with ACS at up to 20 hospitals
in Sweden, Denmark and Norway (SCAAR)**

NSTEMI or STEMI $>12^{\circ}$

IVUS + NIRS (blinded) performed in culprit vessel(s)

Successful PCI of all intended lesions (by angio \pm FFR/iFR)



Formally enrolled



3-vessel imaging post PCI

Culprit artery, followed by non-culprit arteries

Angiography (QCA of entire coronary tree)

IVUS + NIRS (blinded) (prox 6-8 cm of each coronary artery)



PROSPECT II Study PROSPECT ABSORB RCT

900 pts with ACS after successful PCI

3 vessel IVUS + NIRS (blinded)

≥1 IVUS lesion with ≥70% plaque burden present?

Yes

(N=300)

No

(n=600)

R

1:1

**ABSORB BVS
+ GDMT** (N~150)

GDMT
(N=150)

Routine angio/3V IVUS-NIRS FU at 2 years

Clinical FU for ≥3 years

Where are we going?

- 1. Higher technology to visualize cell level morphology and complementary imaging, which allow us to understand vulnerable plaque more details.**
- 2. In the meantime, to develop the best treatment option (BVS stent, super-intensive short term drug therapy, etc) should be evaluated to change the patient outcome ultimately.**
- 3. Fortunately, the current optimal medical therapy reduce hard endpoint significantly, and need to differentiate very high risk patient who has benefit of these technology.**