

Plaque Composition of Diverse Vascular Lesions

Angioplasty Summit Seoul - April 24, 2008

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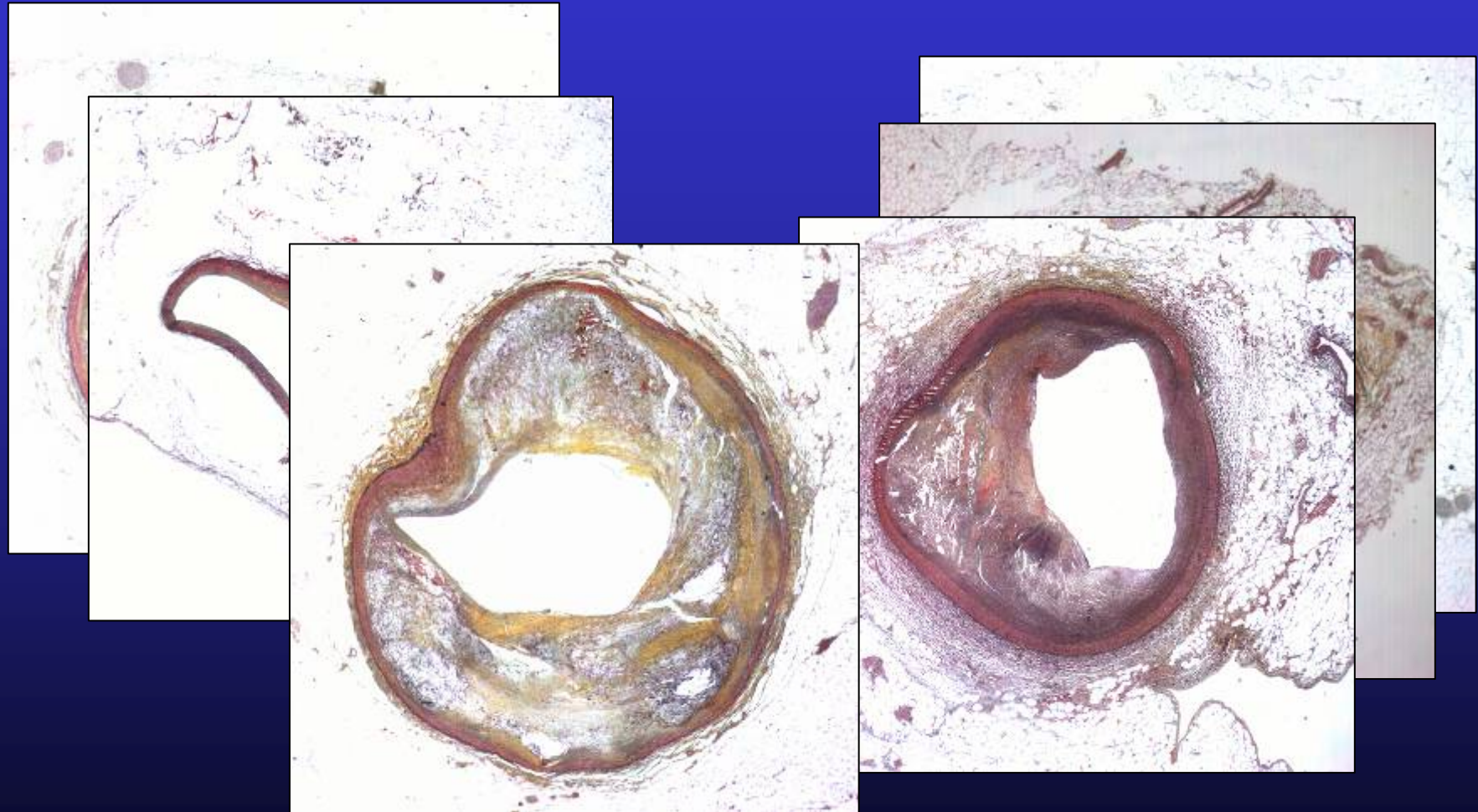
Introduction

- Atherosclerotic plaques are formed by repeated sequences of inflammation, rupture of fibroatheroma, and thrombus formation.
- Although this process is identical in coronary, carotid and peripheral arteries, it manifests itself in different ways depending on lesion locations, flow dynamics and the clinical manifestations of plaque rupture.
- Within the same artery, flow dynamics determine the site of plaque deposition.

Introduction

- Basic Atherosclerosis
- Bifurcation Lesions
- Culprit of Culprit
- Coronary Lesions
- SFA/Popliteal Lesions
- Carotid Lesions

Coronary Atherosclerosis

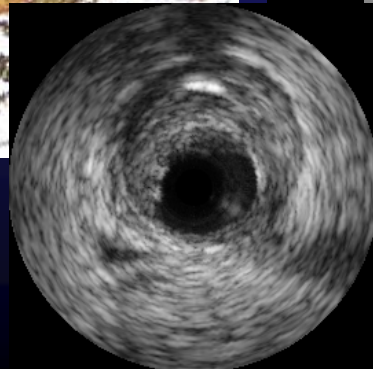
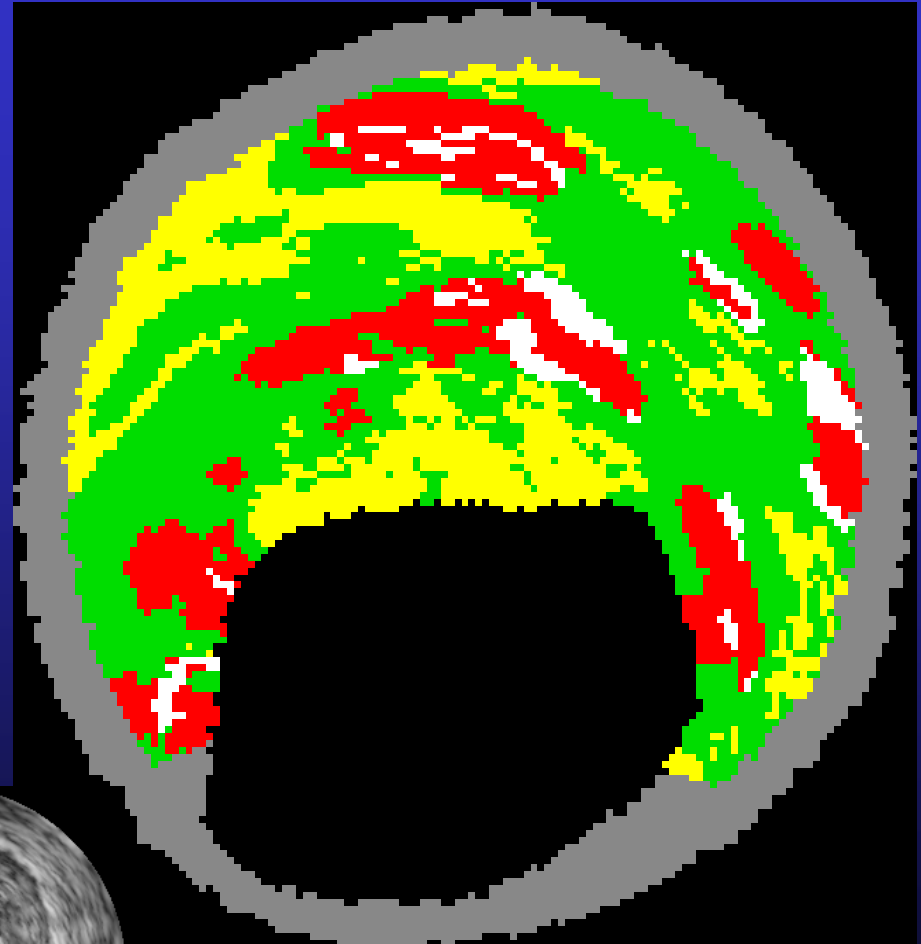
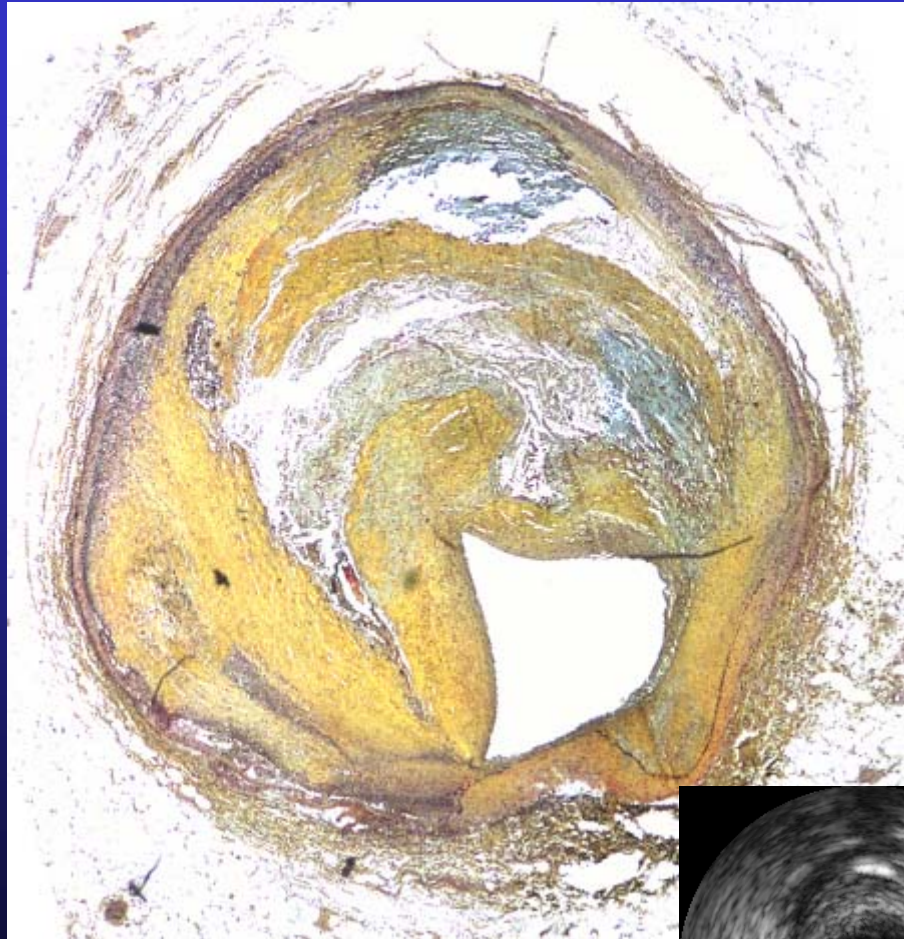


May 14, 2007

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Layering Effect of Repeated Plaque Ruptures



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Plaque Composition and its Acknowledged Shear Stress Patterns in Coronary Arteries;

G. Rodriguez-Granillo, MD et al.; JACC, Vol. 47; No. 4, 2006

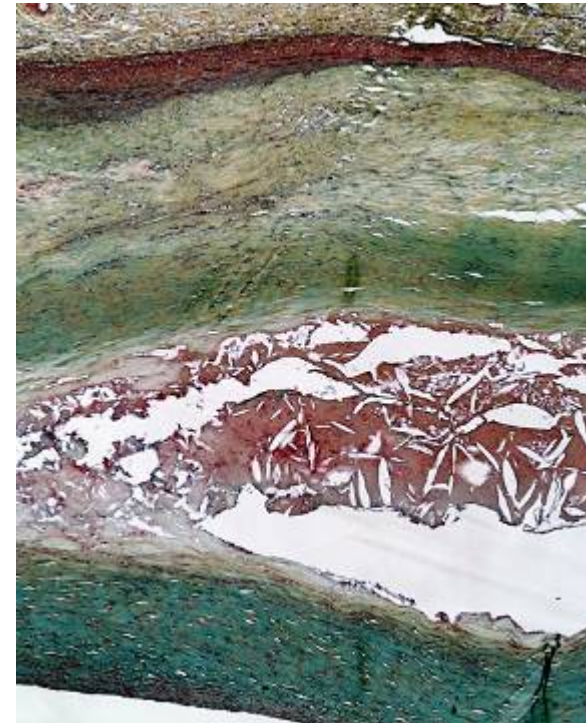
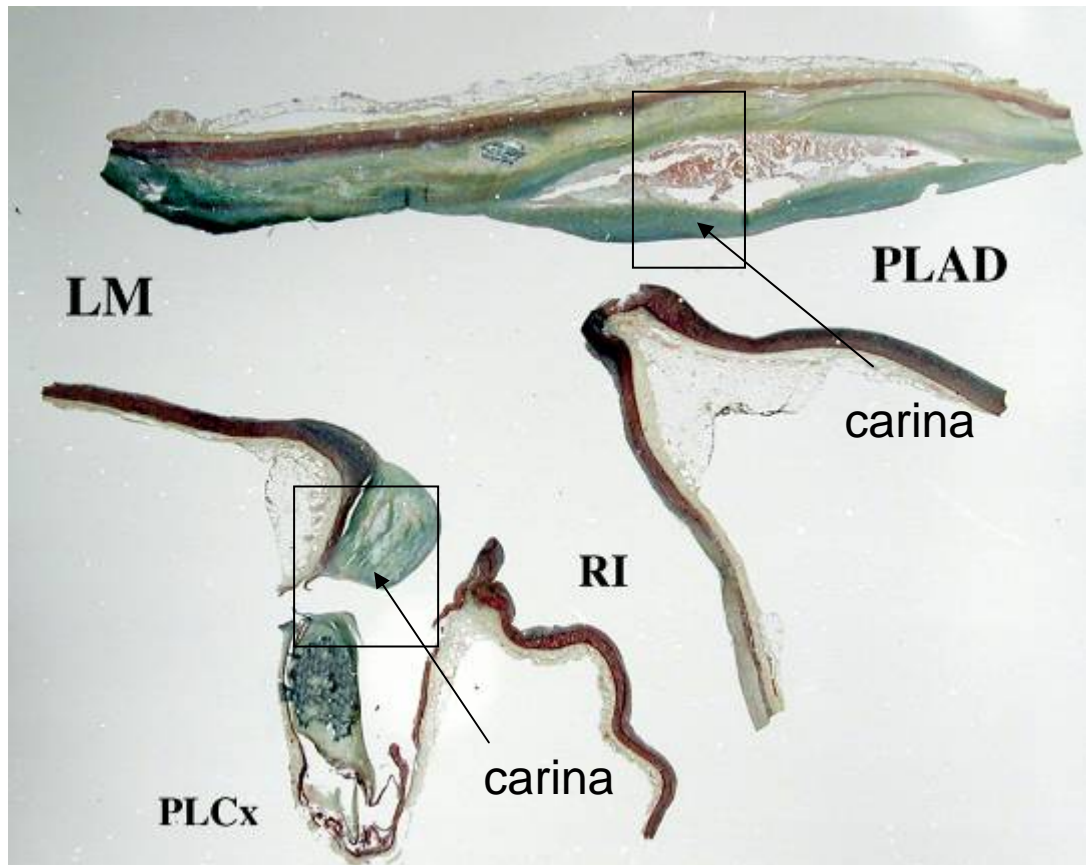
„Several studies in coronary and peripheral arteries have demonstrated that atherosclerosis has a tendency to arise more frequently in low-oscillatory shear stress (LOSS) regions such as in inner curvature of non branching segments and opposite to the flow-divider (FD) at bifurcations. In particular, atherosclerotic disease has certain predilection for the outer wall of the left main coronary artery bifurcation sparing the flow divider. Intravascular ultrasound (IVUS) has been used to describe the extend, distribution and profile of plaques in the proximal left anterior descending coronary artery (LAD)“.

In vivo data regarding tissue content was so far unknown.

In summary it was found ... That the OLAD (ostial LAD) atherosclerotic plaques present larger plaque burden, eccentricity and MPT than DLMCA (distal Left Main CA) plaques. In addition, a larger calcified and necrotic core content was found distal to the circumflex take off. Lesions were predominantly located in the outer wall of the carina and such location was associated with larger necrotic core content.

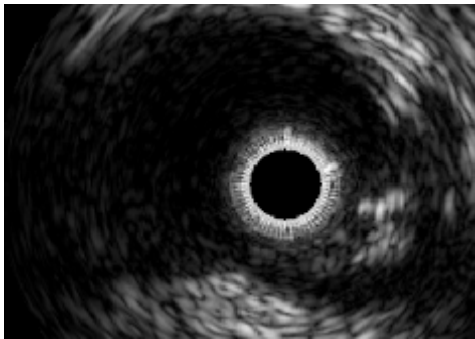


Where to look for the Site of the Problem, the “culprit of the culprit”

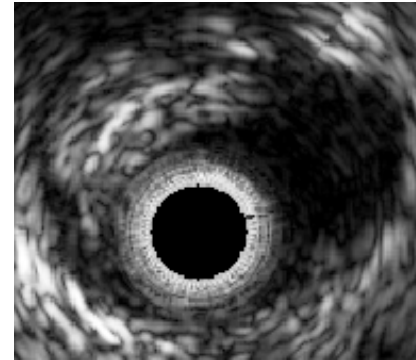
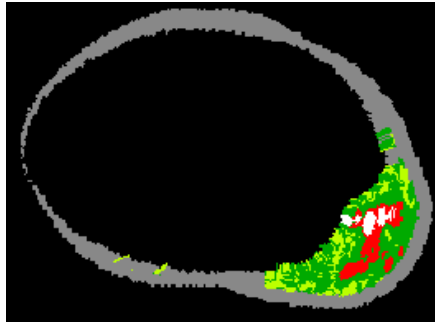


Courtesy to Dr Virmani

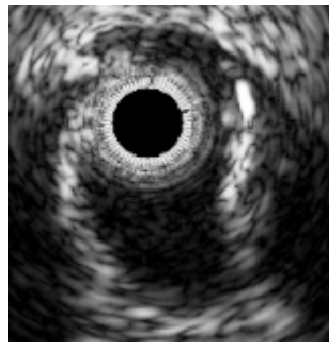
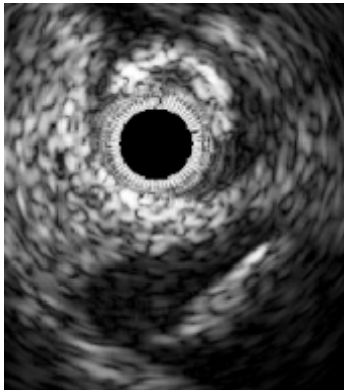
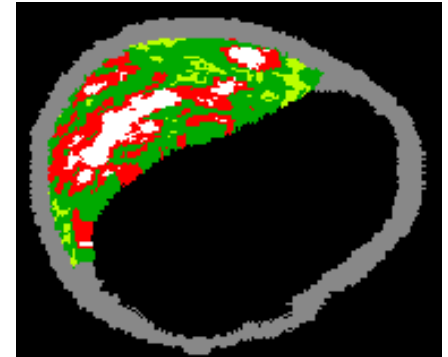
8 consecutive Bifurcation lesions from the VH Registry (bifurcations 1-4)



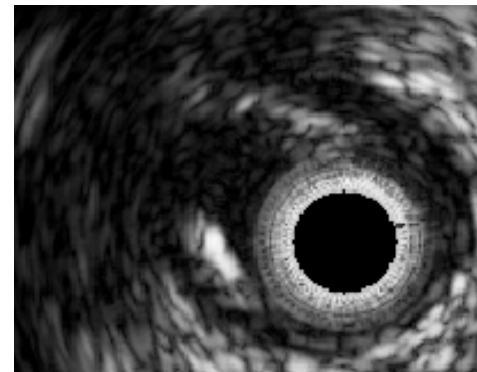
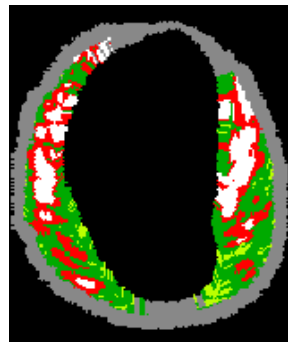
Lesion 1



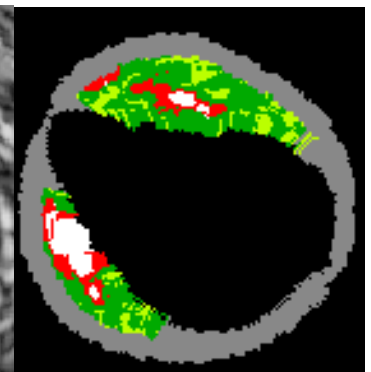
Lesion 2



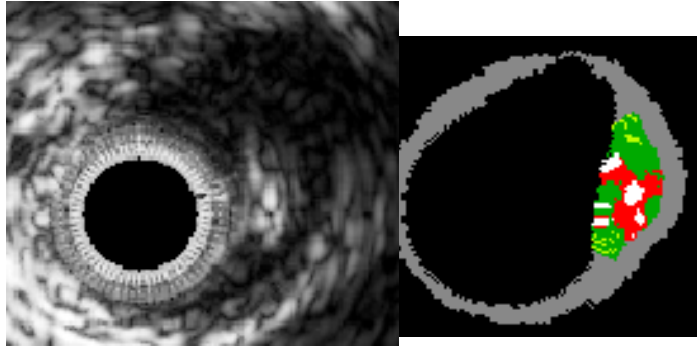
Lesion 3



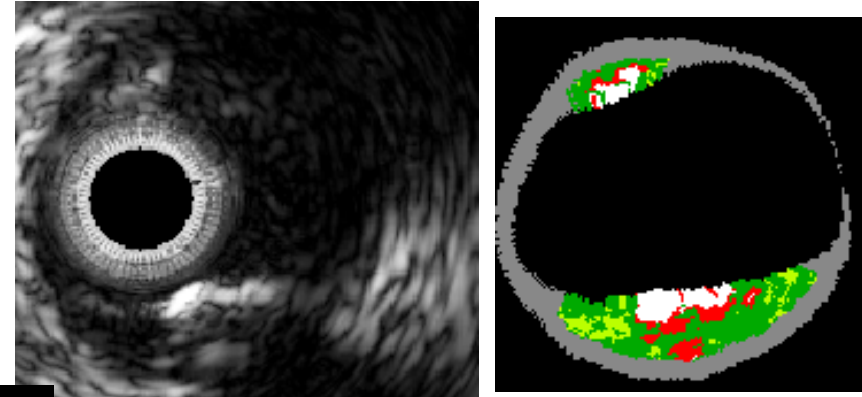
Lesion 4



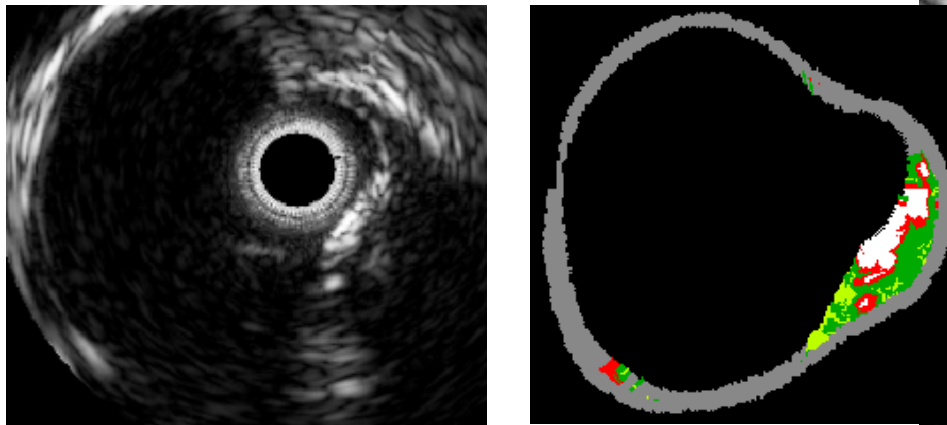
8 consecutive Bifurcation lesions from the VH Registry (bifurcations 5-8)



Lesion 5

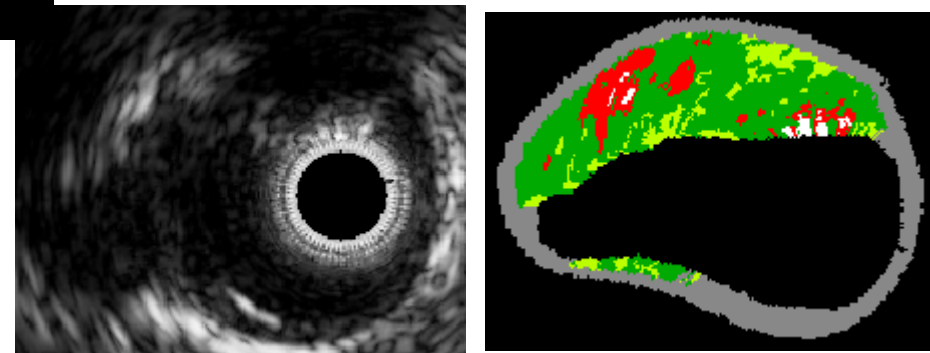


Lesion 6



Lesion 7

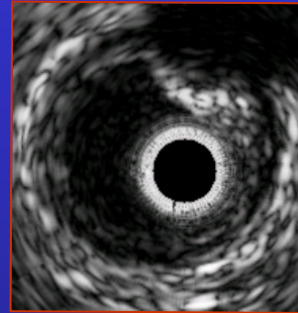
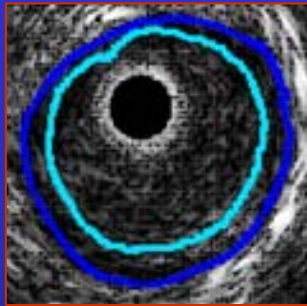
Lesion 8



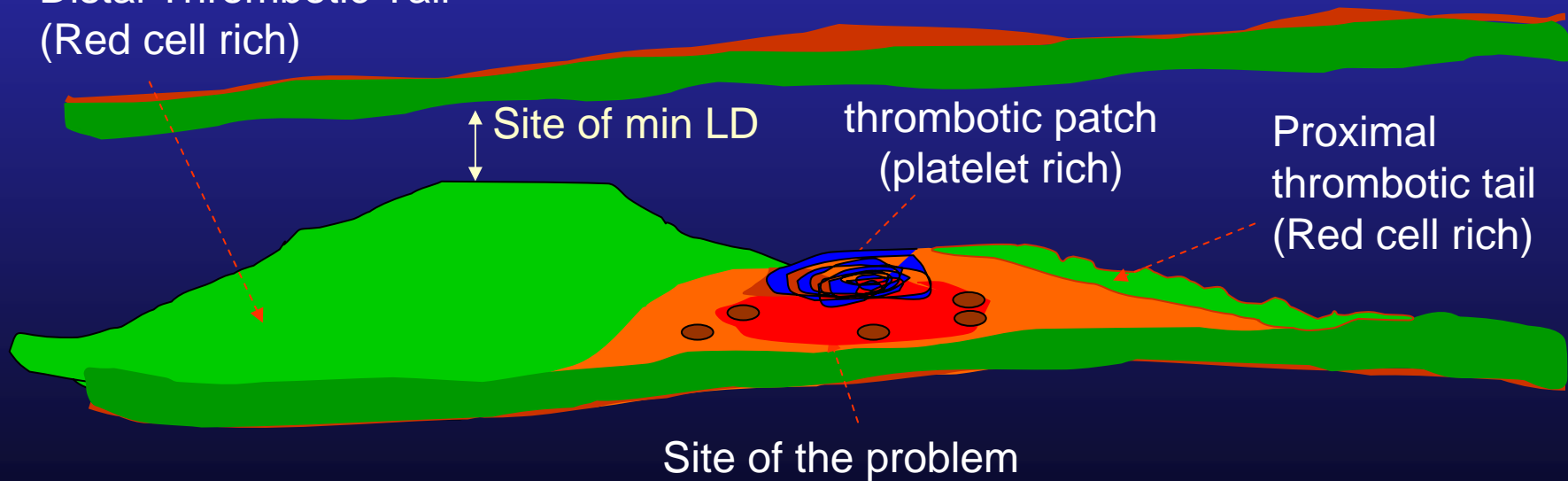
Culprit of the Culprit

- The site of plaque rupture is generally not the site of maximal arterial narrowing.
- When a plaque ruptures, thrombus forms not only at the rupture site but also proximally and distally.
- The greatest narrowing is usually at the site of the distal thrombotic tail, which may be a centimeter or more from the rupture site.

Rupture of an Eccentric TCFA



Fall Out of the problem
Distal Thrombotic Tail
(Red cell rich)



R Virmani, CVPath and
P Margolis, Volcano Corp.

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**CAPITAL Plaque type at
the site of the min LCSA;**

CaTCFA = 10

PIT = 8

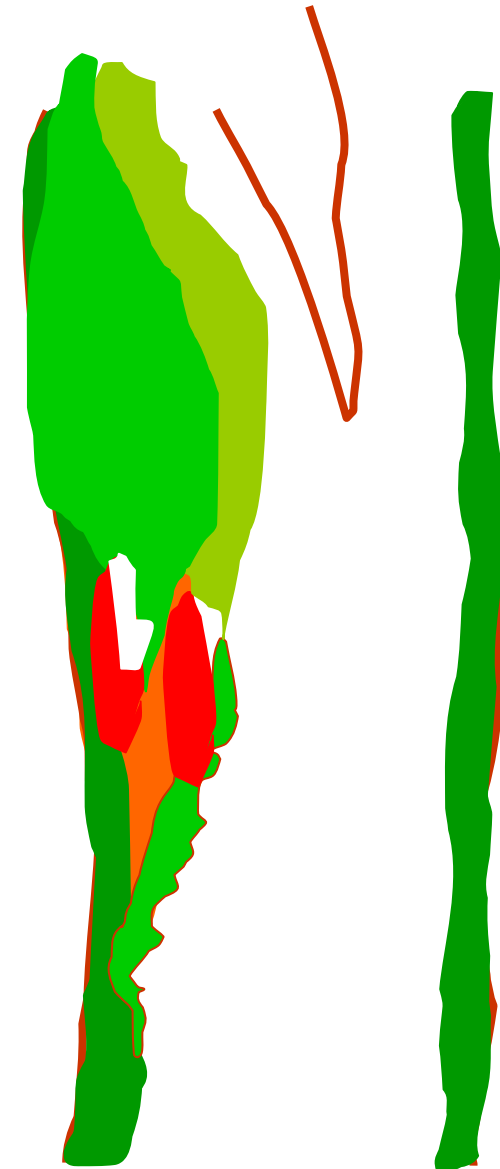
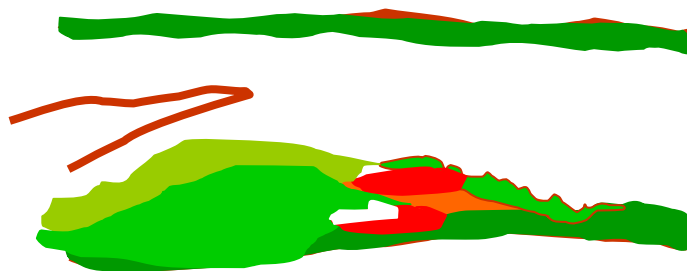
(88% of the time CaTCFA was found proximal to the
site of the min LCSA)

CaFA = 4

FA = 4

FCa = 3

Voila:
In
Carotids

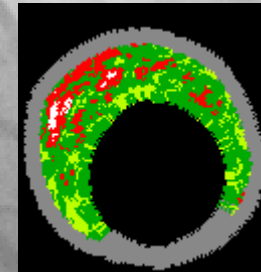


Thrombus Study In Japan

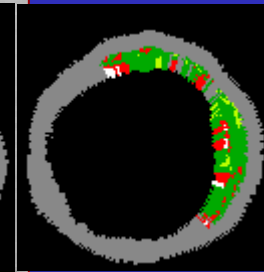
Rupture site



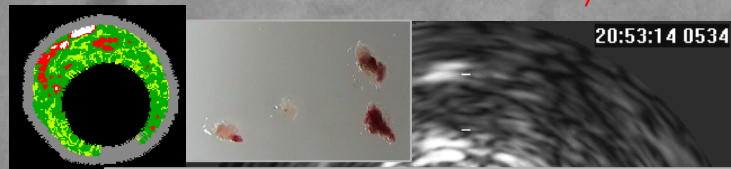
Thick fibrous plaque with necrotic core



Pre DCA



Post DCA

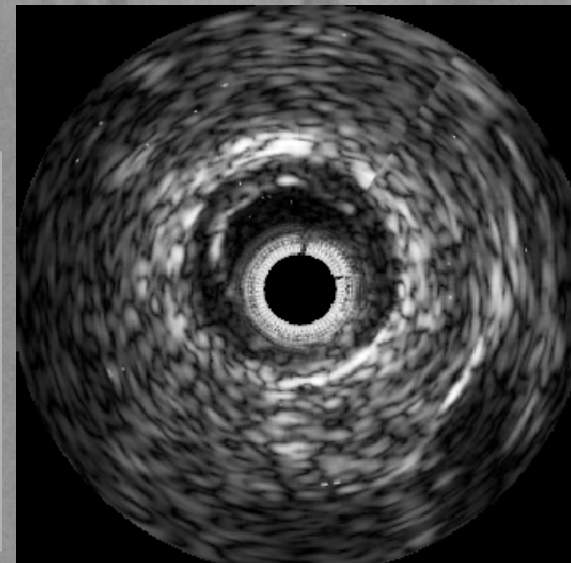


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Red cell rich thrombotic tail



Pre thrombectomy



Post thrombectomy

Courtesy:
O. Katoh
P. Margolis

Consultants



Bifurcation Lesions

- Bifurcations are the preferred sites of plaque deposition in all arteries.
- Atherosclerotic plaque tends to accumulate at sites of turbulence and low shear stress.
- Turbulence with accompanying variations in shear stress is always present at bifurcations.
 - This is particularly true when there is significant size discrepancy between the two bifurcating branches.
 - Characteristically, plaque deposition occurs at sites of lowest shear stress – i.e., the sides opposite the carina.

Coronary Artery Lesions

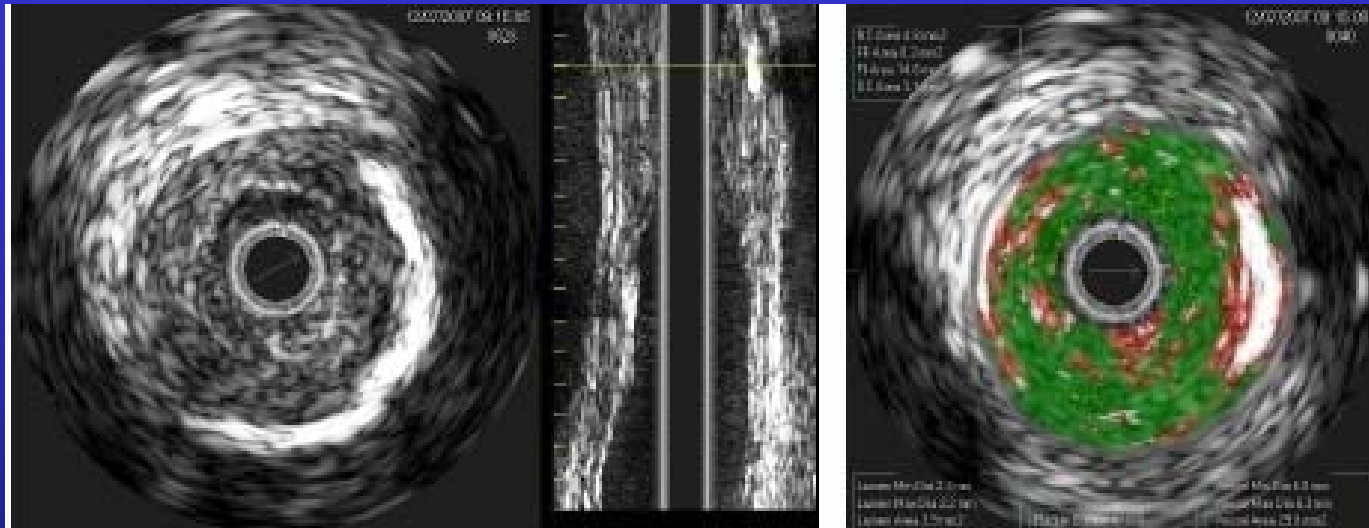
- Plaque ruptures are more likely to cause clinical events in coronary arteries than in peripheral arteries.
 - The relatively smaller size of coronary arteries leads to a greater likelihood of complete occlusion as a result of plaque rupture.
 - Coronary collaterals are less abundant than those in the SFA and Popliteal systems.
 - Acute ischemia is more likely to be clinically manifest in the coronary than the peripheral trees.
- Despite the above, the majority of patients with myocardial infarction and sudden death have had two or more plaque ruptures prior to the development of clinical symptoms.

SFA/Popliteal Lesions

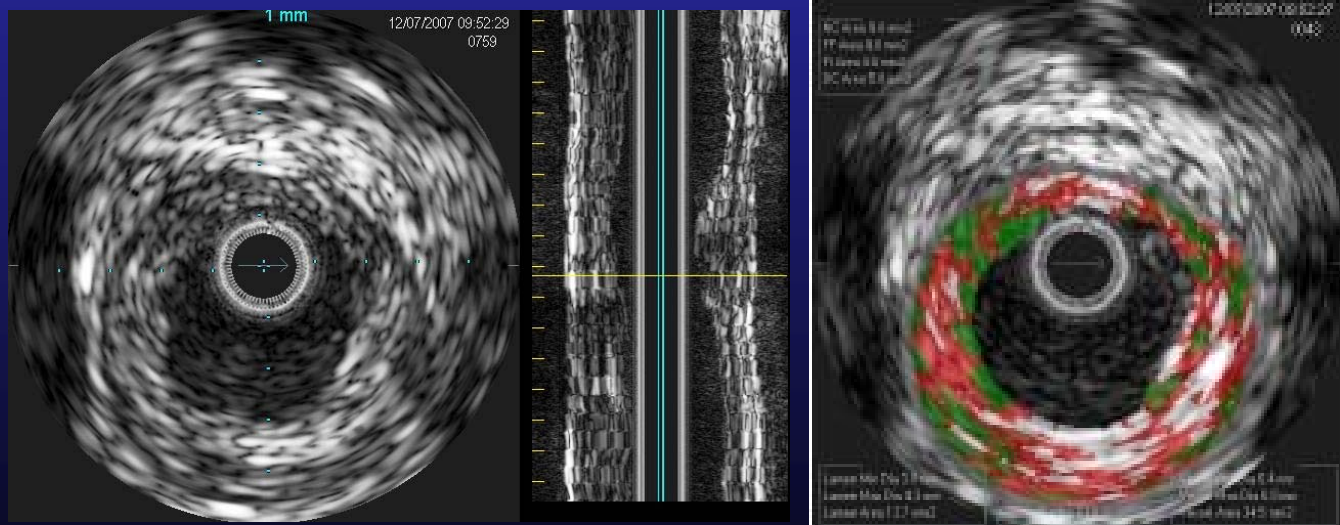
- The superficial femoral artery and proximal Popliteal arteries differ from most other arteries in many ways:
 - They are subject to much greater degrees of stress and strain than other vessels.
 - They have an abundant collateral network.
 - Occlusions in the SFA/Popliteal systems do not generally lead to acute or devastating events.
- Because of these factors, disease in the SFA and Popliteal arteries tends to be more active and more diffuse by the time clinical symptoms are manifest.
- Even chronic SFA lesions are likely to have associated thrombus.

SFA Stenosis

Pre



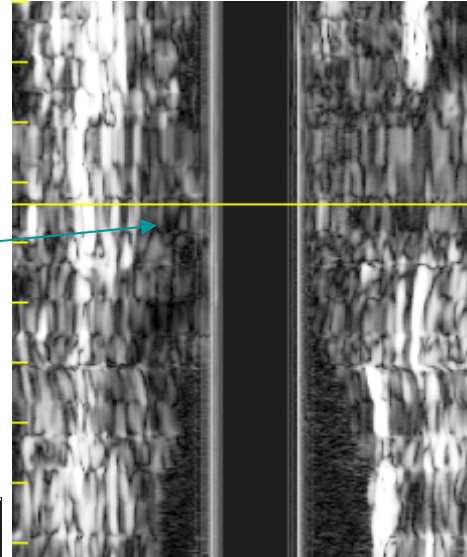
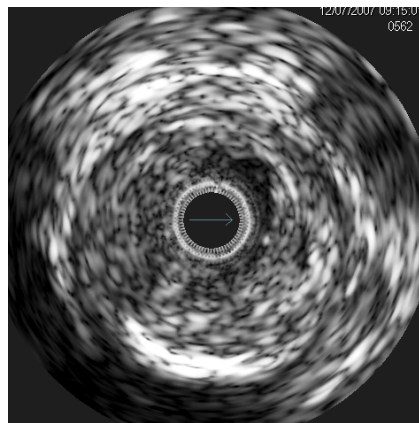
Post



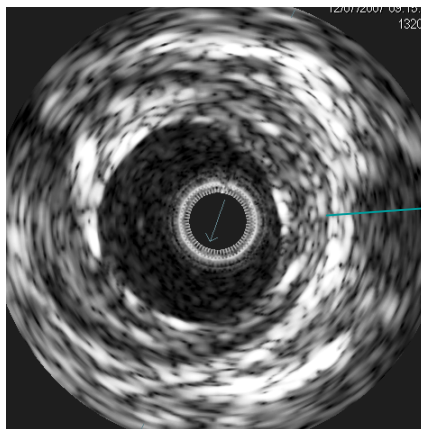
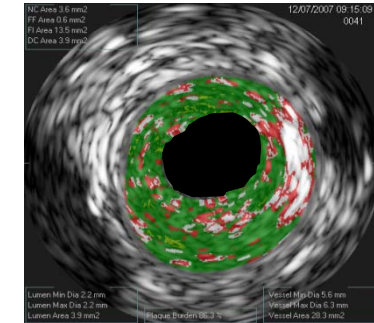
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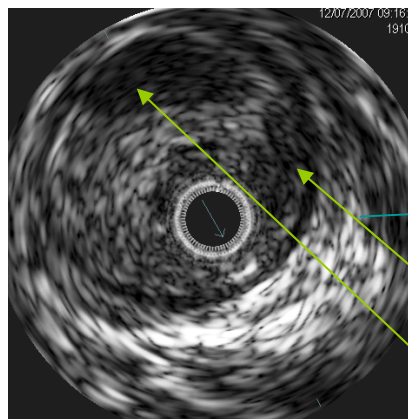
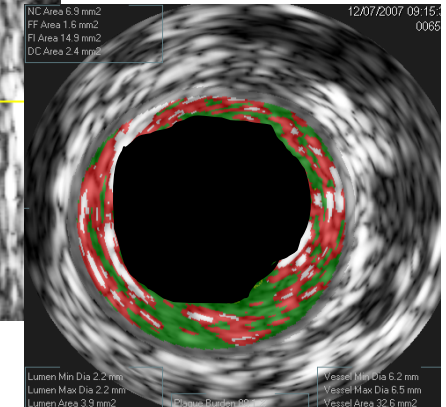
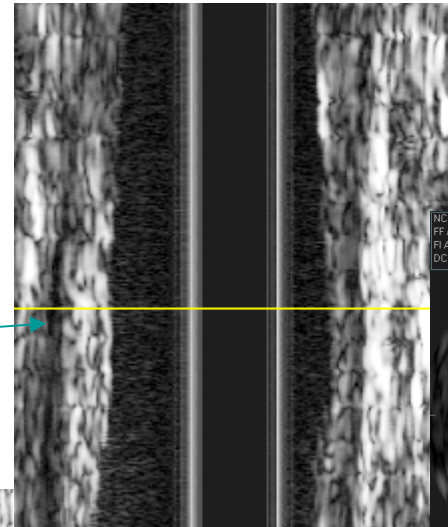
IVUS-VH Pullback in Patient with SFA Stenosis



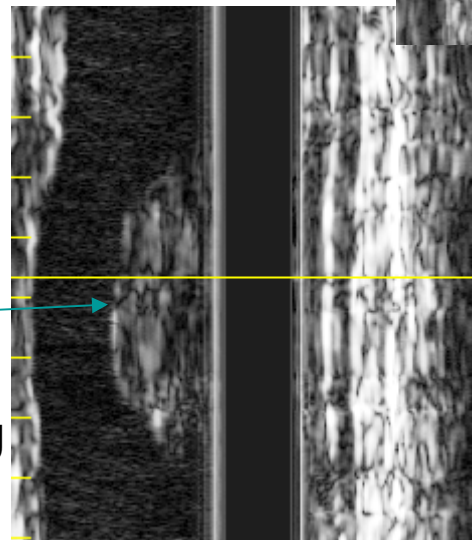
Prox: site of
min LCSA,
organized
thrombus



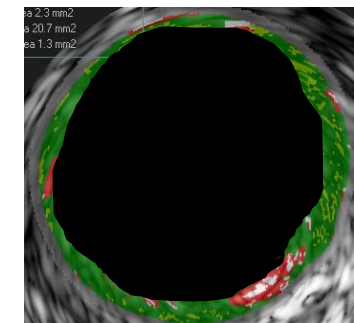
Mid: Deep
calcium and
Necrotic Core



Free
flowing
blood

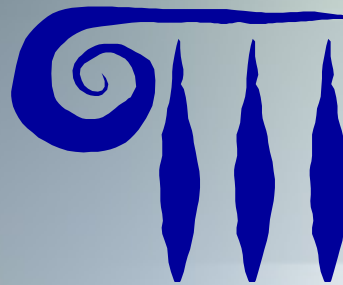
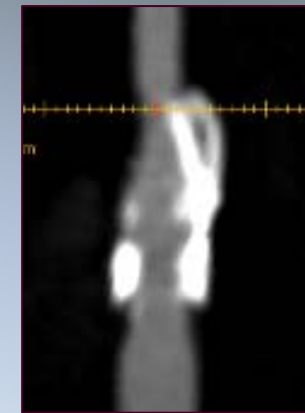
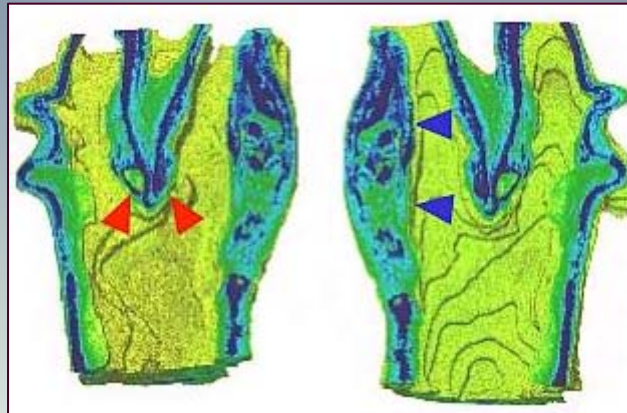
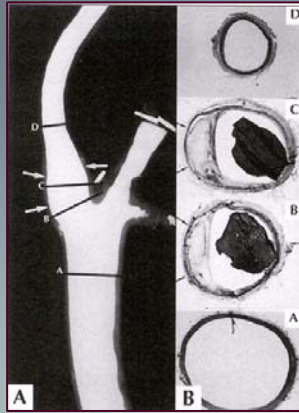


Distal: fresh
thrombus



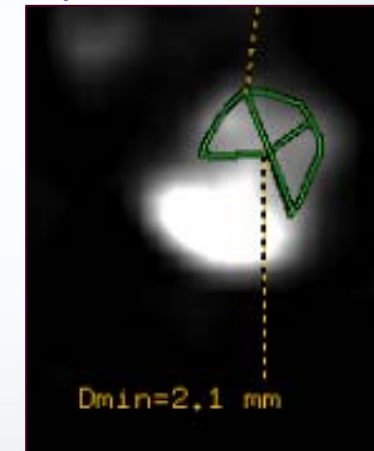
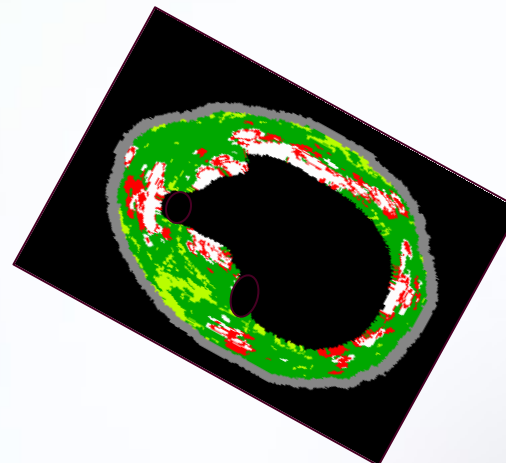
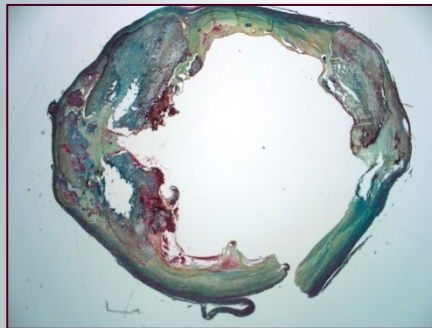
Carotid Lesions

- Whereas, lesions in the coronary and peripheral vessels generally cause clinical problems by virtue of acute and sub-acute occlusion, carotid lesions manifest themselves clinically by virtue of embolization.
- Although the basic atherosclerotic process in the carotid arteries does not differ from that in other vessels, flow dynamics are particularly important in the carotid system as determinants of plaque characteristics, sites of deposition and clinical manifestations.



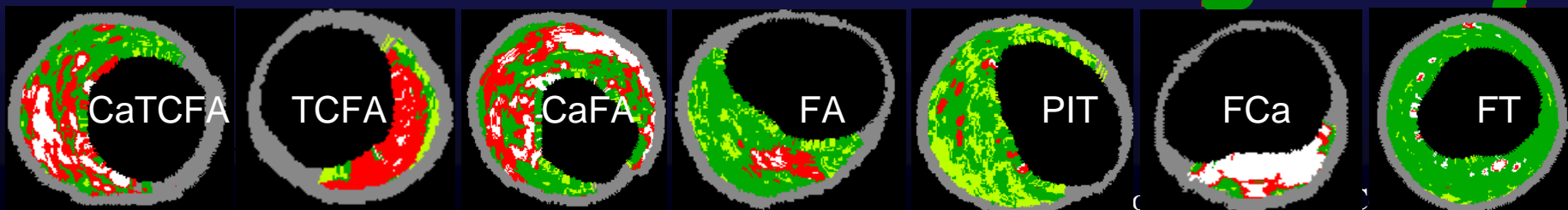
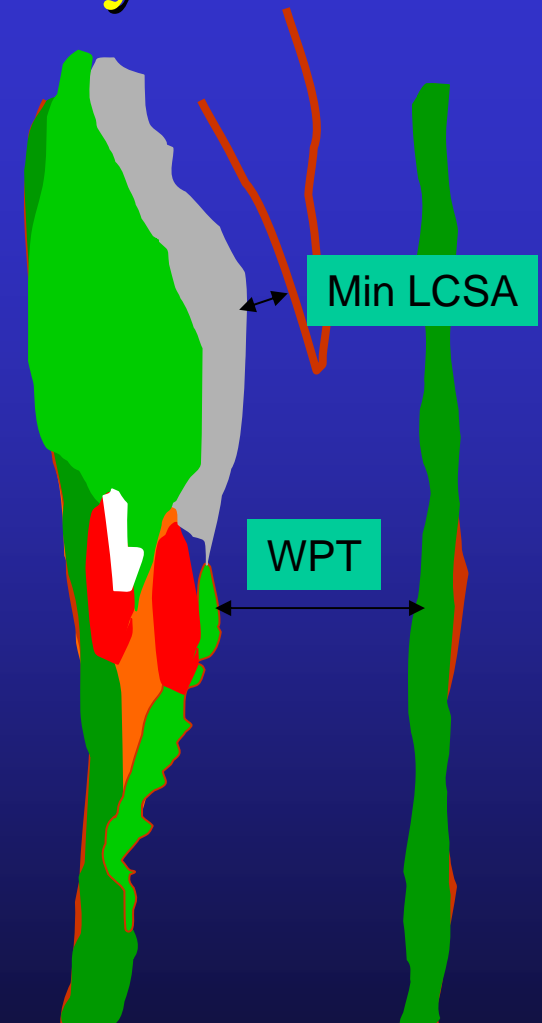
CAPITAL Study: Pathophysiology of Carotid Artery Disease by Histology, Grayscale and VH IVUS, and MSCT J ENDOVASC THER 2007;14:676-686

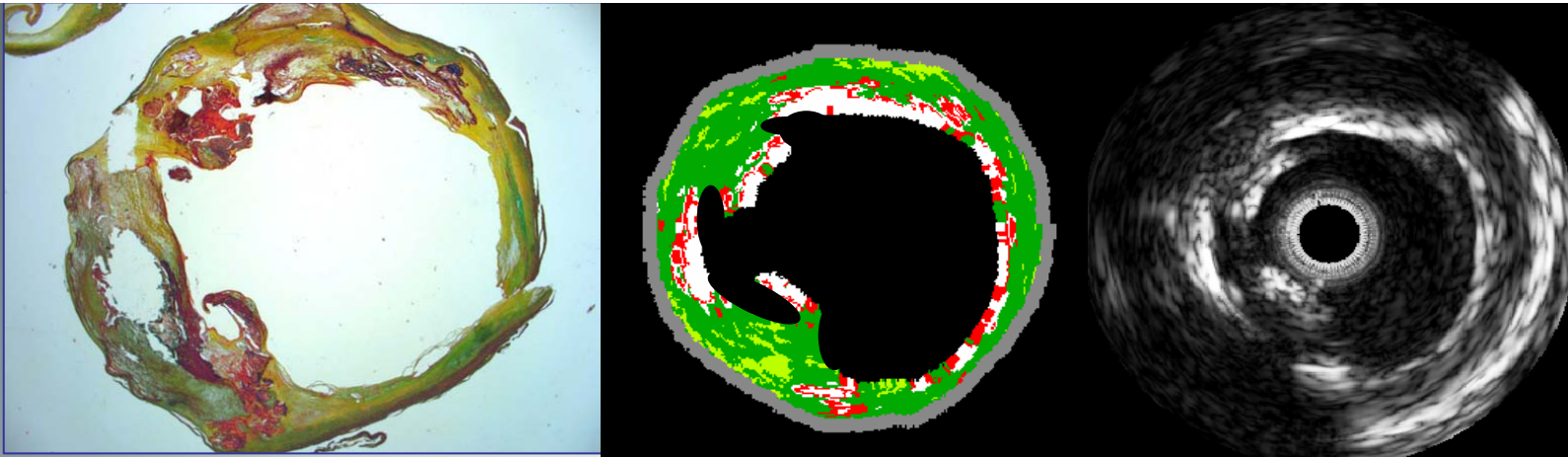
Edward B Diethrich, M.D. (PI)*, M Paulina Margolis, M.D., Ph.D. **, Donald Reid M.D., Venkatesh Ramaiah, M.D. *, Julio Rodriguez, M.D. *, Grayson Wheatley, M.D. *, Dawn Olson, P.A. *, Allen Burke, MD***, Renu Virmani, MD***. *Arizona Heart Institute, Arizona, Texas, **Volcano Corp. Rancho Cordova, ***CVPPath, Chevy Chase, MD, USA



Histology and VH IVUS Analysis

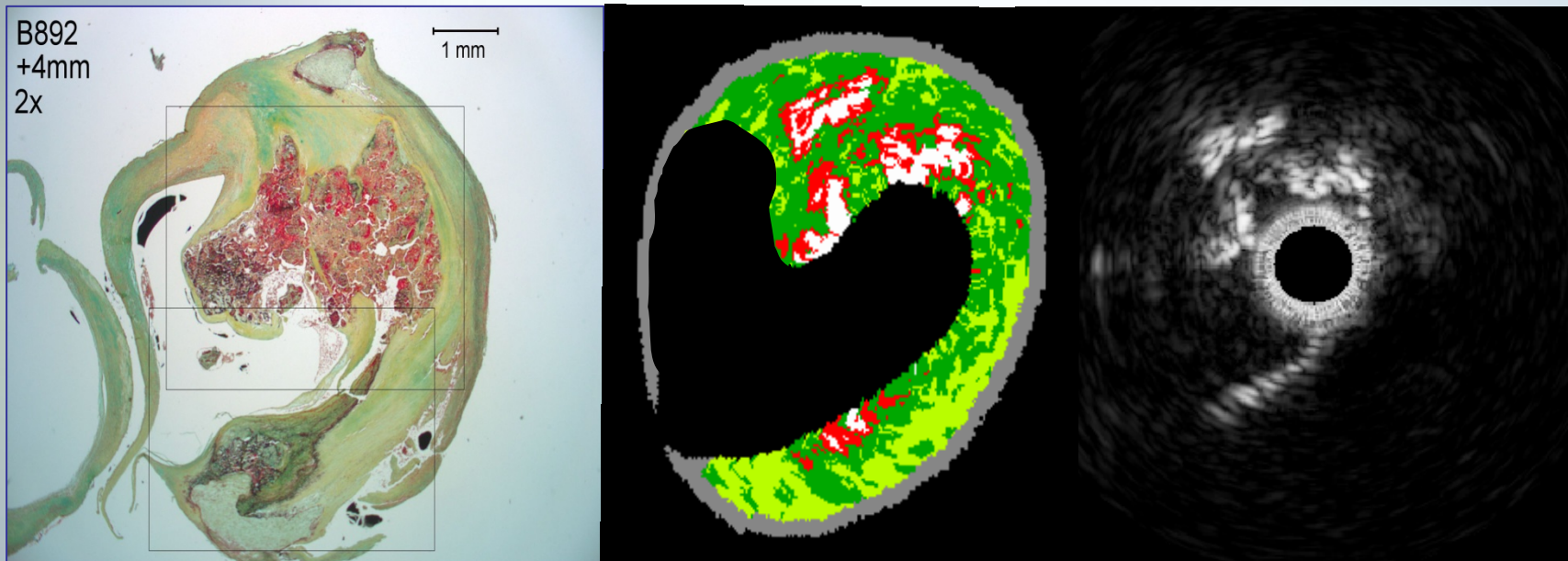
- X-ray of whole extracted plaque to assess presence of calcium was taken prior to histological processing.
- The extracted plaque was cut every 1-2 mm and its composition and type evaluated by independent pathologists.
- Grayscale and VH IVUS images and data (amount, composition, and type of plaque) were processed every 0.5 mm and matched with actual histology using anatomical landmarks.
- Any evidence of calcium was assessed by angiogram, MSCT, grayscale IVUS, VH IVUS, X-ray, and histology.
- Total of 158 sites were matched, mean 12.2 matched sites / lesion (range 3-20)
- Plaque type was assessed by histology and VH IVUS also at the site of min LCSA and at the site of “worst plaque type” (WPT) by classical histology classification (Stary et al 1995, Virmani et al 2000)





Ca Nodule

(Calcific Protrusion into the Lumen)





Ca Nodule

- 5/14 CEA lesions had a Ca nodule by both histology and VH IVUS
- 7/15 stented lesions had Ca nodule by VH IVUS
- Total of 41% of all cases had a Ca nodule

Summary

- Atherosclerotic plaques are formed in a similar manner in all coronary, carotid and peripheral arteries.
- A continuing sequence of inflammation, plaque rupture and thrombus formation is inherent to this process.
- Manifestations of these events differ in various vessels, because of vessel size, collateral circulation and end organ characteristics.
- Bifurcations show a particular predilection to plaque deposition due to turbulence causing areas of low shear stress at these sites.
- The site of plaque rupture is usually not the site of severest luminal narrowing, since narrowing is caused by organization of the fibrotic tale resulting from plaque rupture.

Summary - II

- Plaque ruptures are more likely to cause clinical events in coronary arteries than in peripheral arteries.
- Disease in the SFA and Popliteal arteries tends to be more active and more diffuse by the time clinical symptoms are manifest.
- Carotid lesions:
 - Are particularly influenced by the disturbed flow dynamics that are inherent to bifurcations.
 - Are usually manifest clinically by the effects of embolization.
 - Calcified nodules in the carotid artery are particularly prone to embolization.