

# Physiology Guided Complex PCI

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# “Physiology” for “Complex PCI”?

Physiology itself is complex. Therefore, any physiology-guided PCI is “Complex PCI”.

Invasive physiologic study is only for intermediate and simple lesion. There is no role of physiology for complex PCI.

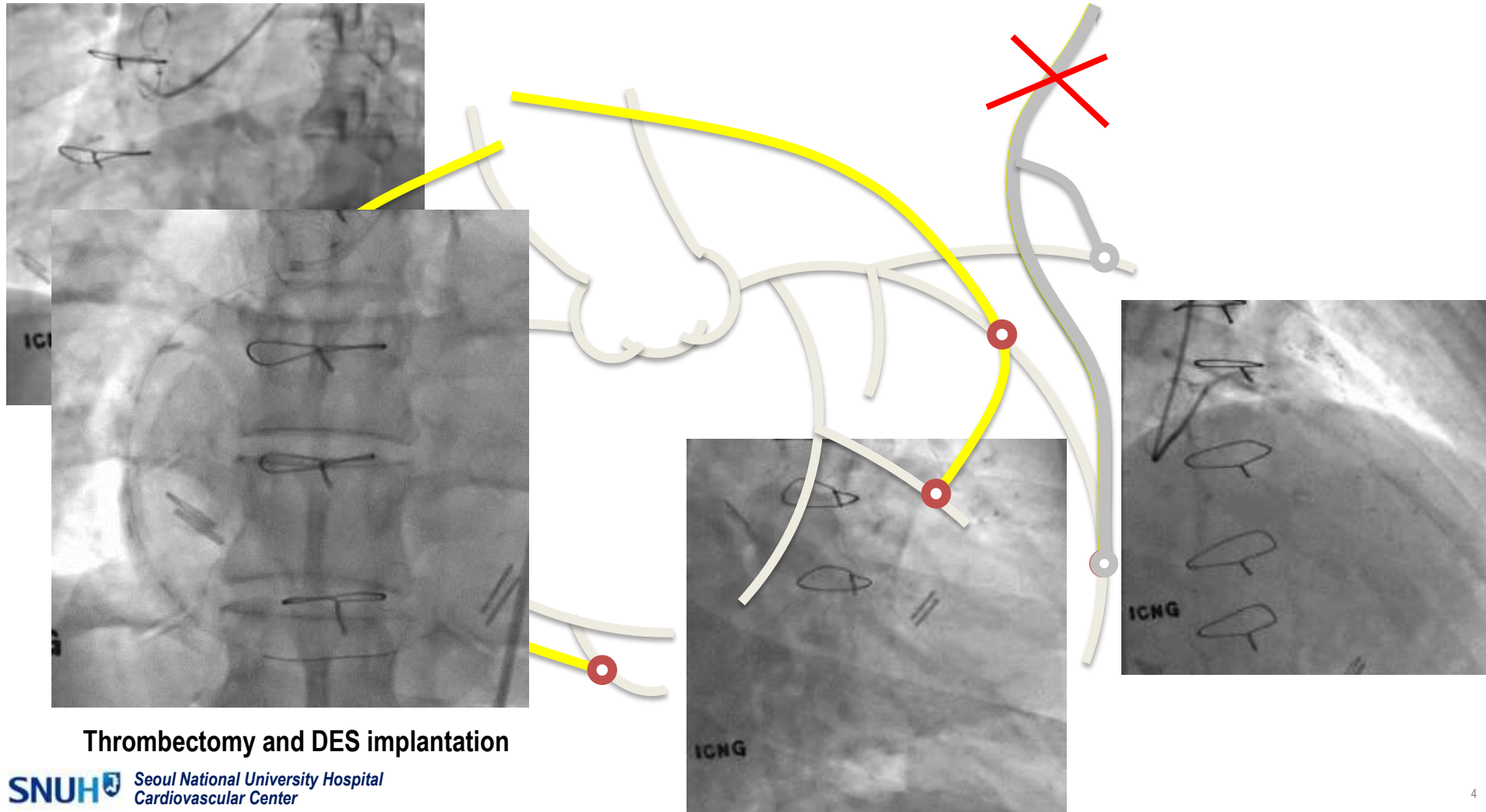


# Physiology for Complex lesions

- Defining ischemia-causing stenosis
- Reduction of un-necessary revascularization
- Selection of treatment strategy
- Risk stratification
  - Epicardial stenosis
  - Microvascular disease

**M/65**

- Aggravated angina, Hypertension(+) Diabetes mellitus (+)
- s/p coronary artery bypass surgery 20 years ago  
2 years later after the surgery → Arterial graft to LAD/Dg: totally occluded



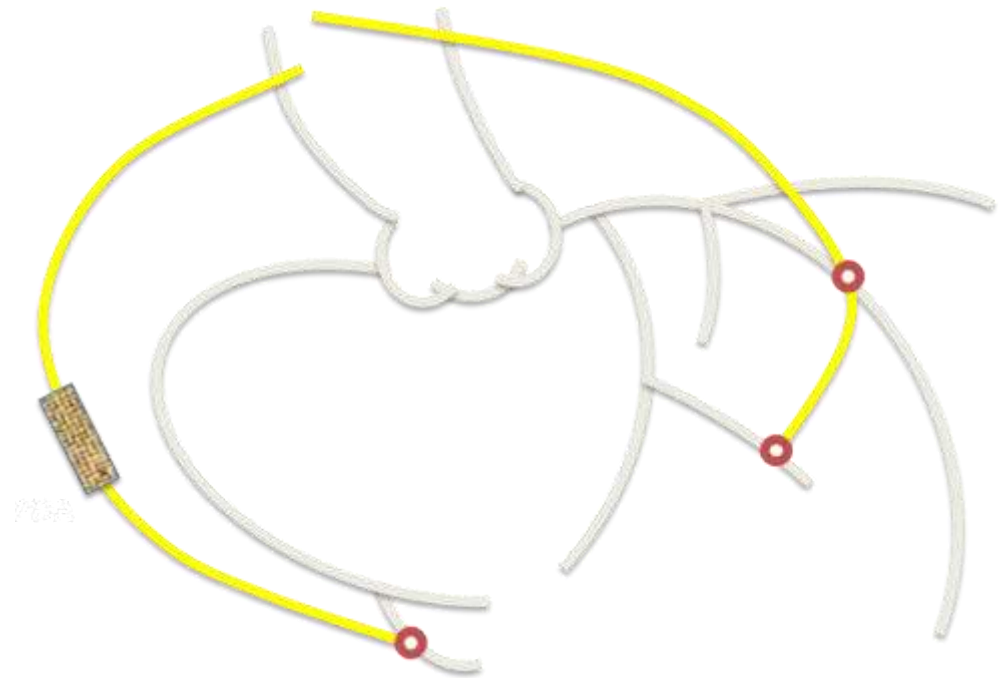
**Thrombectomy and DES implantation**

## ***Graft failure had been successfully treated, but...***

# Troublesome exertional chest pain was reported again at the following visit.

# Despite full-dose antianginal medications, patient complained angina.....

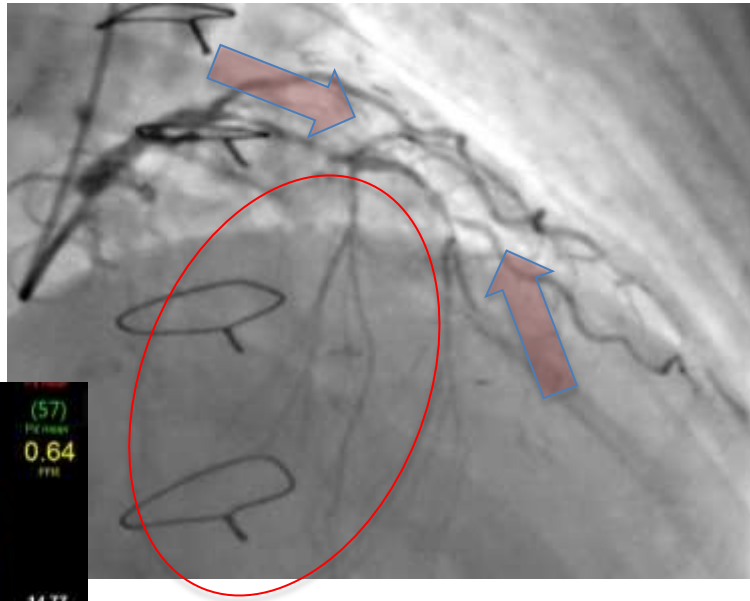
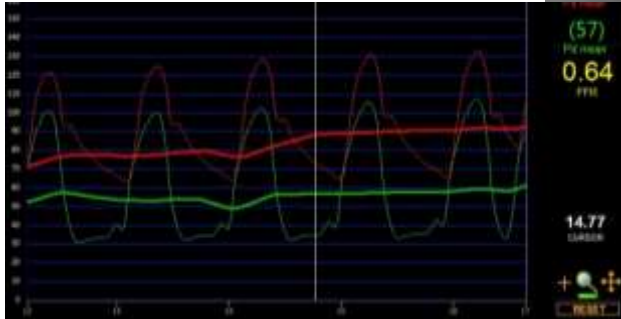
# **Why angina after “CABG” and “STENT”?**



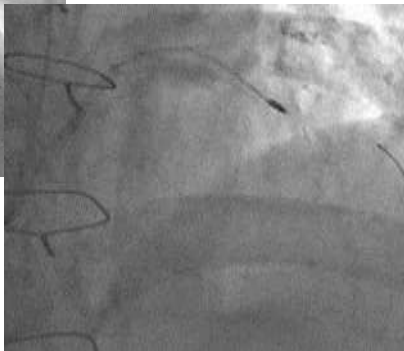
## Septal branch FFR

= 0.64

= 64% of normal flow is being supplied despite patent SVG and antegrade flow



**Not dilatable**

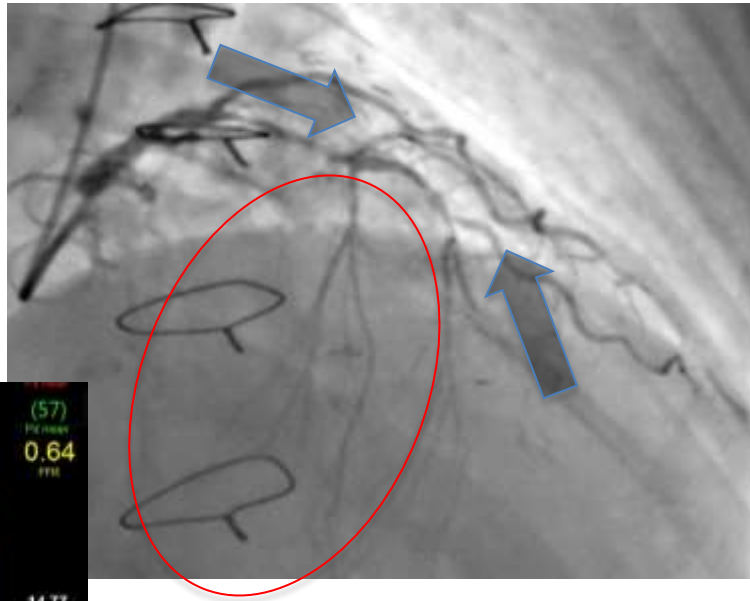
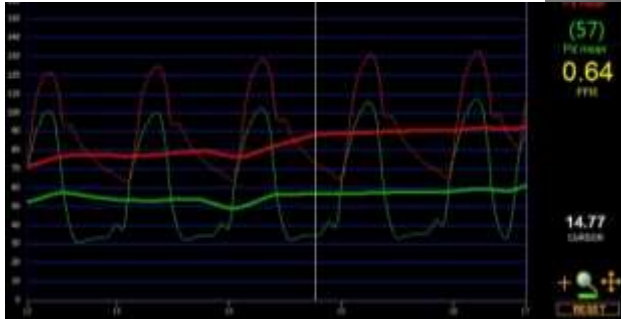


**Rotatable**

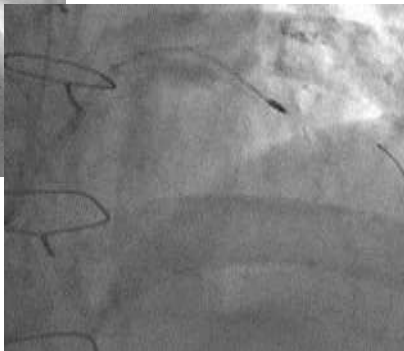
**Septal branch FFR**

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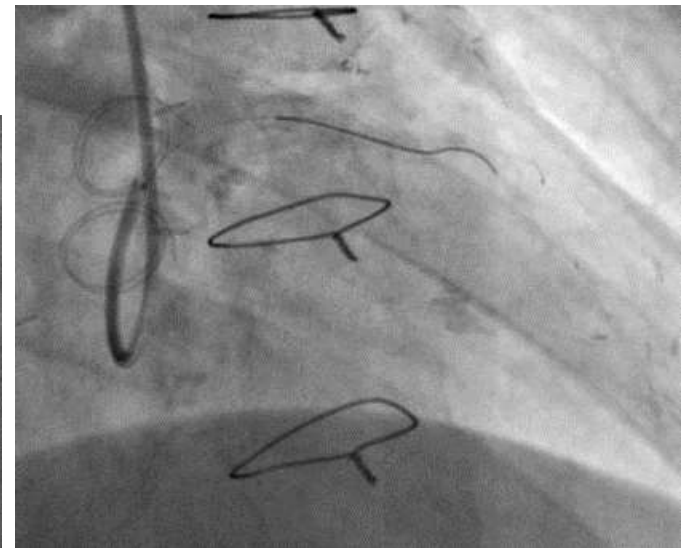
**Not dilatible**



**Rotablation**



**DES implantation**



# *Different world from different view*

Proportions of functionally diseased (ischemia+) coronary arteries  
in patients with angiographic 3 vessel disease



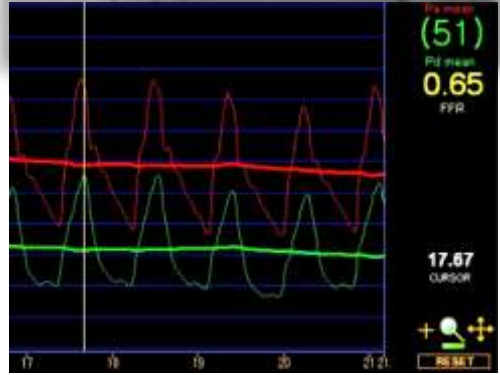
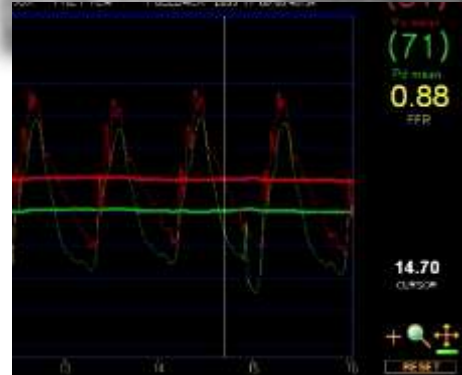
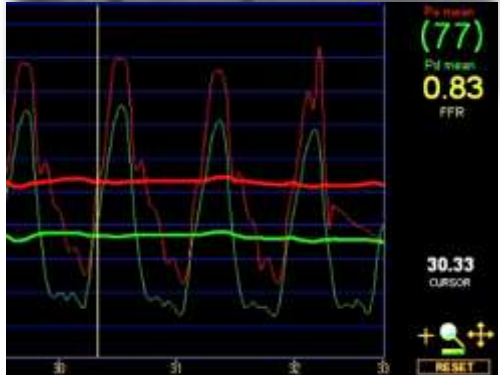
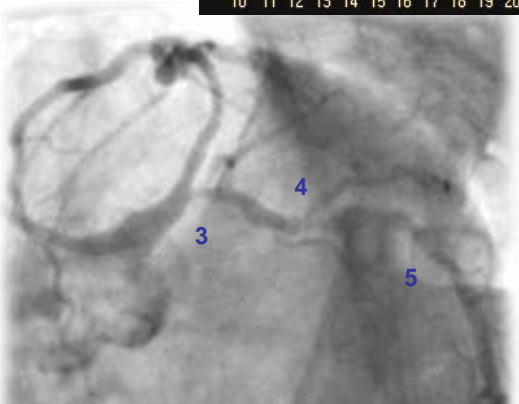
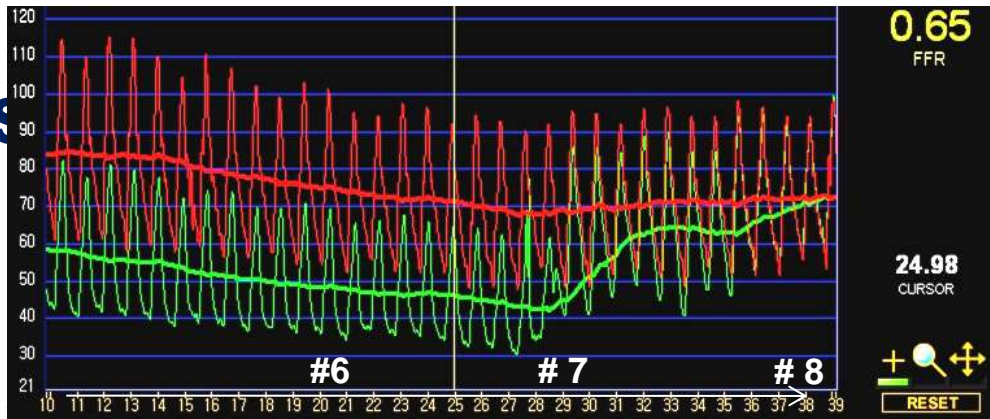
Tonino P, JACC 2010



# Patient with multi-ves

F/52

Stable angina

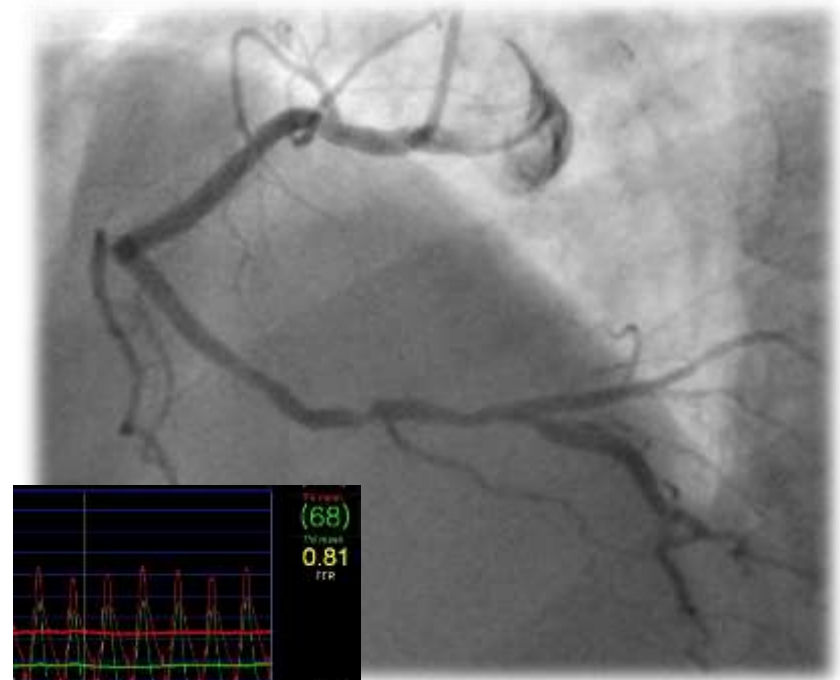
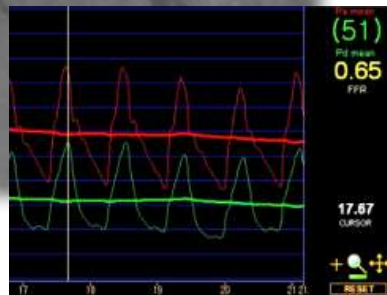
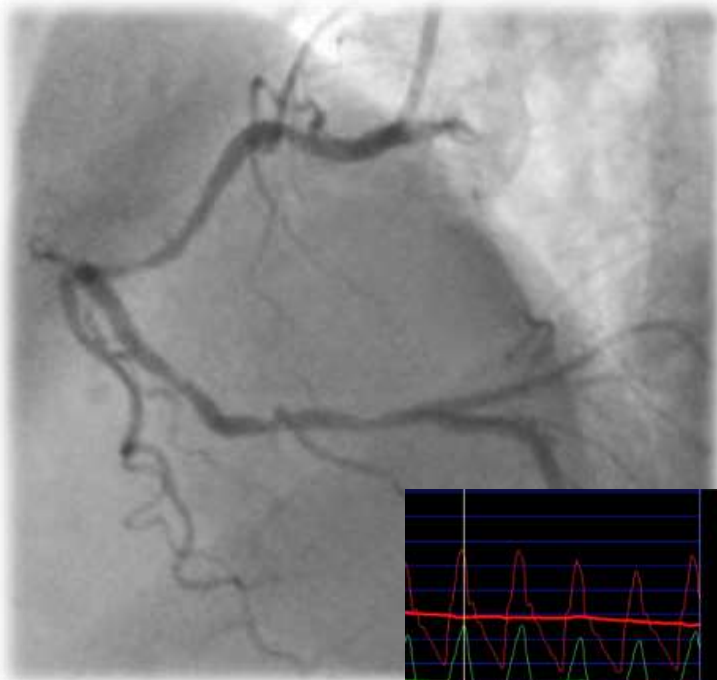


Distal left main disease + 3VD, 8 lesions

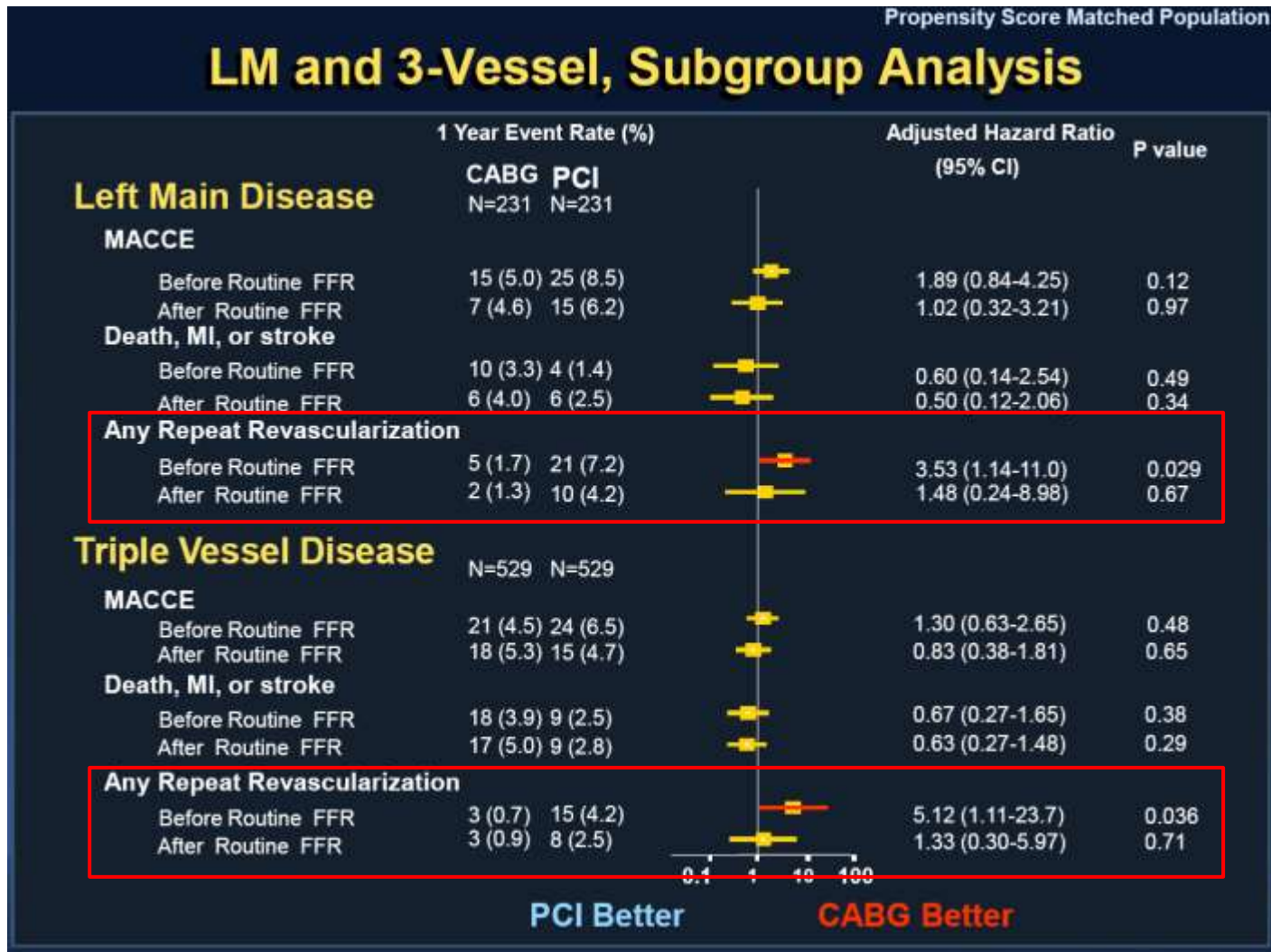
# Patient with multi-vessel, multi-lesion disease???

F/52

Stable angina, 3VD, 8 lesions by coronary angiography → 1VD, single lesion by FFR



# Changes of outcome after “Routine use of FFR”



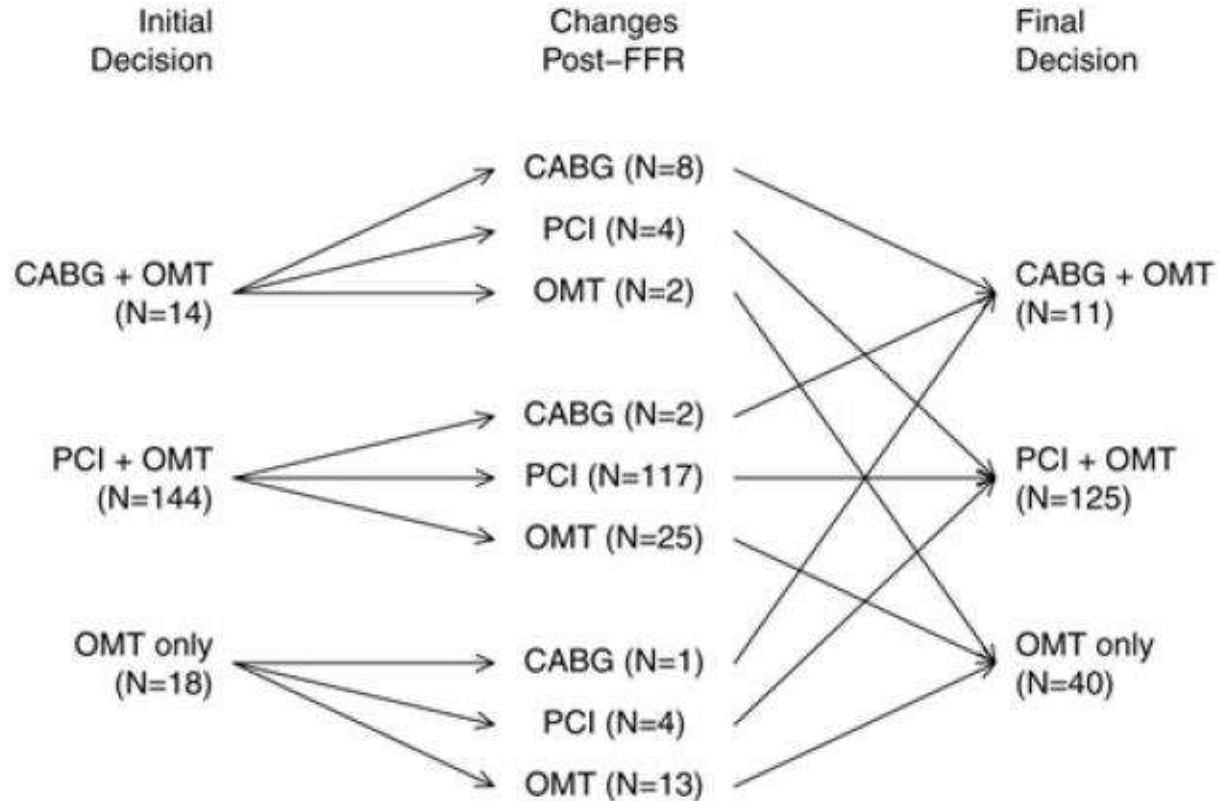
Courtesy of SJ Park, MD, PhD, Asan Medical Center  
Am J Cardiol 2015;116:1163-71

# How to use physiology for complex PCI?

- Defining ischemia-causing stenosis
- Reduction of un-necessary revascularization
- **Selection of treatment strategy**
- Risk stratification
  - Epicardial stenosis
  - Microvascular disease

# Changes in treatment plan after FFR measurement

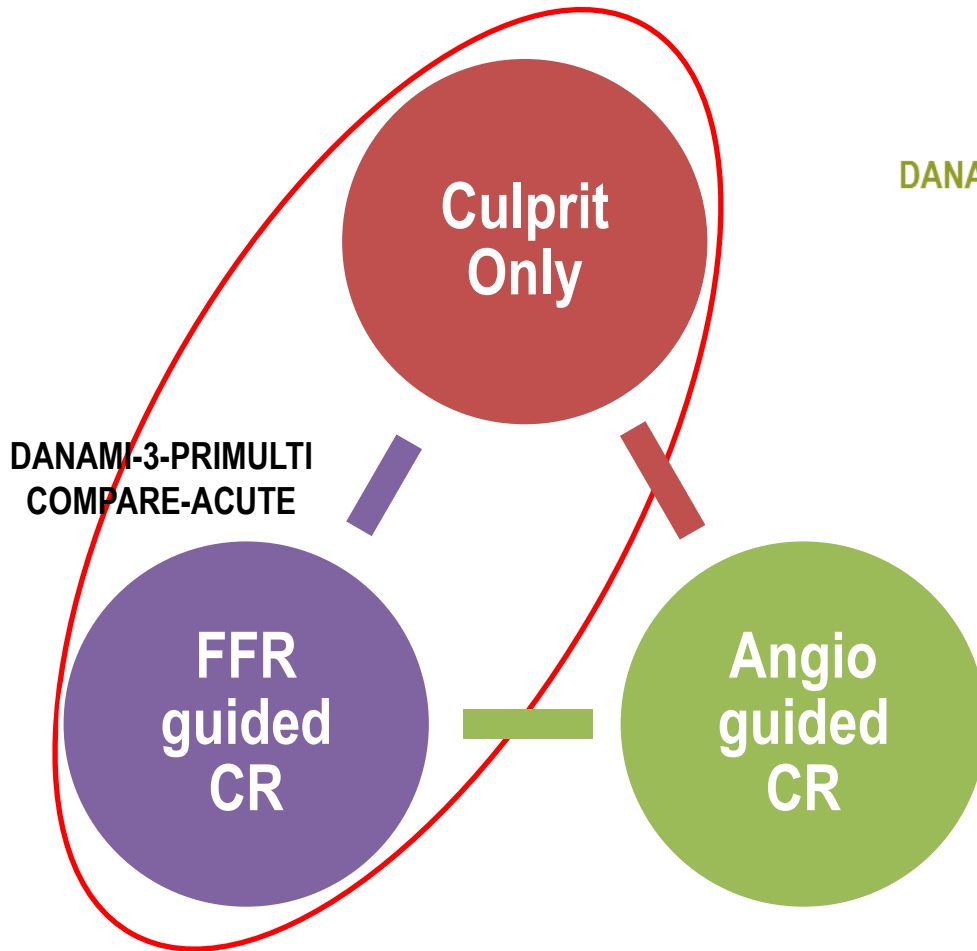
(350 NSTEMI, multi-vessel patients)



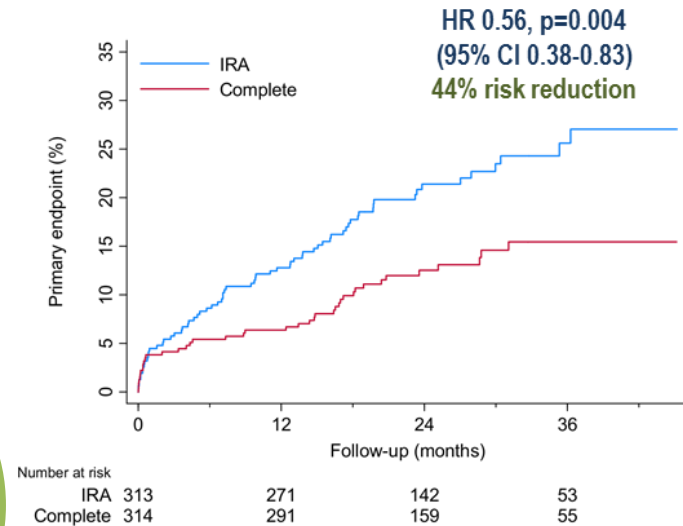
FFR-disclosure → Treatment plan change in 22%

Layland et al. EHJ 2014;36:100-11

# FFR-guided non-culprit lesion PCI in AMI



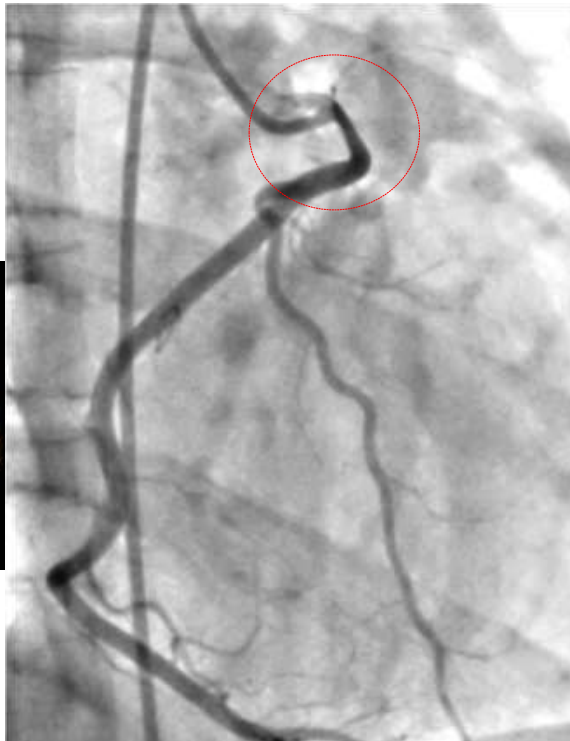
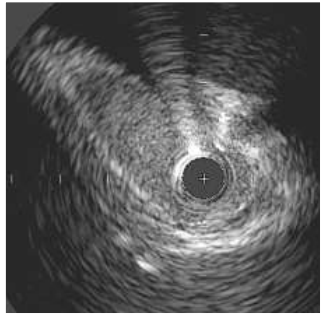
## DANAMI-3-PRIMULTI – all death, MI, ischemia-revascularization



Engstrom T, et al. Lancet 2015  
Smits PC, et al. NEJM 2017

# Complex physiologic study for complex anomaly

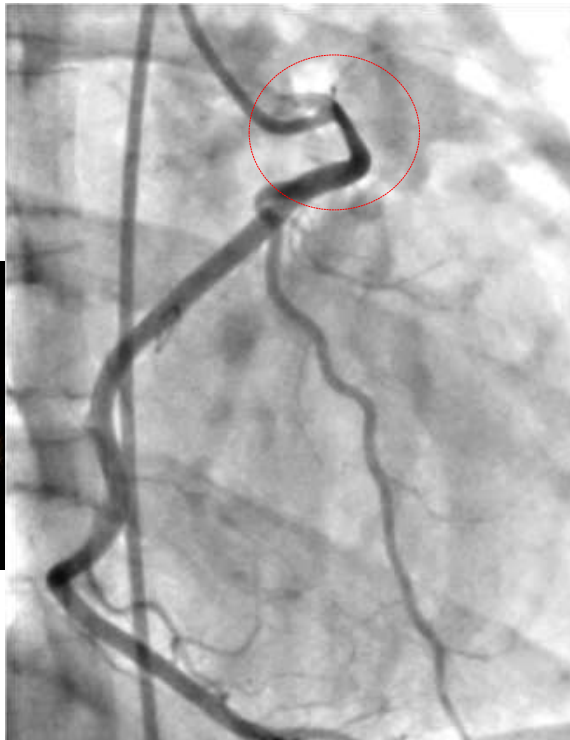
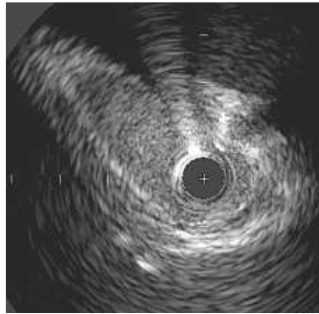
## Anomalous RCA from Left sinus



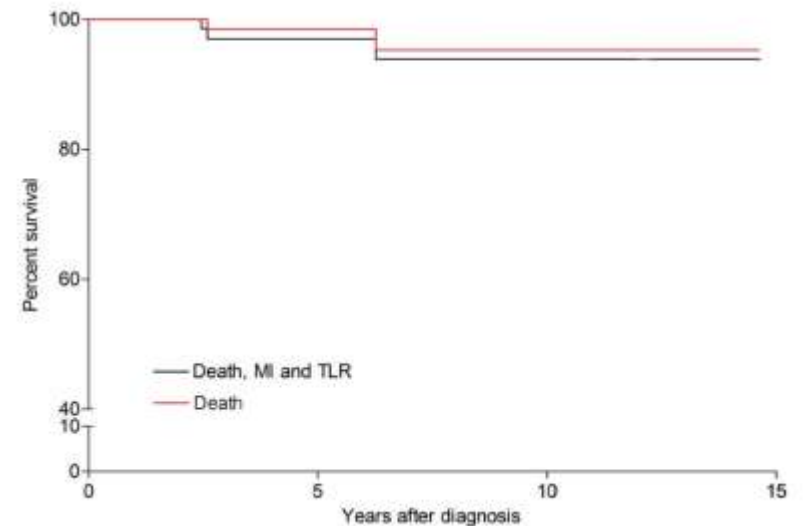
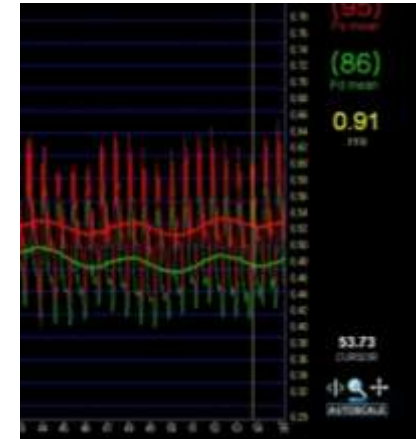
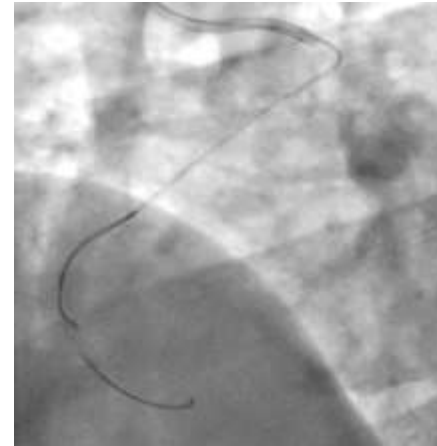
Park KI...Koo BK, *Can J Cardiol* 2011  
Lee SE... Koo BK, *Heart* 2015

# Complex physiologic study for complex anomaly

## Anomalous RCA from Left sinus



## Dobutamine + Atropine + Adenosine



Park KI...Koo BK, *Can J Cardiol* 2011  
Lee SE... Koo BK, *Heart* 2015

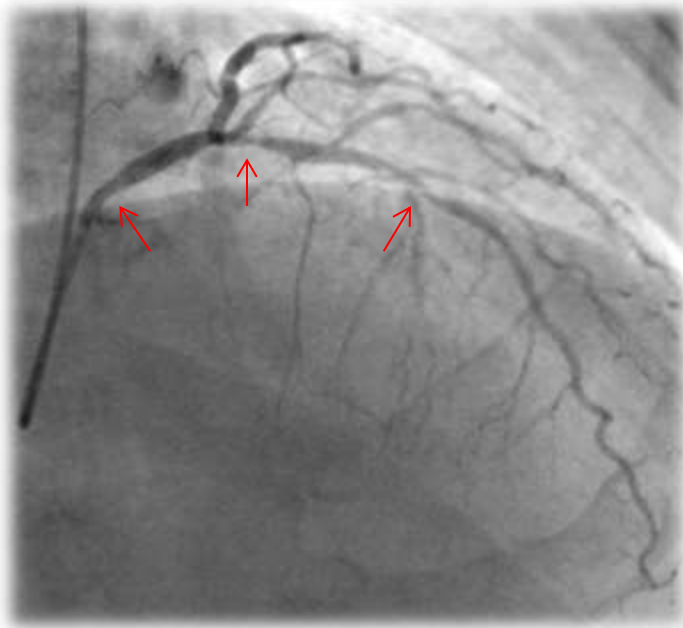


# How to use physiology for complex PCI?

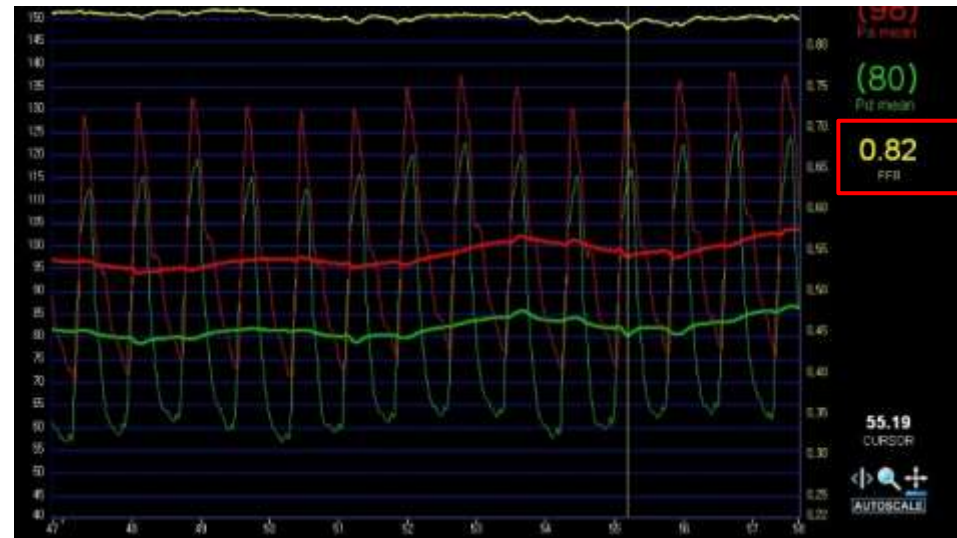
- Defining ischemia-causing stenosis
- Reduction of un-necessary revascularization
- Selection of treatment strategy
- **Risk stratification**
  - **Epicardial stenosis**
  - **Microvascular disease**

# SYNTAX score vs. Functional SYNTAX score

: Counting only the lesions in the functionally significant vessels



**SYNTAX score:** LM ostial + proximal  
LAD + mid LAD bifurcation lesions **> 22**

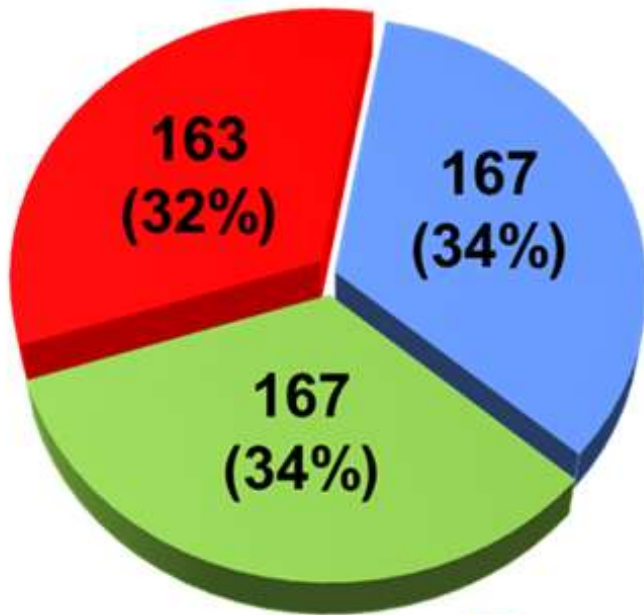


**Functional SYNTAX score = 0**

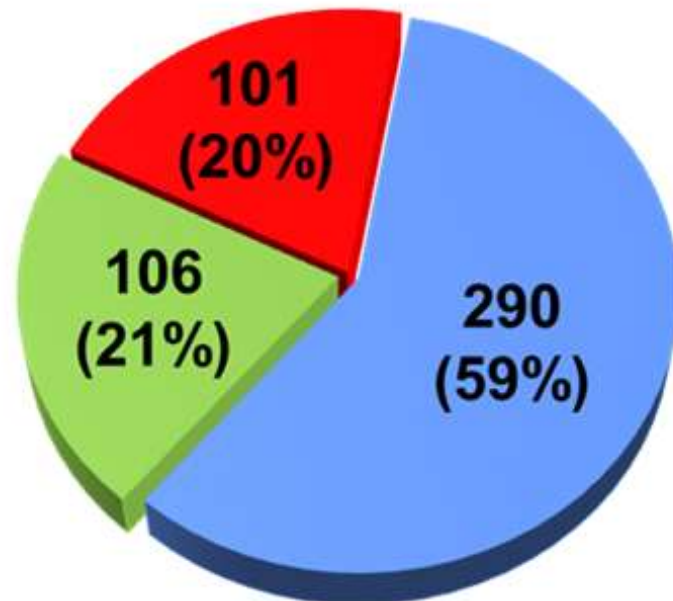
# SYNTAX score vs. Functional SYNTAX score

: FAME study subjects

## SYNTAX score



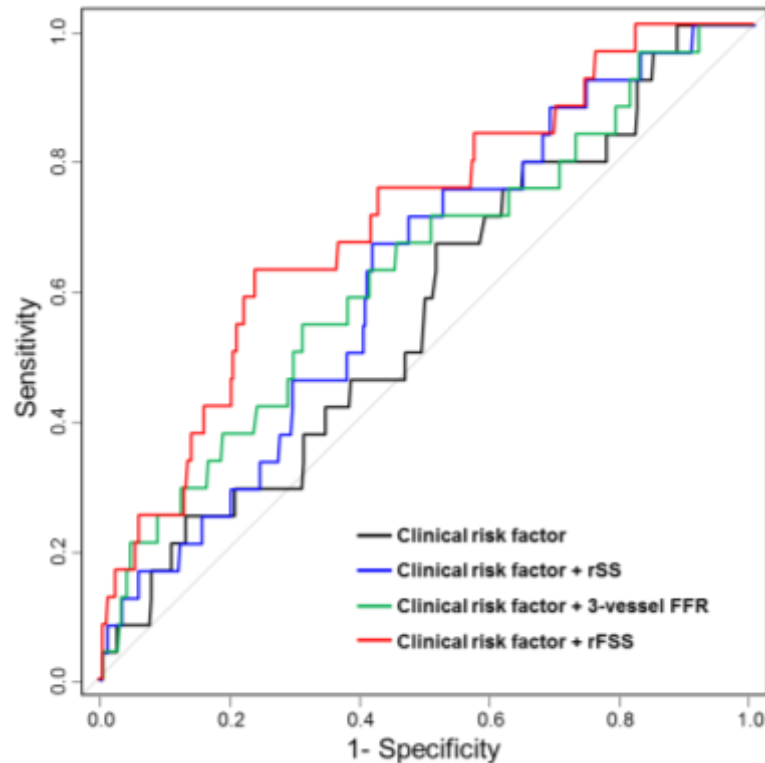
## Functional SYNTAX score



- Low SYNTAX score
- Medium SYNTAX score
- High SYNTAX score

# Prediction of clinical outcome

Clinical Risk Factor, Residual SYNTAX score, 3-vessel FFR, Residual Functional SYNTAX score



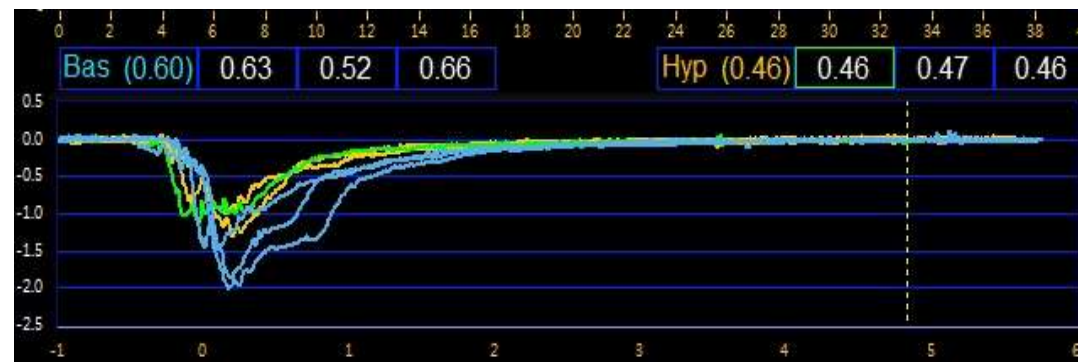
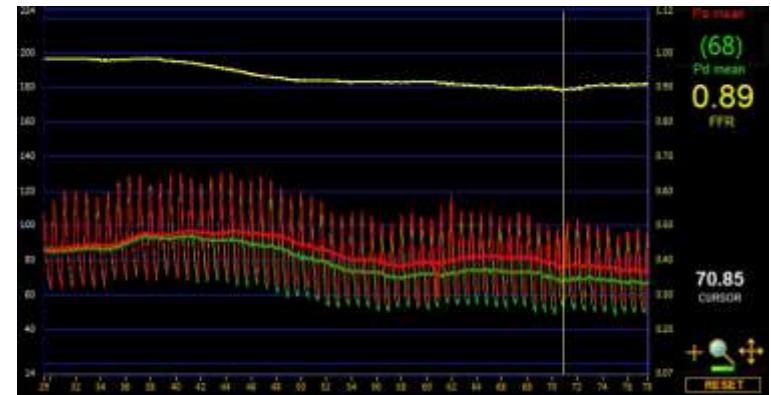
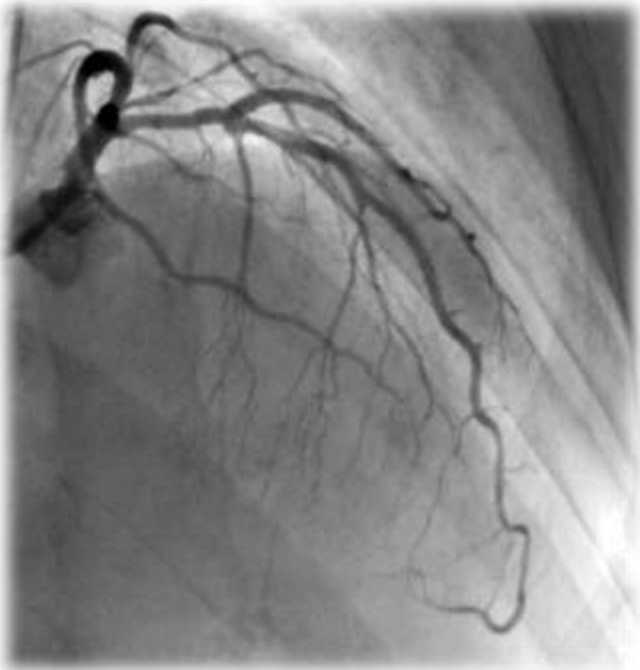
Model	C index	P	NRI	P	IDI	P
Clinical risk factor*	0.563	0.301	Ref		Ref	
Clinical risk factor + rSS	0.618	0.053	0.336	0.101	0.7%	0.122
Clinical risk factor + 3-vessel FFR	0.625	0.041	0.342	0.087	1.0%	0.047
<b>Clinical risk factor + rFSS</b>	<b>0.701</b>	<b>&lt;0.001</b>	<b>0.679</b>	<b>0.001</b>	<b>3.5%</b>	<b>0.002</b>

Choi KH, Lee JM.... Koo BK. JACC intervention, in press

Lee JM, Koo BK et al. Eur Heart J 2017

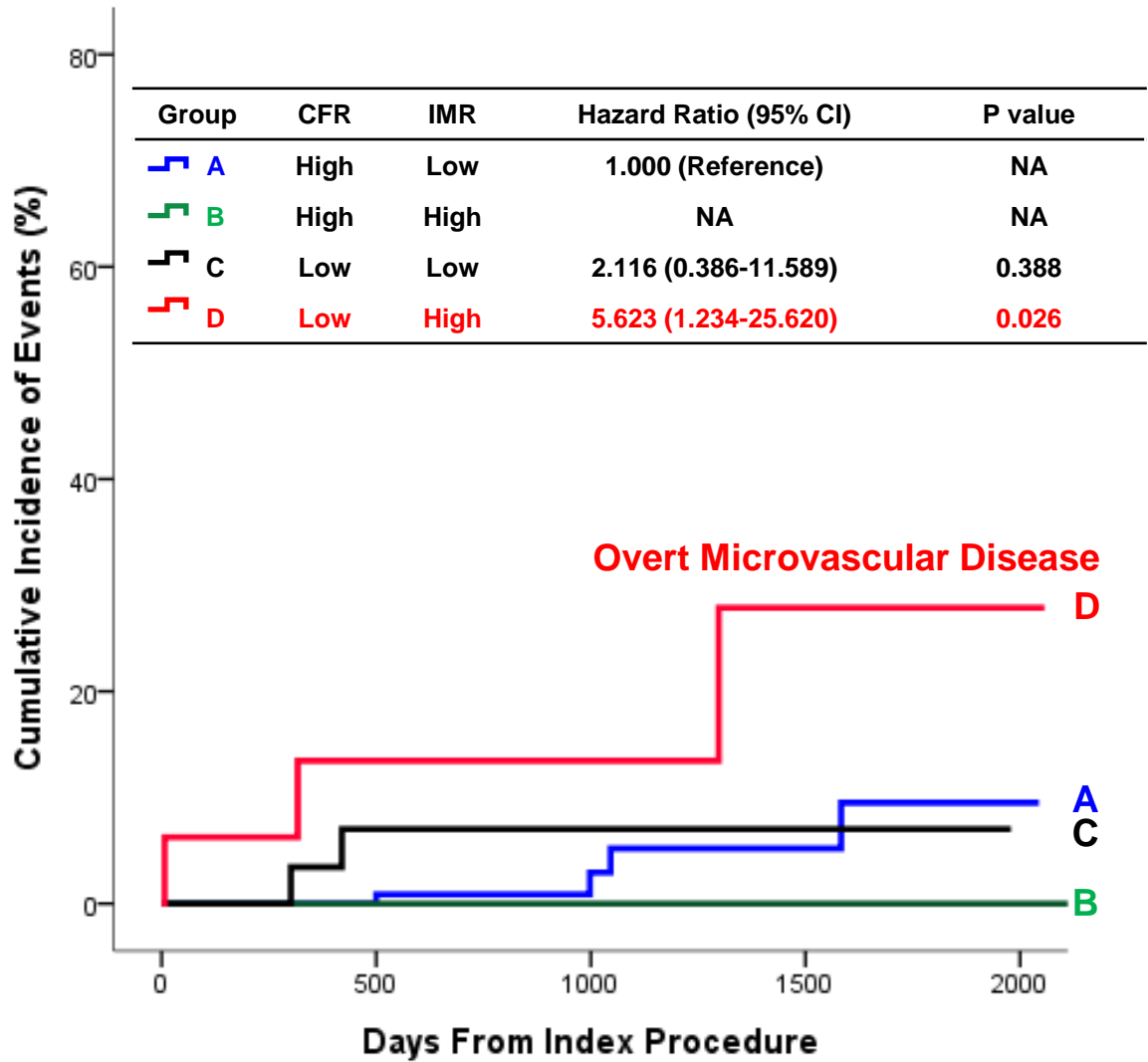
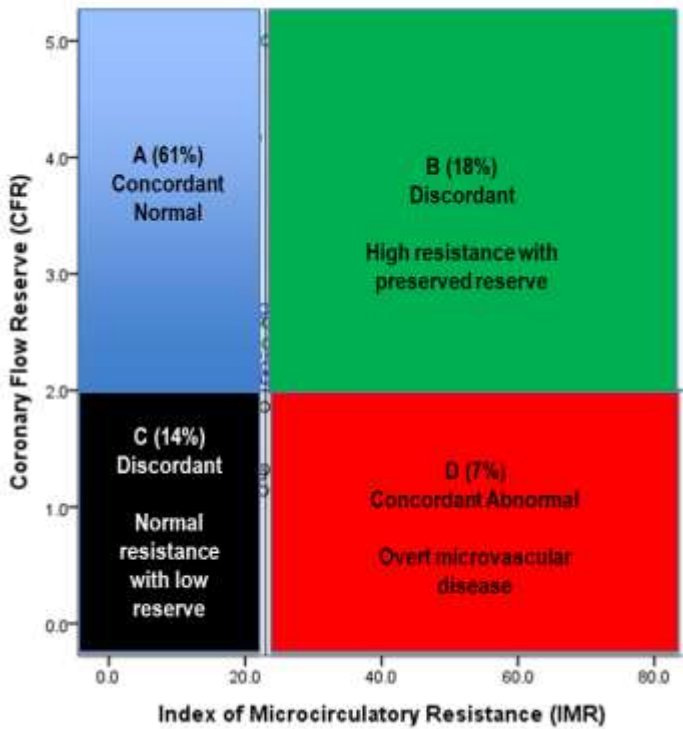
# Why angina and ischemia in this case?

- Hidden disease
- Diffuse coronary atherosclerosis
- Microvascular dysfunction



$$\text{IMR} = \text{Pd} \times \text{Tmn} = 68 \times 0.46 = 31.3$$

# Importance of microvascular assessment

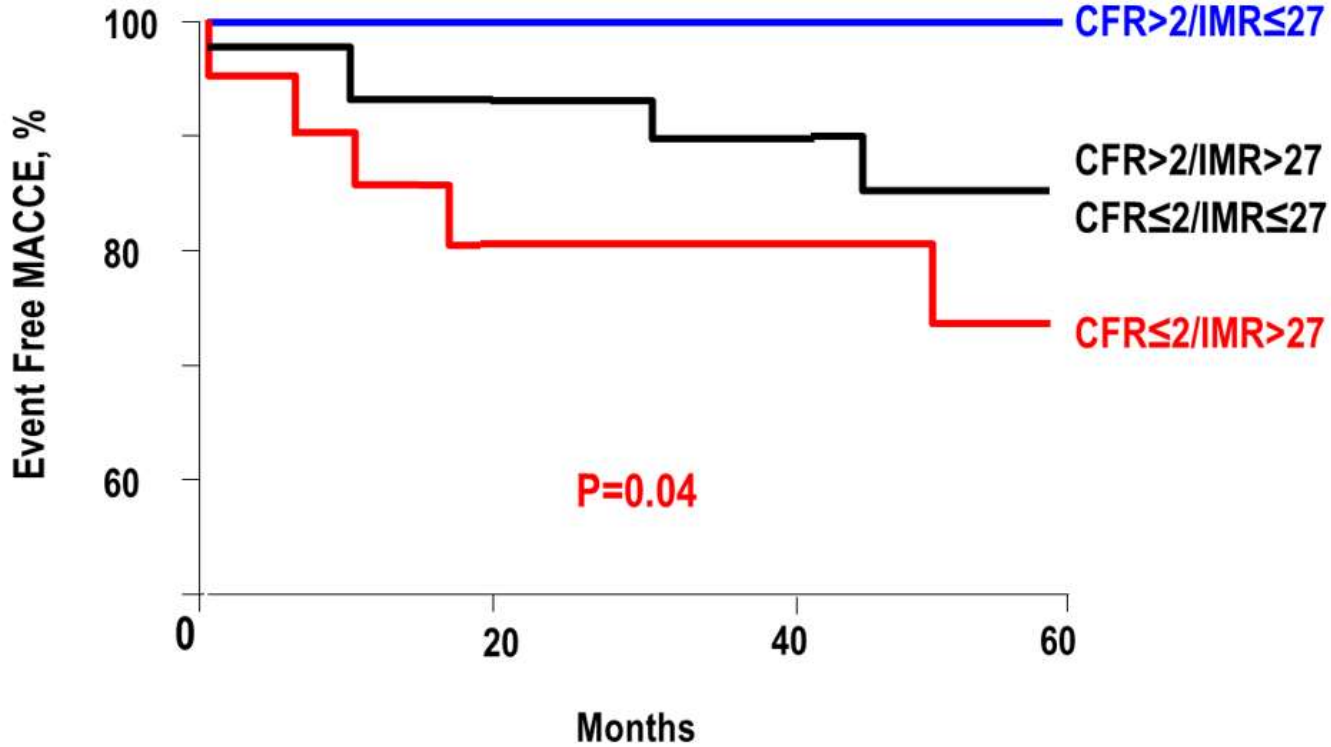


Lee JM , Koo BK et al. JACC 2016

# Assessment for microvascular injury after primary PCI for AMI

Cardiac MRI

After 1' PCI



Park SD et al Coronary Artery Disease 2016, 27:34–39

Overt MV damage(-)

# Physiology-guided complex PCI

- “Coronary physiology (or physiologic assessment) may be complex, but it is an essential element in understanding the patient’s disease status and clinical decision making.
- Clinical application of FFR/iFR and its extended concept can provide better stratification and management for patients with complex coronary artery disease.