

Tandem lesions, diffuse disease

FFR and resting physiology

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Disclosure Statement of Financial Interest

Within the past 12+ months, Nils Johnson has had a financial interest/arrangement or affiliation with the organization(s) listed below.

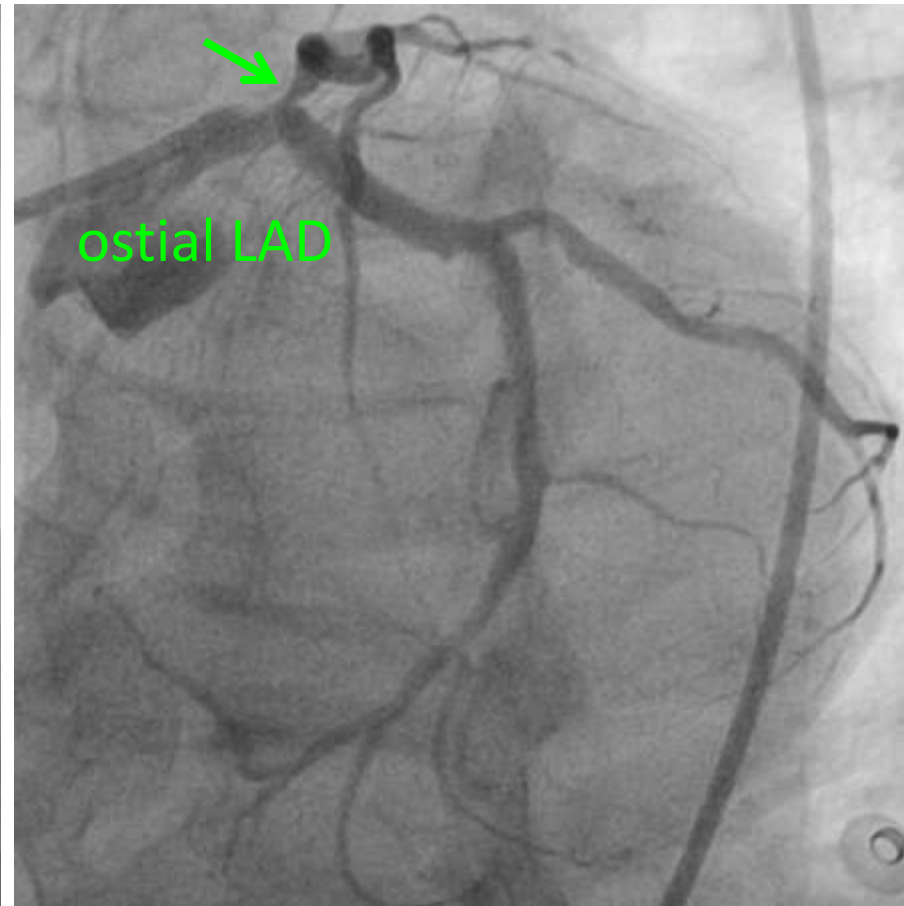
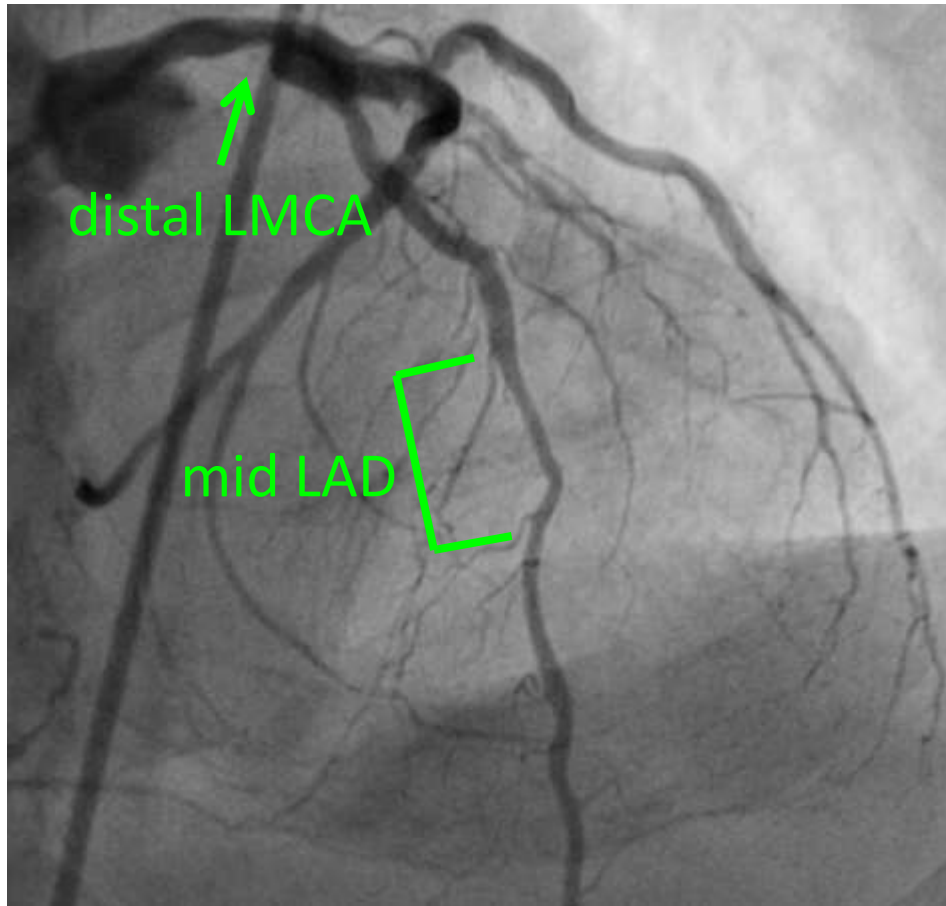
Affiliation/Financial Relationship

- Grant/research support
(to *institution*)
- Licensing and associated consulting
(to *institution*)
- Support for educational meetings/training
(honoraria/fees donated to *institution*)

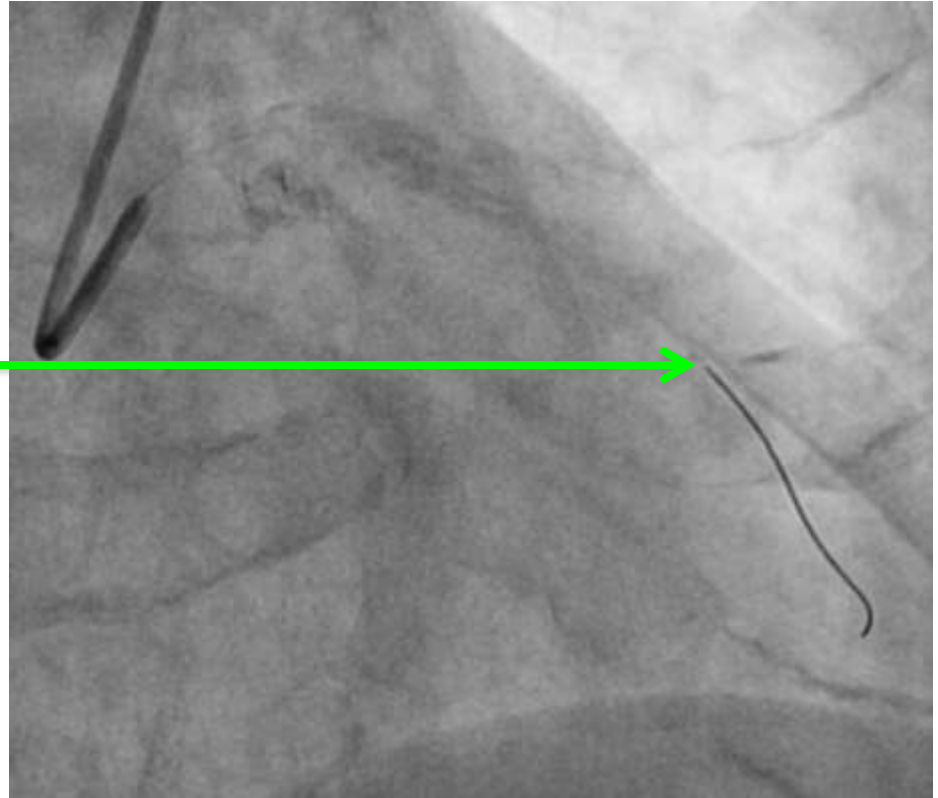
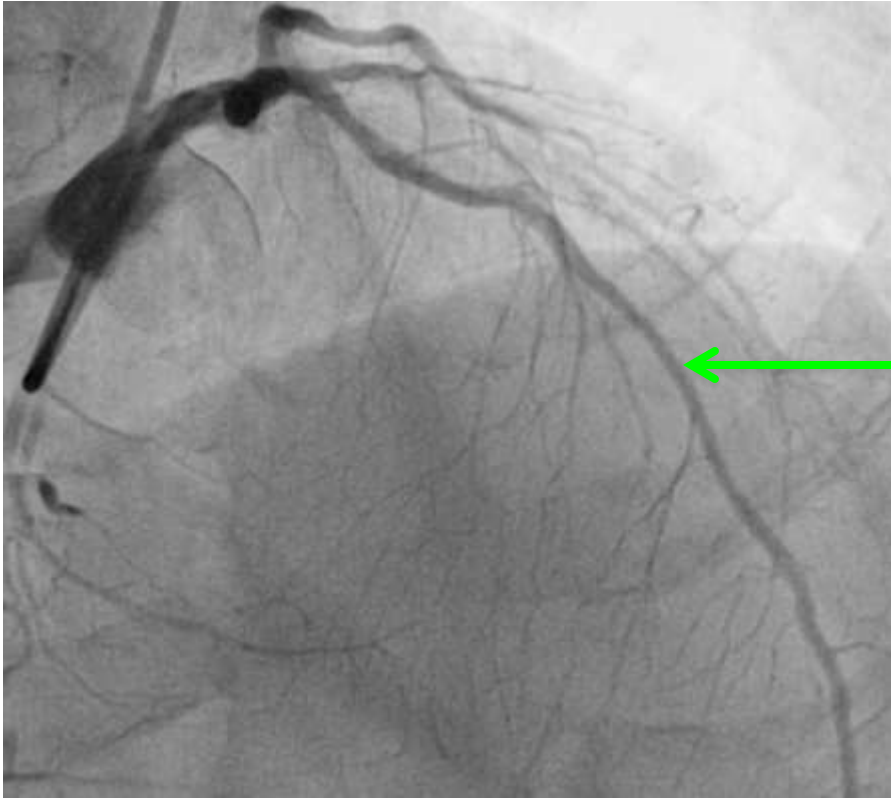
Organizations (alphabetical)

- St Jude Medical (for CONTRAST study)
- Volcano/Philips (for DEFINE-FLOW study)
- Boston Scientific
(for smart-minimum FFR algorithm)
- Various, including academic and industry

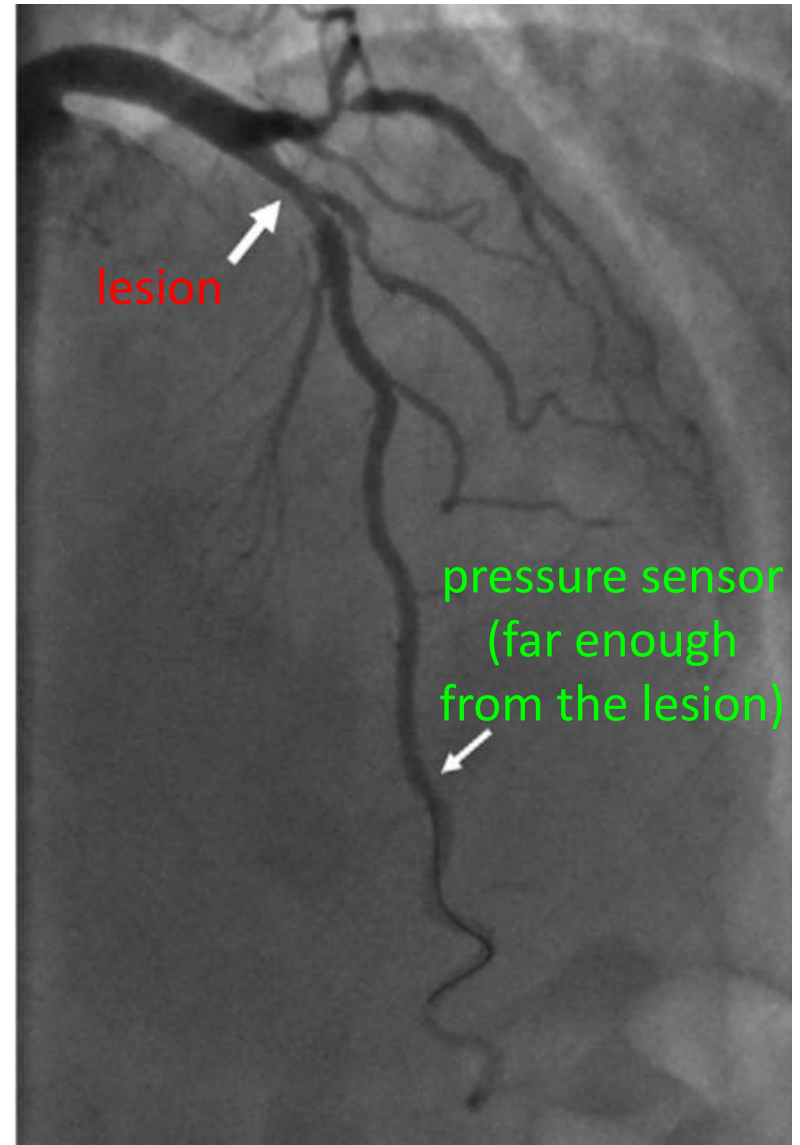
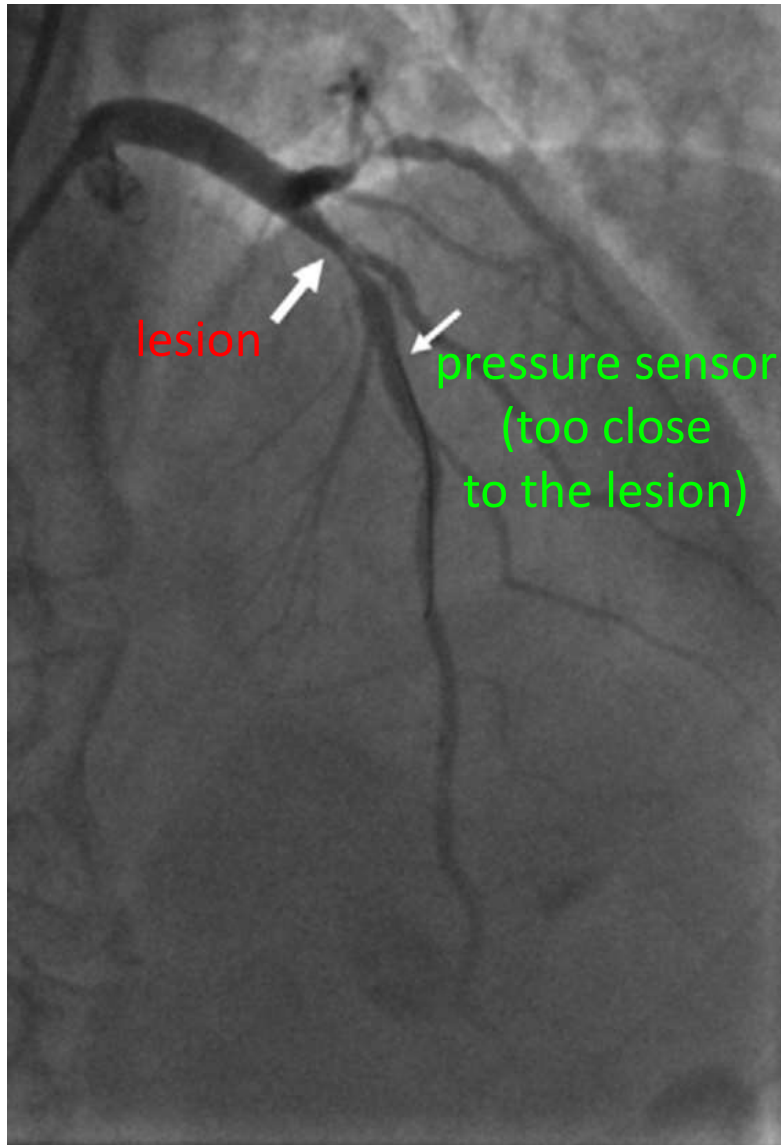
Serial LMCA and LAD lesions



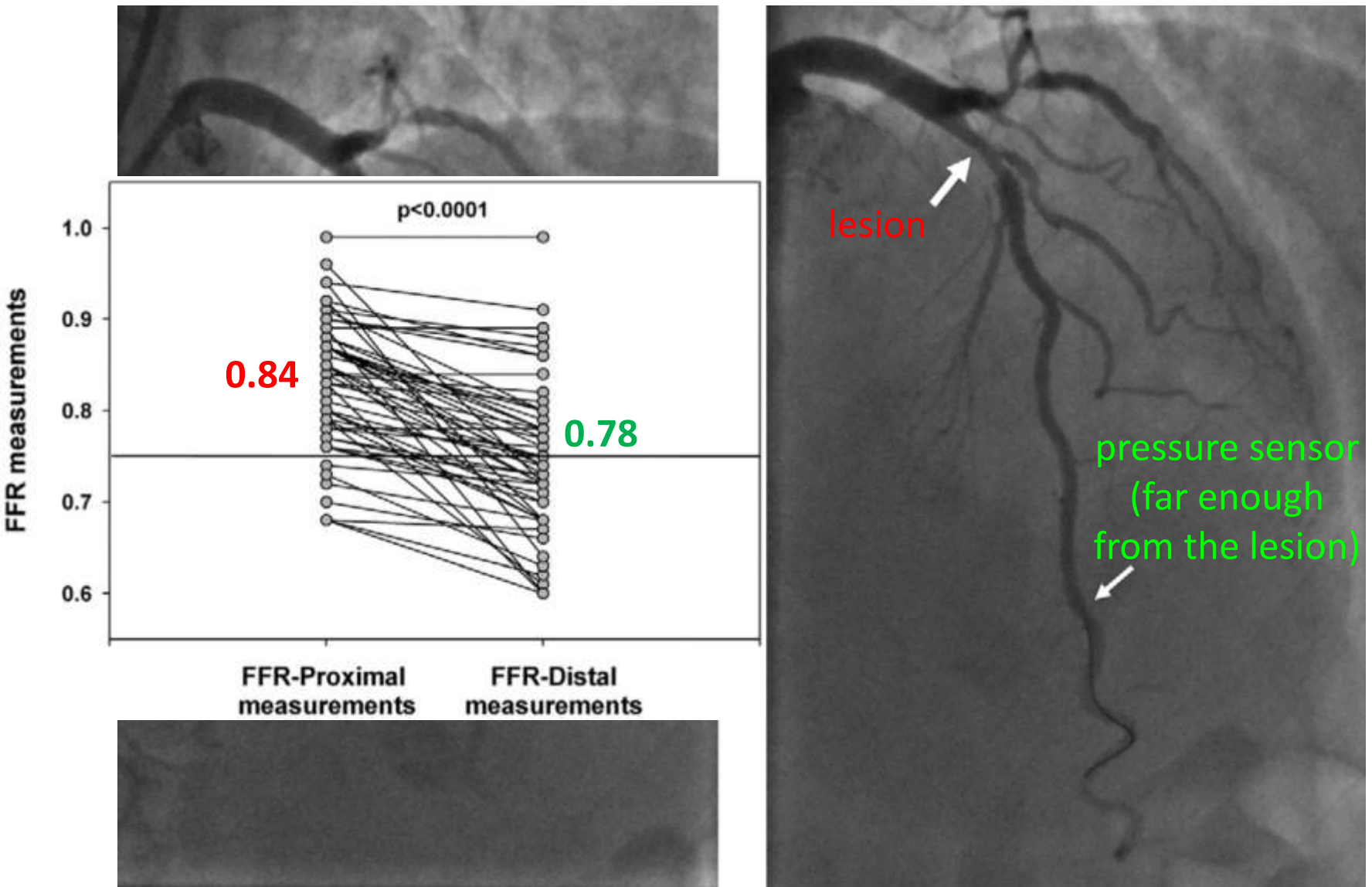
Place pressure wire *distal* to all lesions



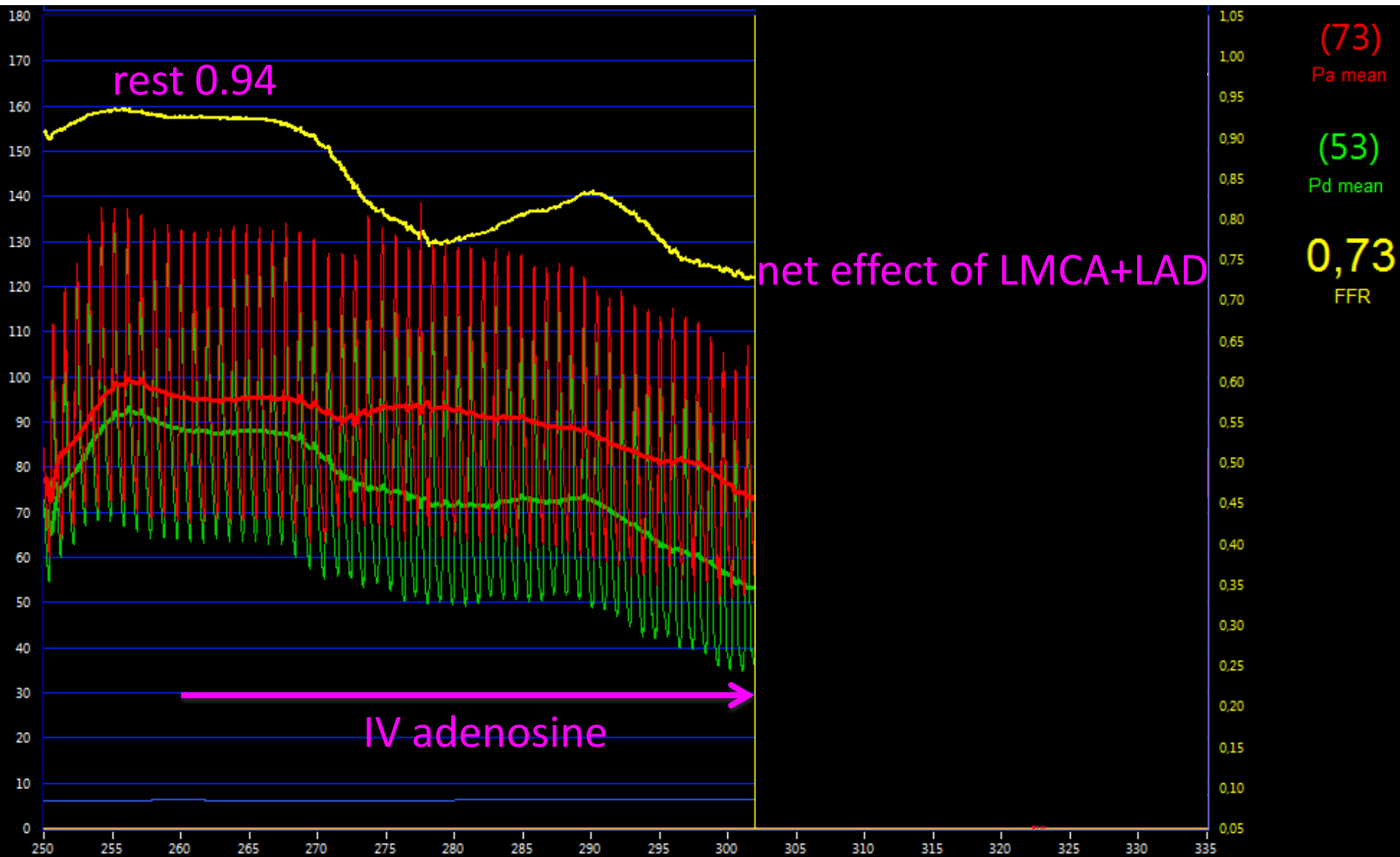
How distal is far enough?



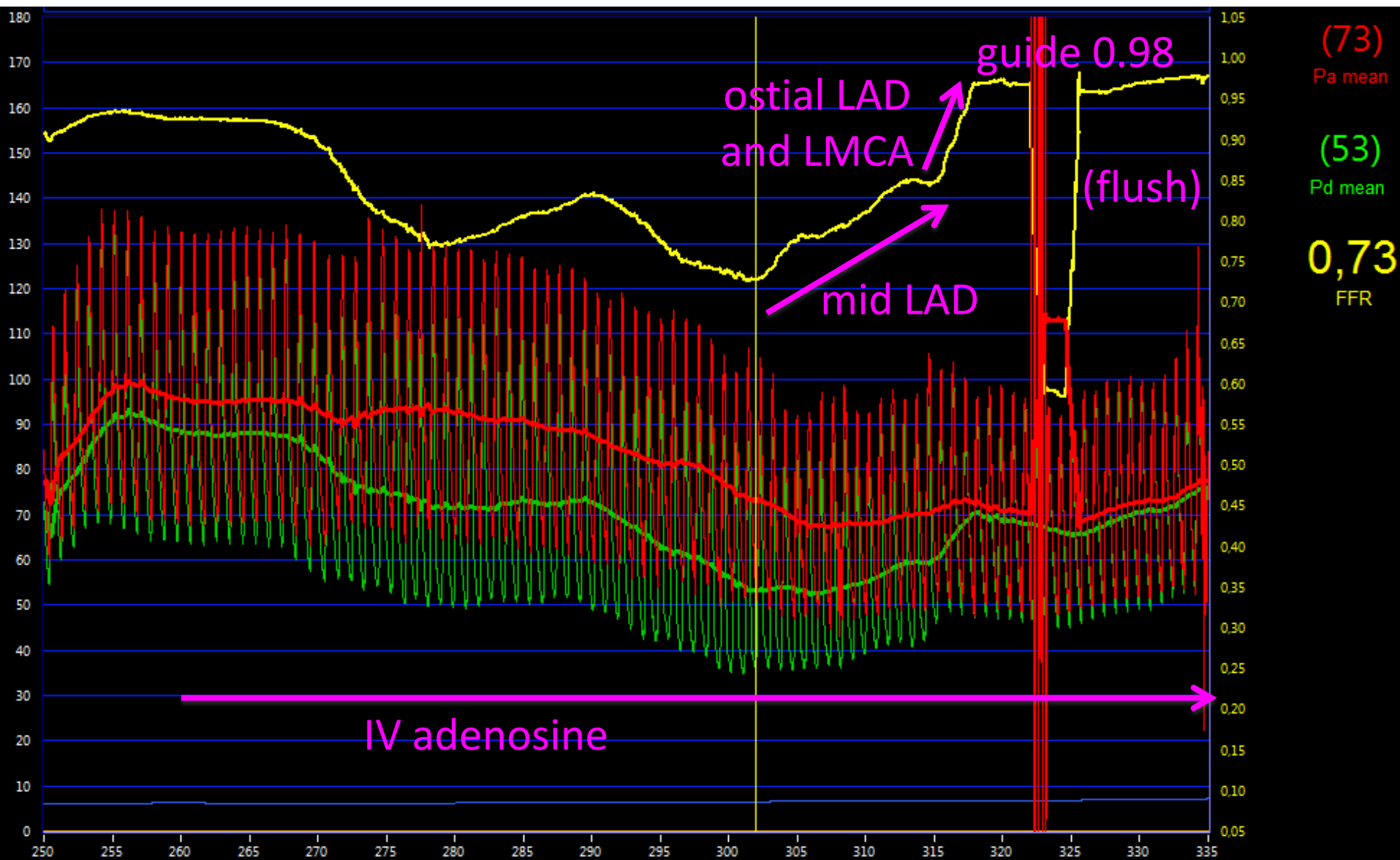
Most distal = all focal + diffuse



Start IV adenosine to assess *net* effect



Move pressure sensor back to guide



Pullback curve: fundamental technique

1996 NEJM

2000 review

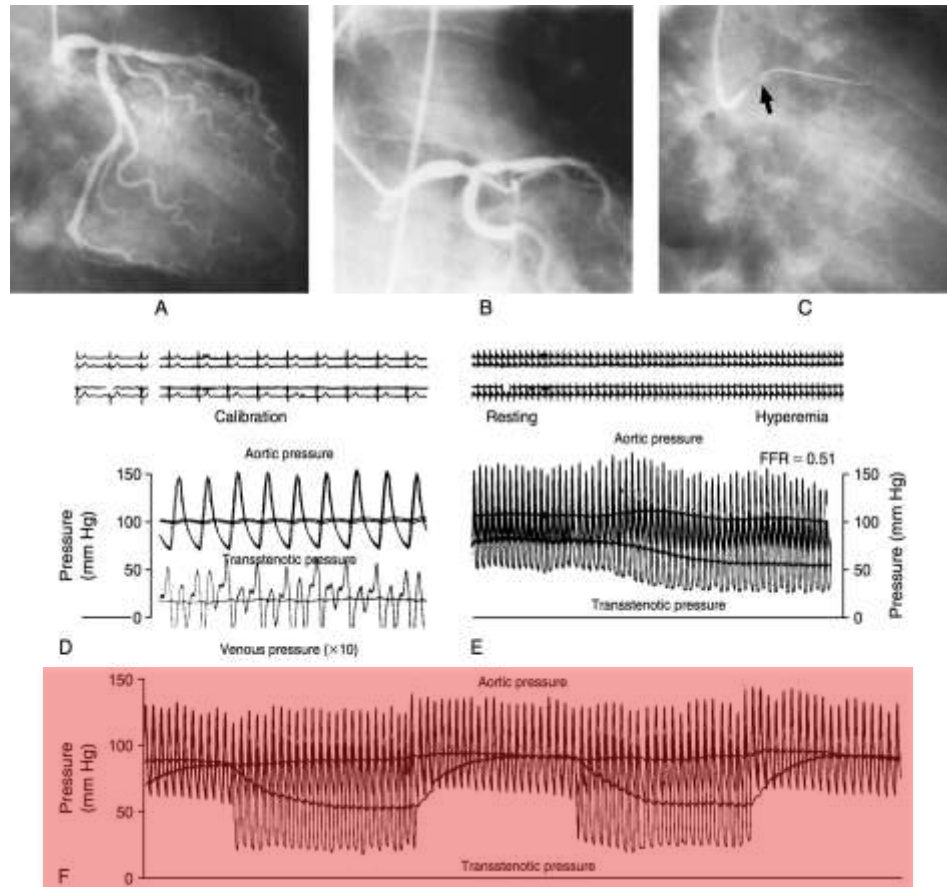
Practice and Potential Pitfalls of Coronary Pressure Measurement

Nico H.J. Pijls,^{1*} MD, PhD, Morton J. Kern,^{2†} MD, Paul G. Yock,^{3‡} MD and Bernard De Bruyne⁴ MD, PhD

Pullback Pressure Recordings

The most convincing and reproducible demonstration of the exact location and severity of a coronary stenosis is the so-called pressure pullback curve (Fig. 5).

If a large resting gradient is already present and the main purpose of the pullback recording is to determine the exact location of the stenosis, additional hyperemia is not required during the pressure pullback curve. The pullback curve also enables the assessment of serial lesions within one vessel (Fig. 6) or the presence of diffuse disease (gradually decreasing hyperemic pressure gradient during pullback from distal coronary artery to coronary ostium; Fig. 7)



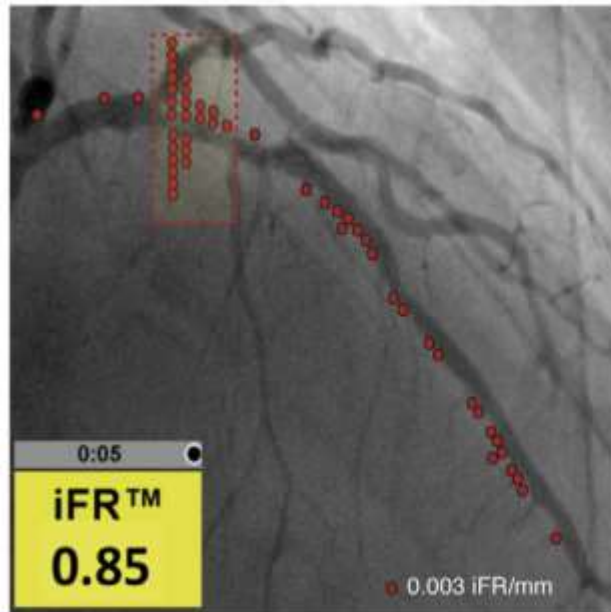
left = Pijls NH, *NEJM*. 1996 Jun 27;334(26):1703-8. (Figure 1)

right = Pijls NH, *Catheter Cardiovasc Interv*. 2000 Jan;49(1):1-16. (Text excerpts)

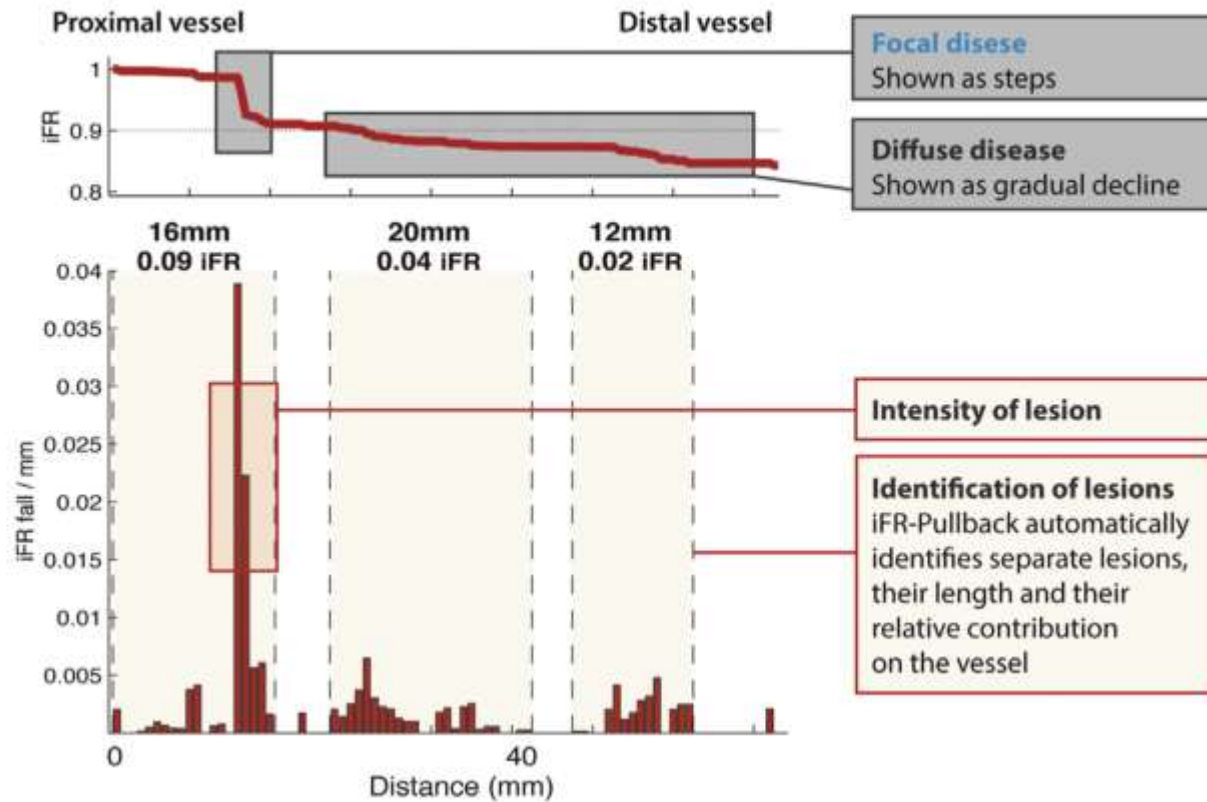
Overlay of pullback and angiogram

iFR Pullback recording: iFR throughout vessel

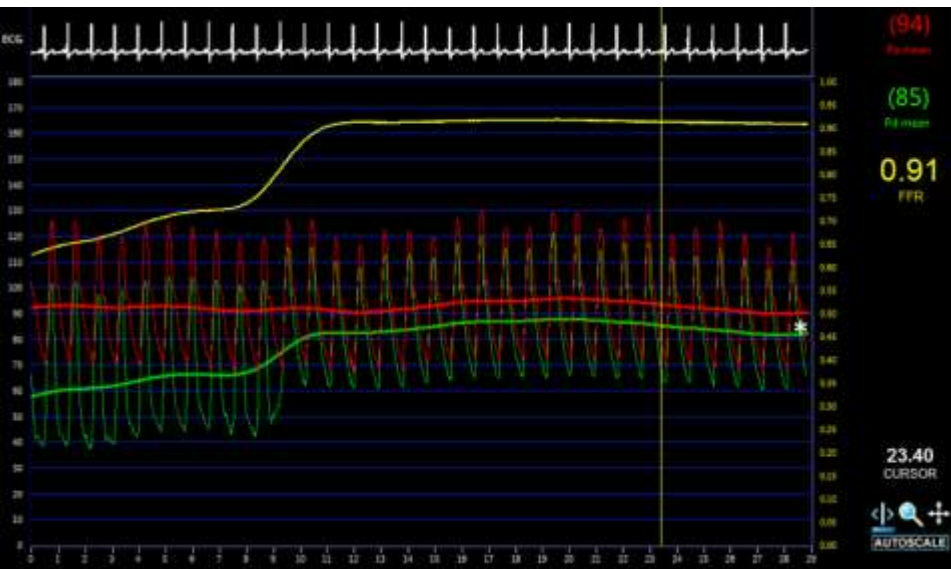
iFR intensity overlaid onto angiogram



Angiographic stenosis corresponds to region with highest change in iFR fall/mm (intensity)



Pullback information #1: *drift*

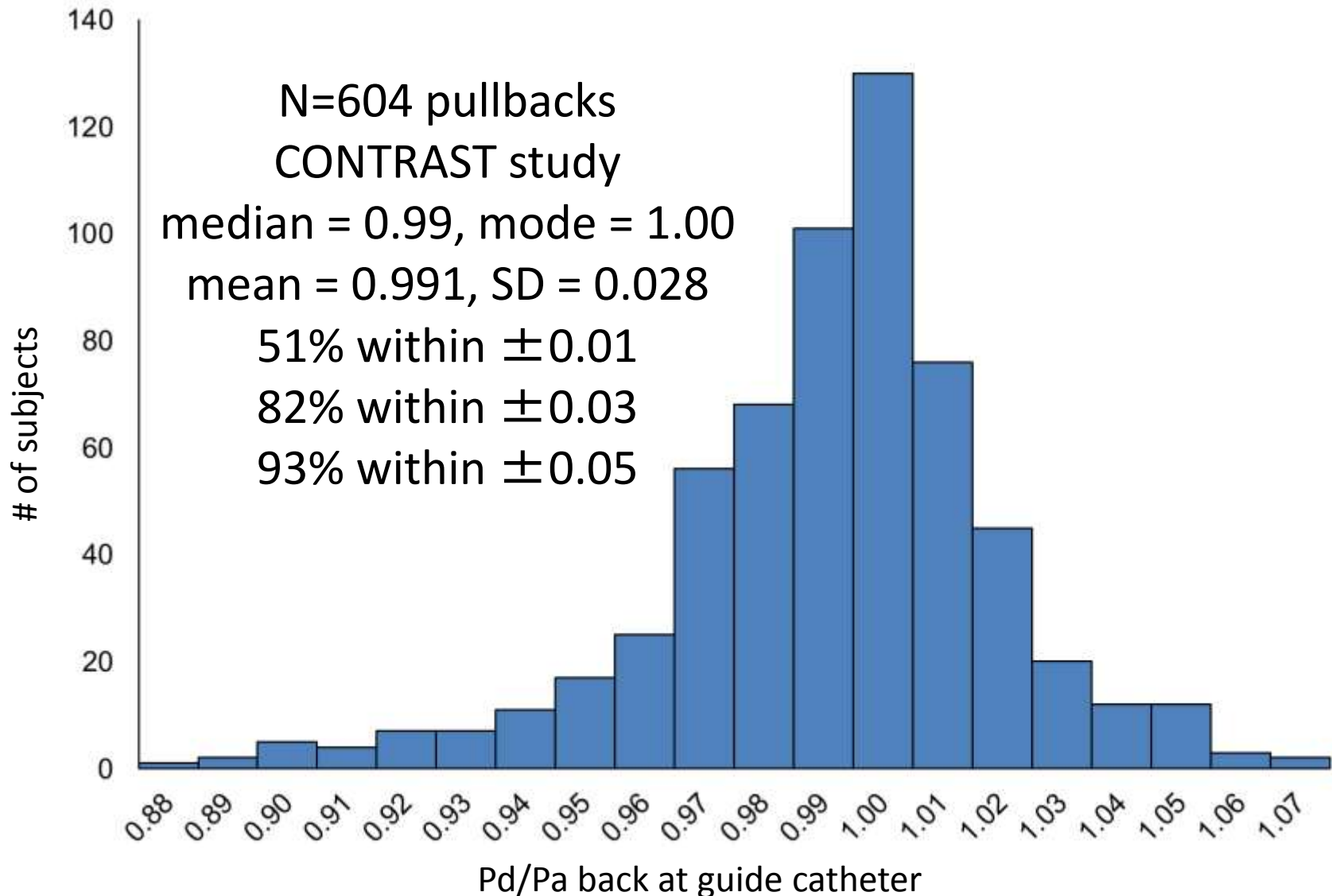


$Pd/Pa=0.91$
back at guide
→ *drift!*

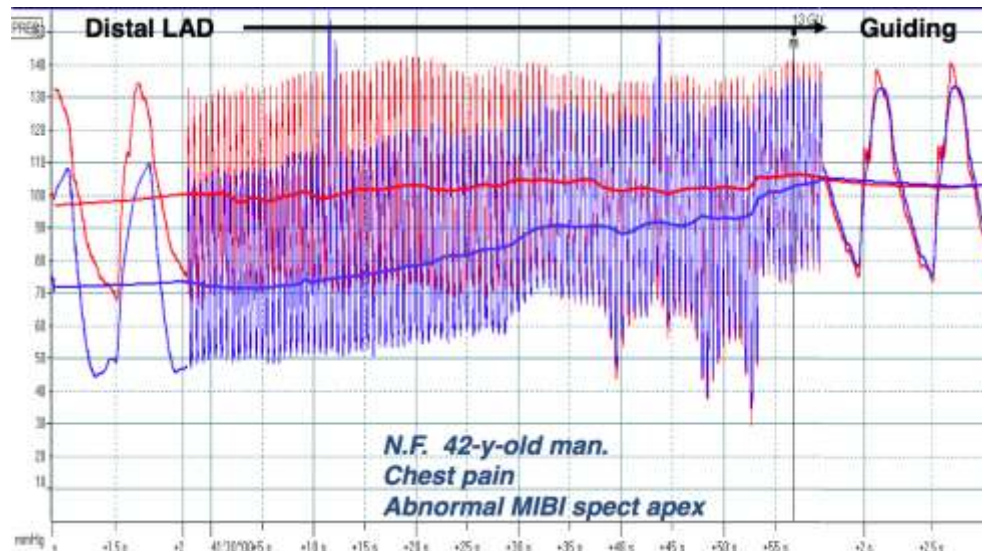
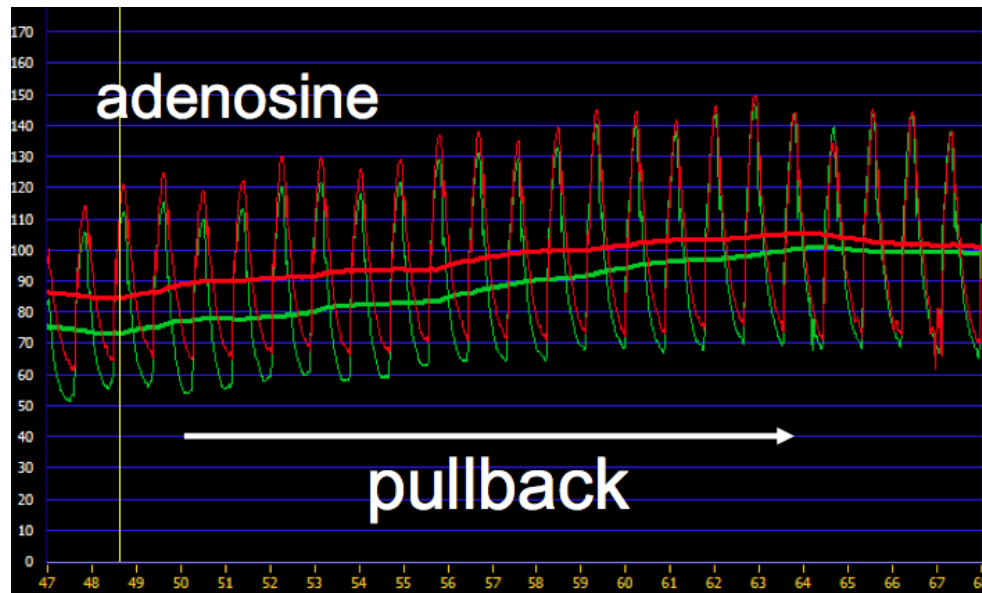


$Pd/Pa=1.00$
back at guide
→ *no drift!*

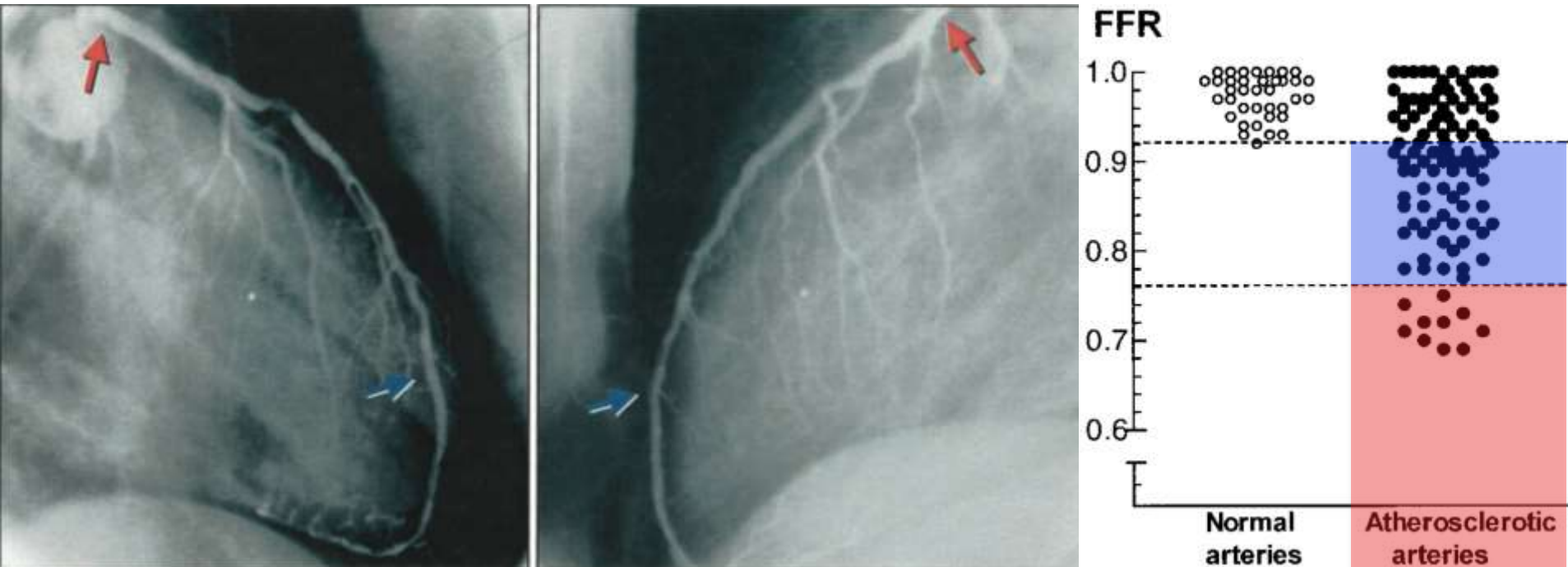
How much *drift* can we expect?



Pullback information #2: *diffuse*



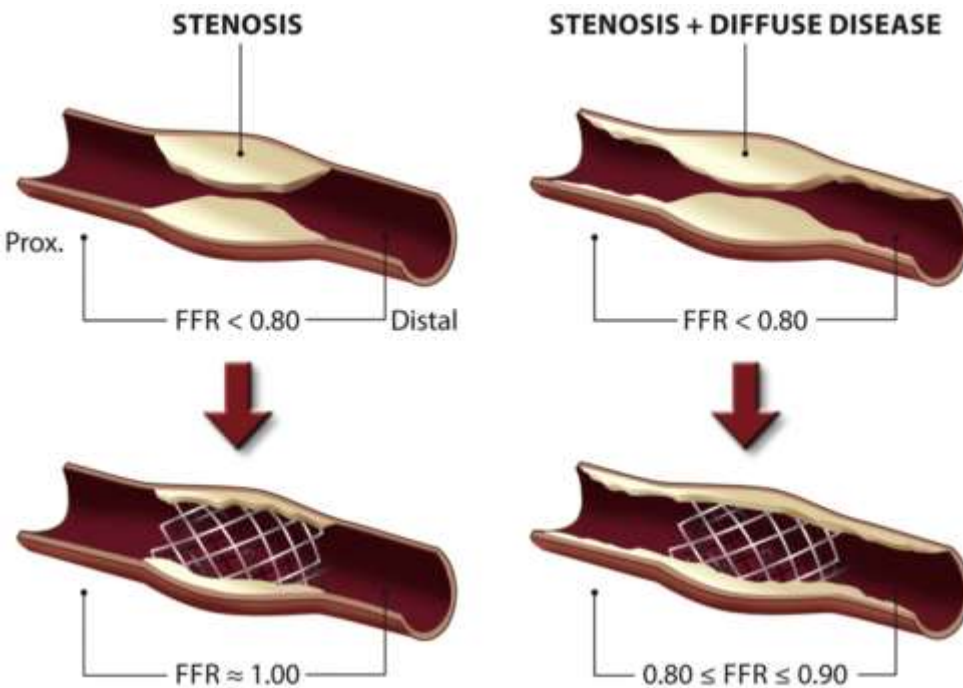
Diffuse disease common in CAD



44 year-old man, stable angina
FFR 0.76 distally, *Pa-Pd=23mmHg*
but diffuse on pullback

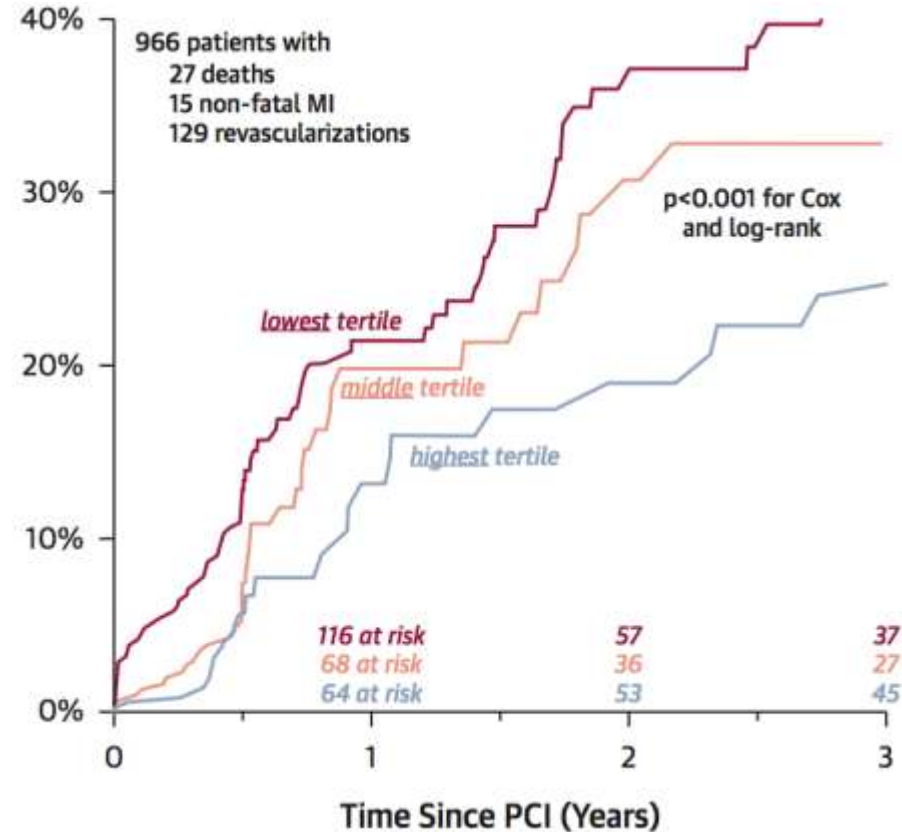
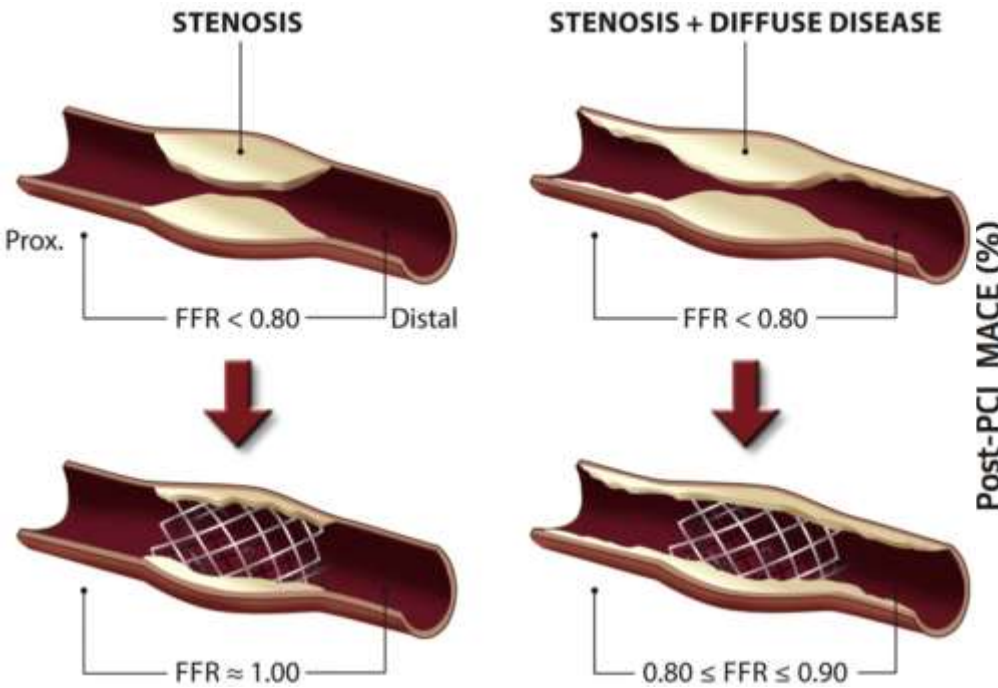
- N=106 with CAD
- All diffuse on pullback
- **8% with FFR<0.75**
- **57% with FFR<0.92**

Post-PCI FFR \approx diffuse disease burden



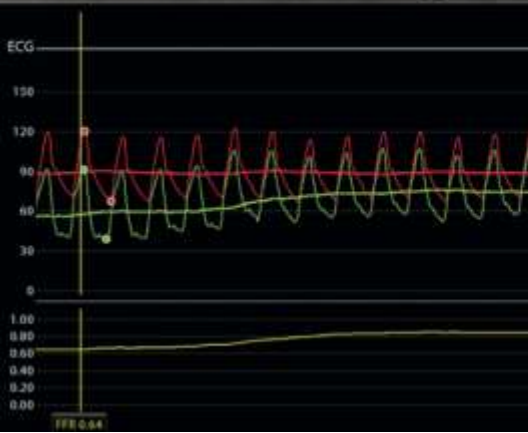
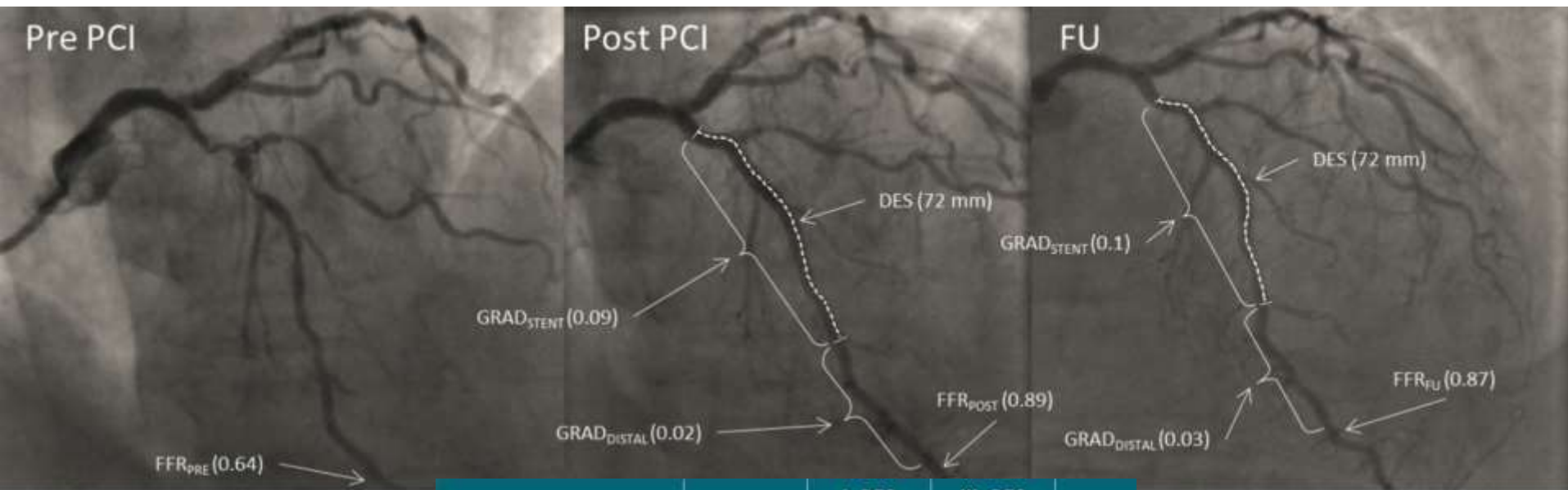
FFR measured **AFTER** stenting
Focal disease largely gone
Diffuse disease left behind

Diffuse disease impacts prognosis

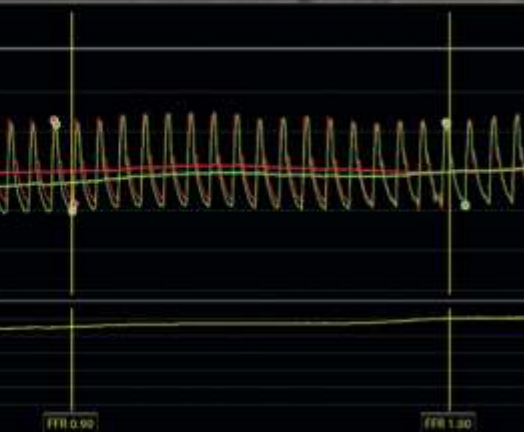


FFR measured **AFTER** stenting
 Focal disease largely gone
 Diffuse disease left behind

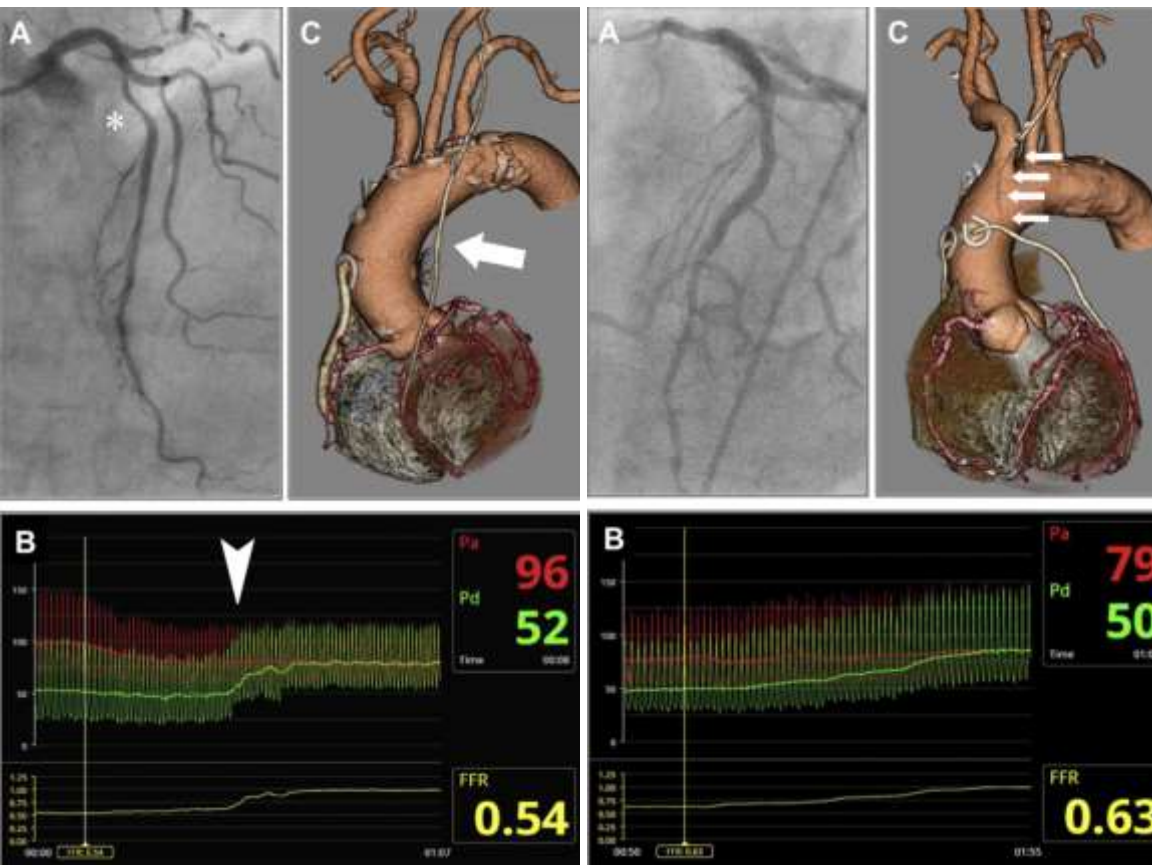
Treat diffuse disease with PCI?



	All patients n=74	L-DES (≤50 mm) n=37	UL-DES (>50 mm) n=37	p-value
Baseline (FFR _{PRE})	0.61±0.11	0.64±0.11	0.57±0.11	0.007
Post PCI	n=74	n=37	n=37	
FFR _{POST}	0.88±0.06	0.89±0.07	0.87±0.04	0.05
GRAD _{STENT}	0.06±0.03	0.04±0.03	0.07±0.03	0.001
GRAD _{DISTAL}	0.05±0.05	0.04±0.05	0.06±0.05	0.25
Follow-up	n=61	n=31	n=30	
FFR _{FU}	0.85±0.08	0.86±0.08	0.85±0.09	0.48
GRAD _{StentFU}	0.07±0.07	0.05±0.05	0.09±0.08	0.04
GRAD _{DistalFU}	0.05±0.04	0.05±0.05	0.04±0.03	0.38

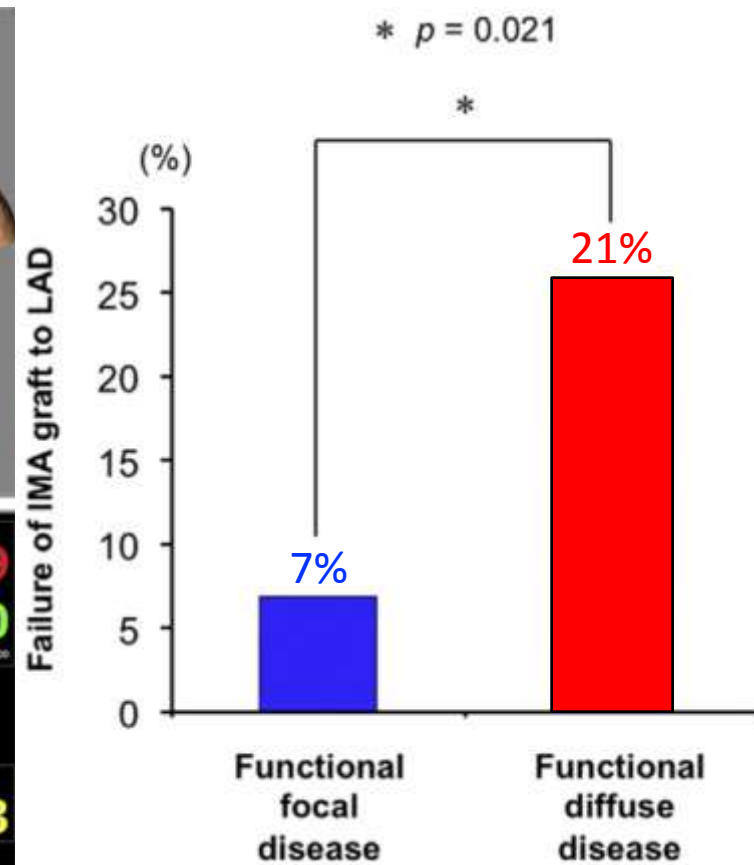


Treat diffuse disease with LIMA?



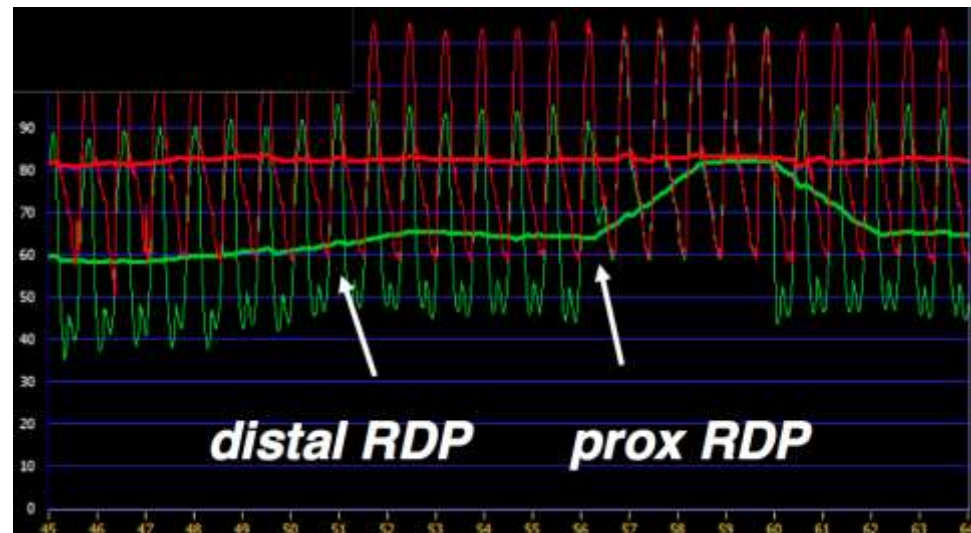
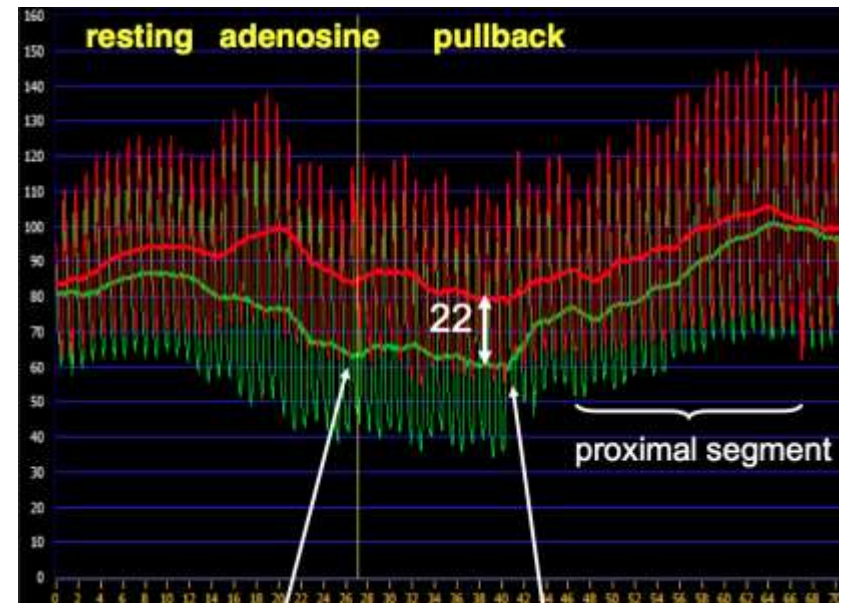
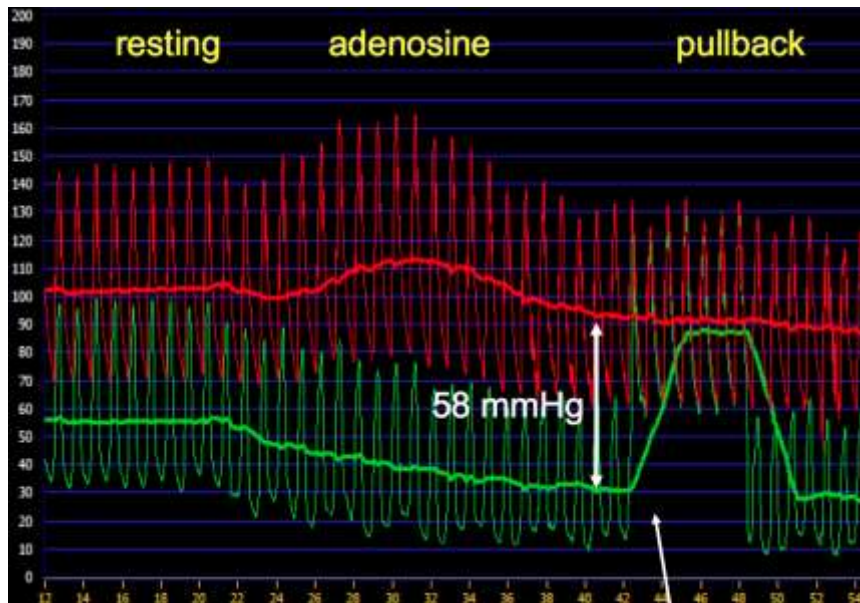
Focal disease
Patent LIMA
@ 3 months

Diffuse disease
Atretic LIMA
@ 4 months



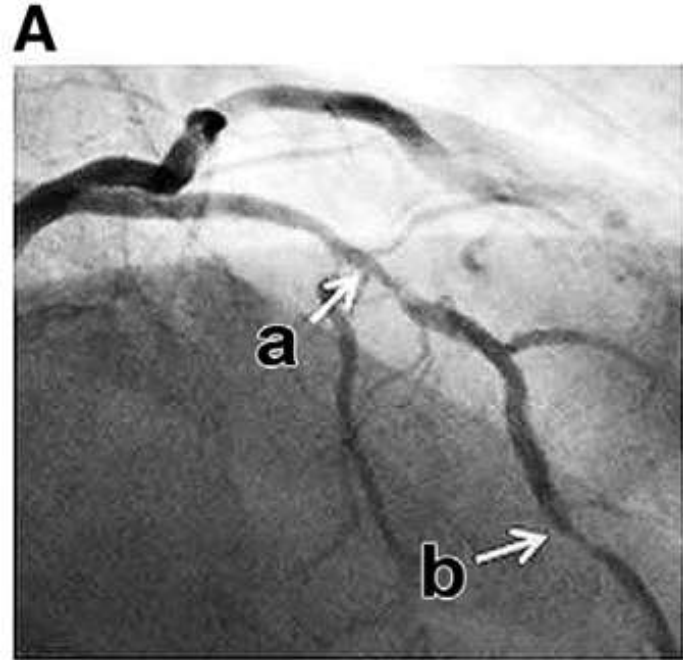
7% versus 21%

Pullback information #3: *focal*



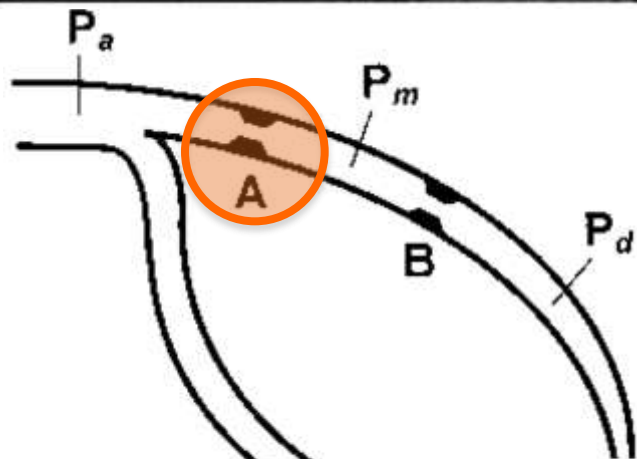
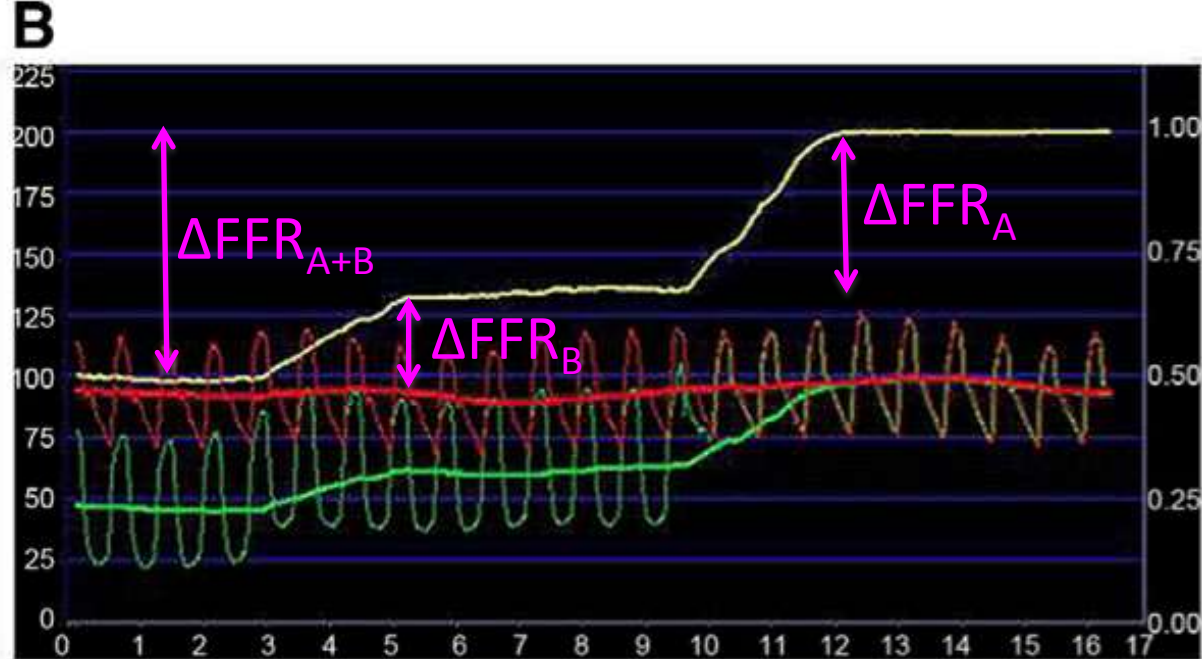
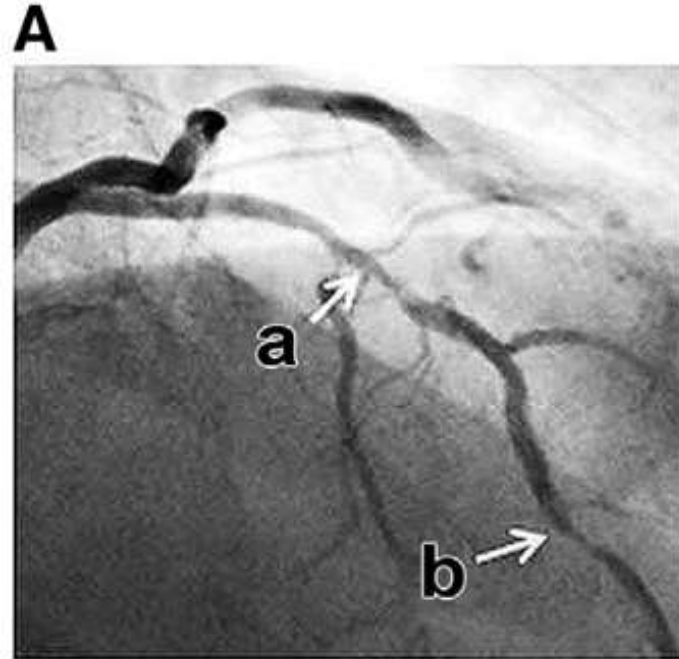
Various pressure tracings from ETP and TCT presentations (De Bruyne, Pijls, Tonino)

Serial/tandem stenoses



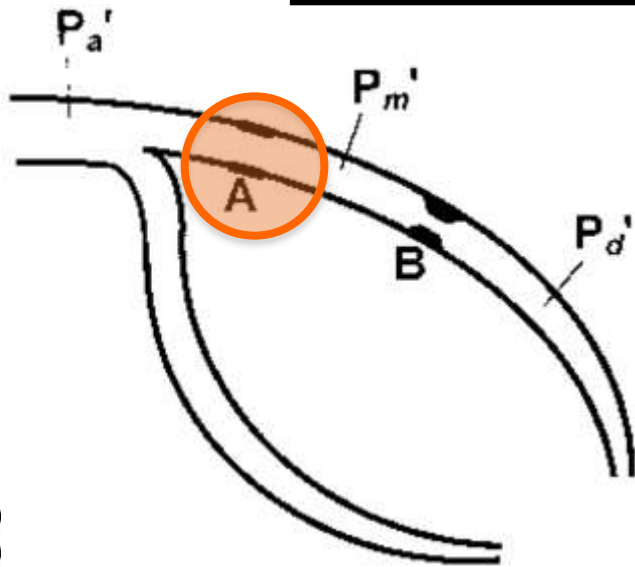
Treat A?
or Treat B?
Treat A *and* B?

Two lesions on pull-back curve

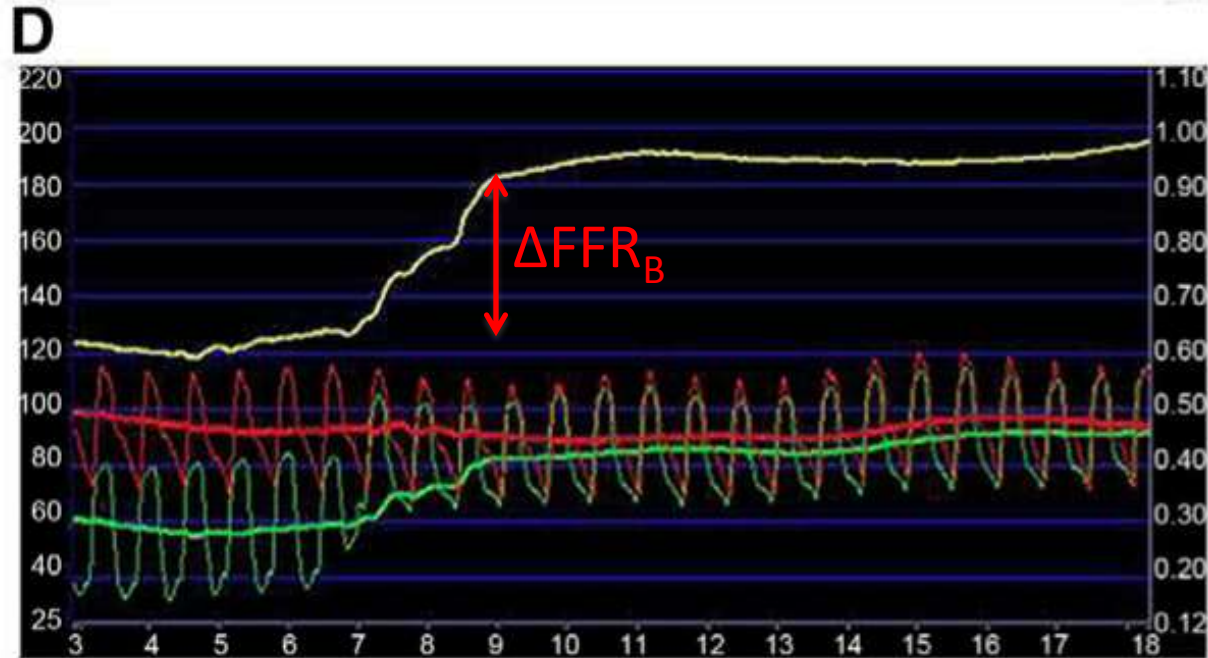


$$\Delta FFR_A > \Delta FFR_B$$

Treat worst and remeasure



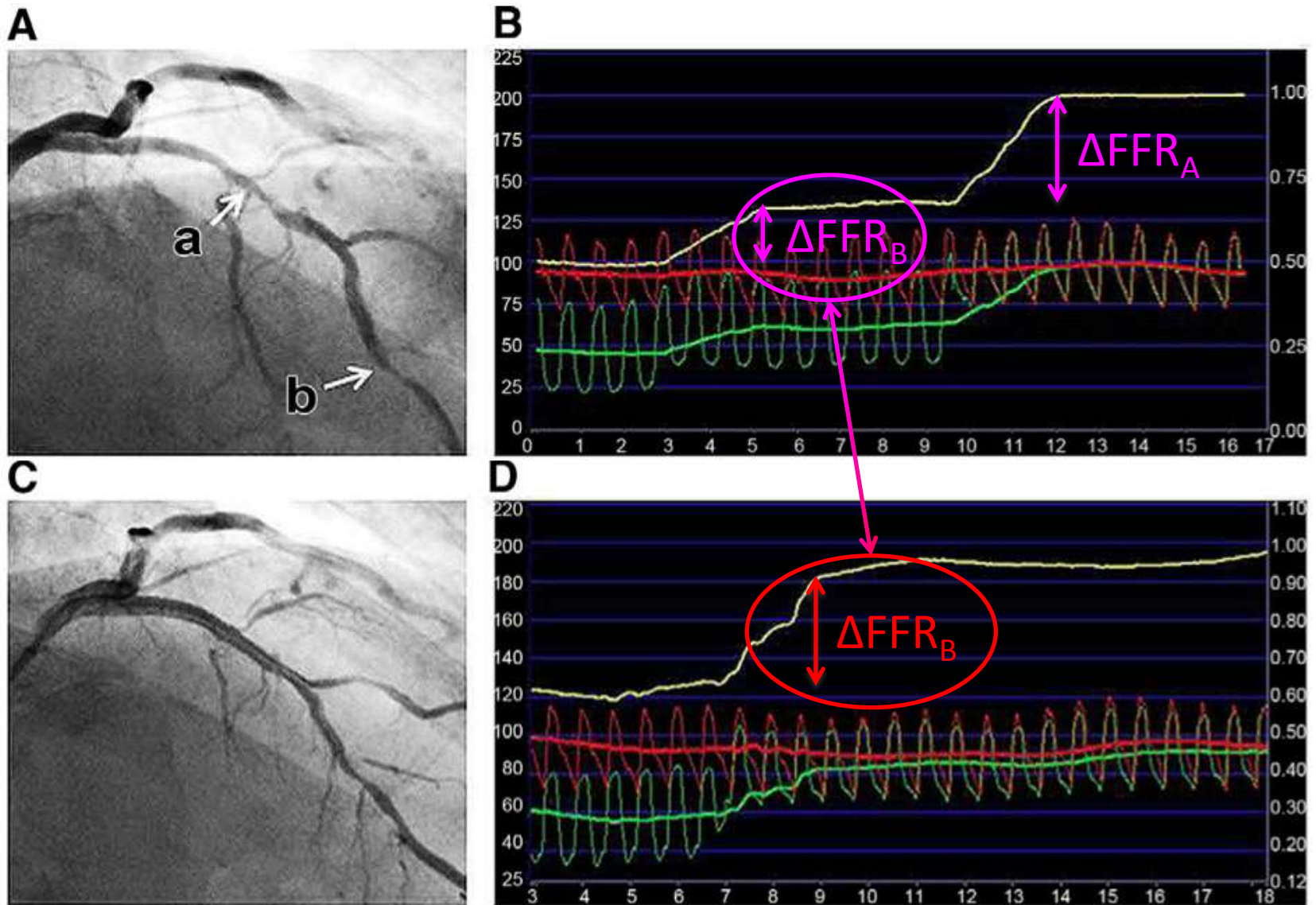
$\Delta FFR_A \approx 0$
(good PCI)



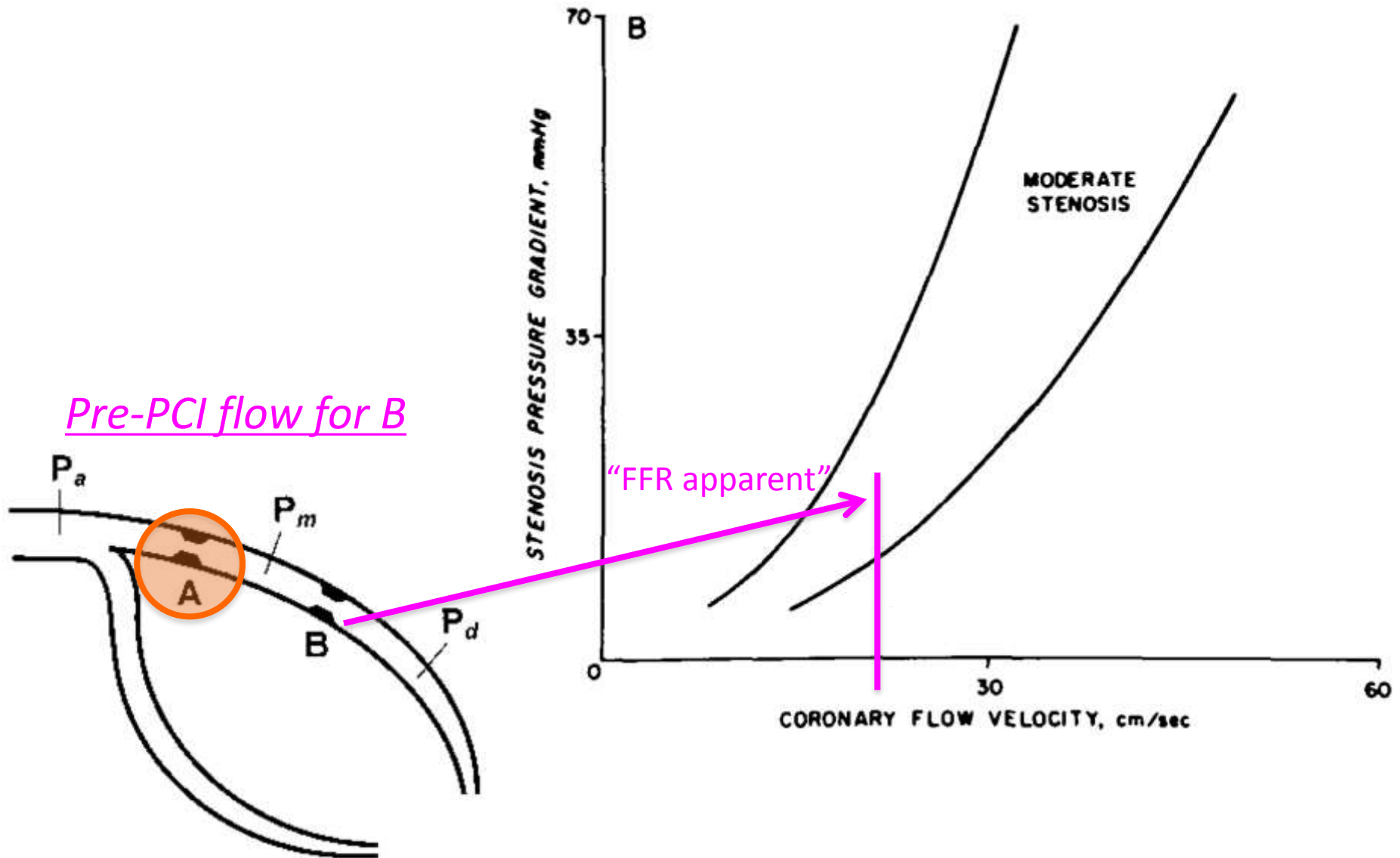
top = Pijls NH. *Circulation*. 2000 Nov 7;102(19):2371-7. (Portions of Figure 2)

bottom = Kim HL. *JACC Cardiovasc Interv*. 2012 Oct;5(10):1013-8. (Figure 1 C and D, annotated)

Why is there “cross talk”?



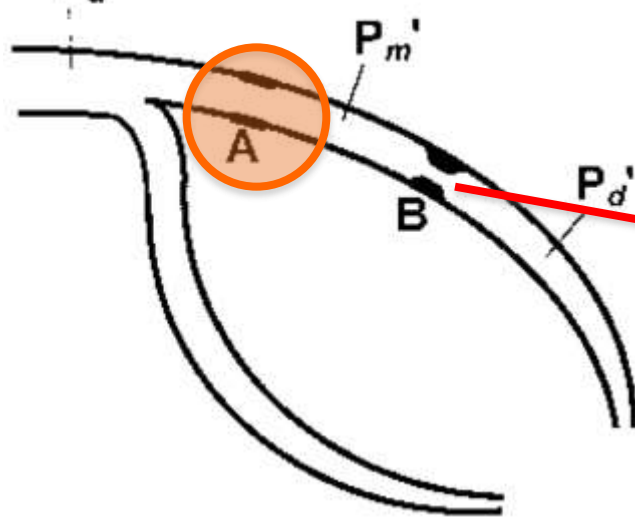
Why is there “cross talk”?



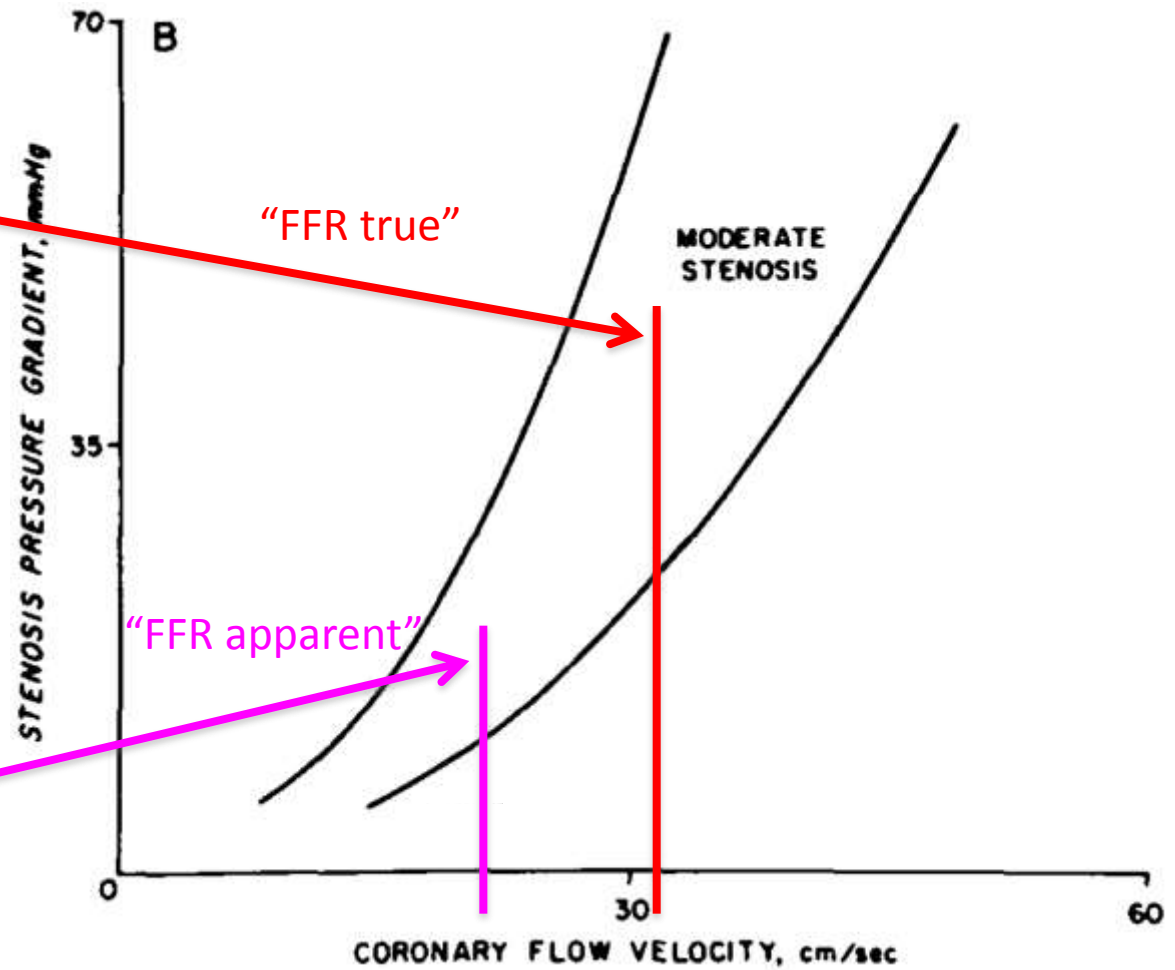
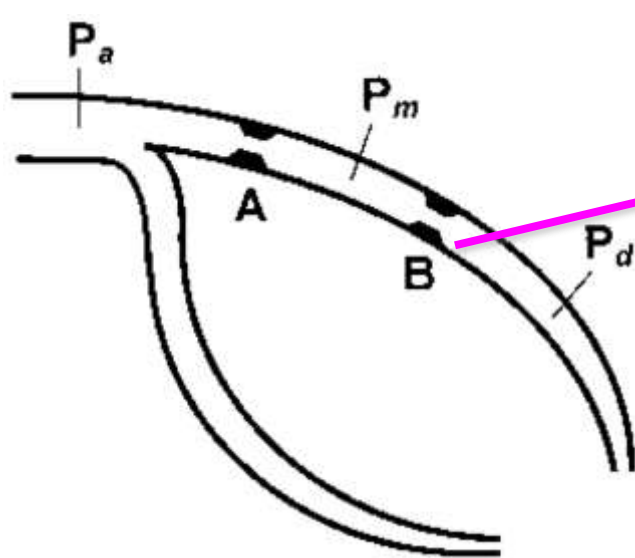
Pijls NH. *Circulation*. 2000 Nov 7;102(19):2371-7. (Portions of Figure 2)

Gould KL. *Circ Res*. 1978 Aug;43(2):242-53. (Figure 8, modified)

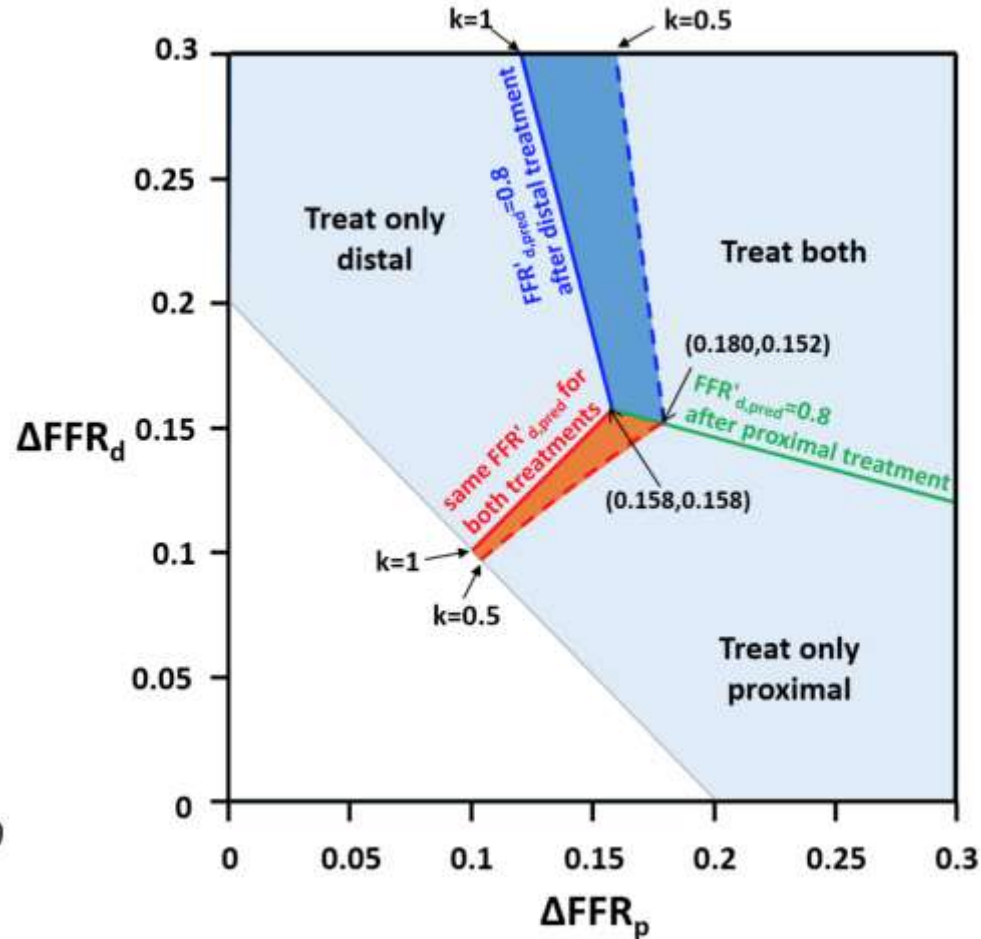
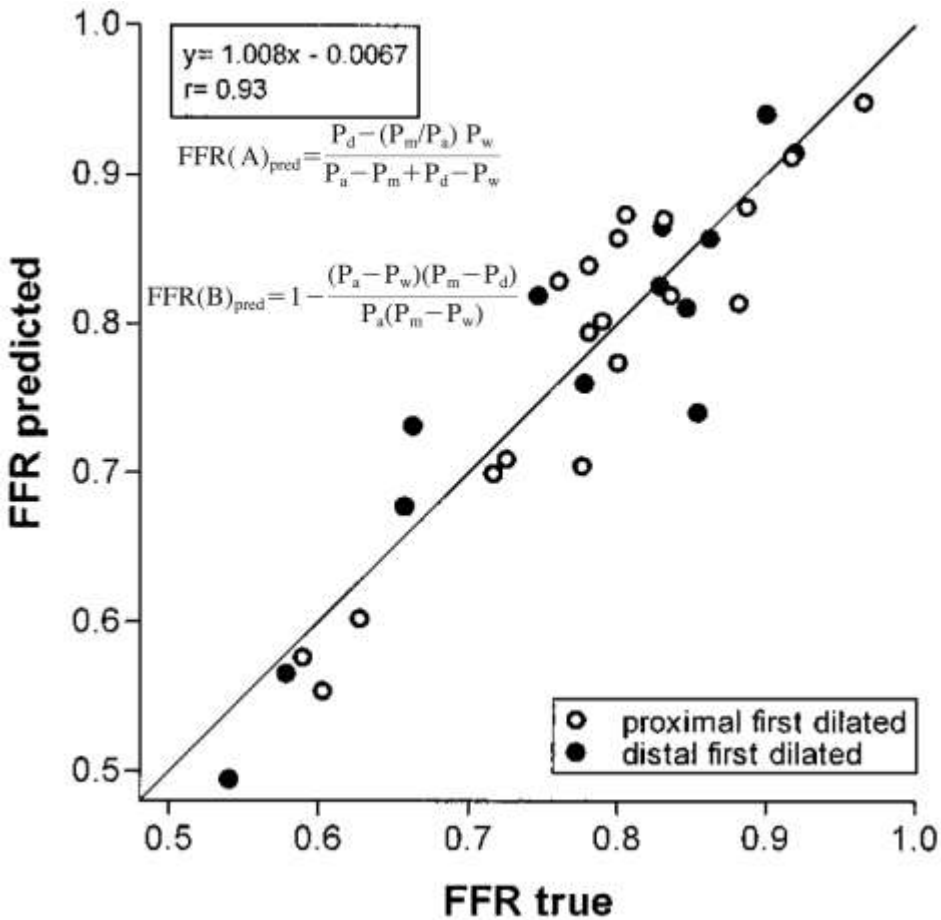
Post-PCI flow for B



Pre-PCI flow for B



Can we predict?



left, graph = Pijls NH. *Circulation*. 2000 Nov 7;102(19):2371-7. (Figure 5B)

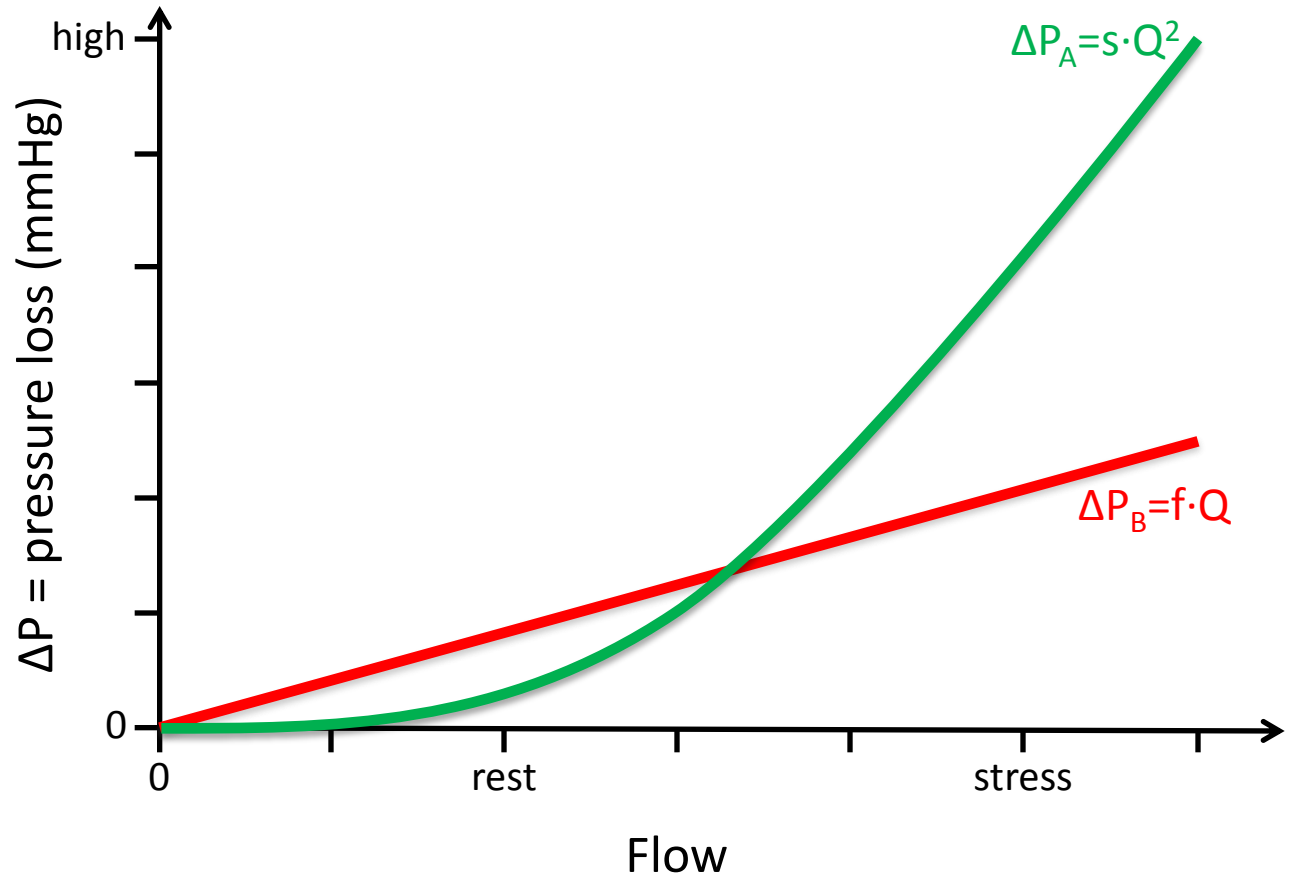
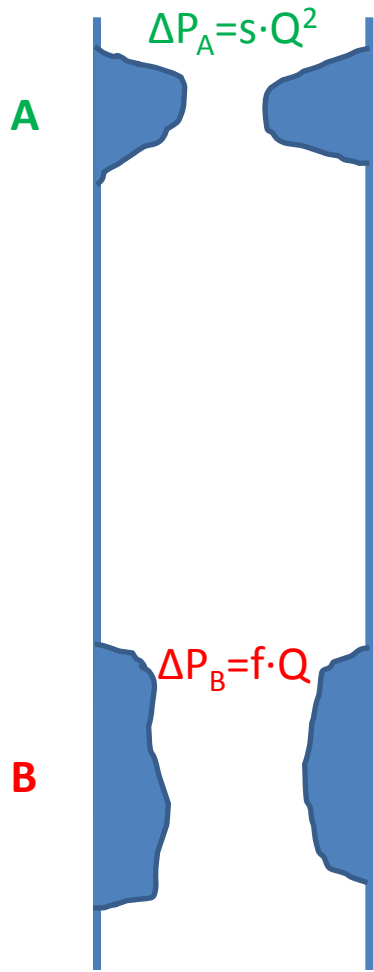
left, equations = De Bruyne B. *Circulation*. 2000 Apr 18;101(15):1840-7. (Equations 3 and 4)

right = Kweon J, *EuroIntervention*. 2016 Nov 22. [Epub ahead of print] (Figure 6)

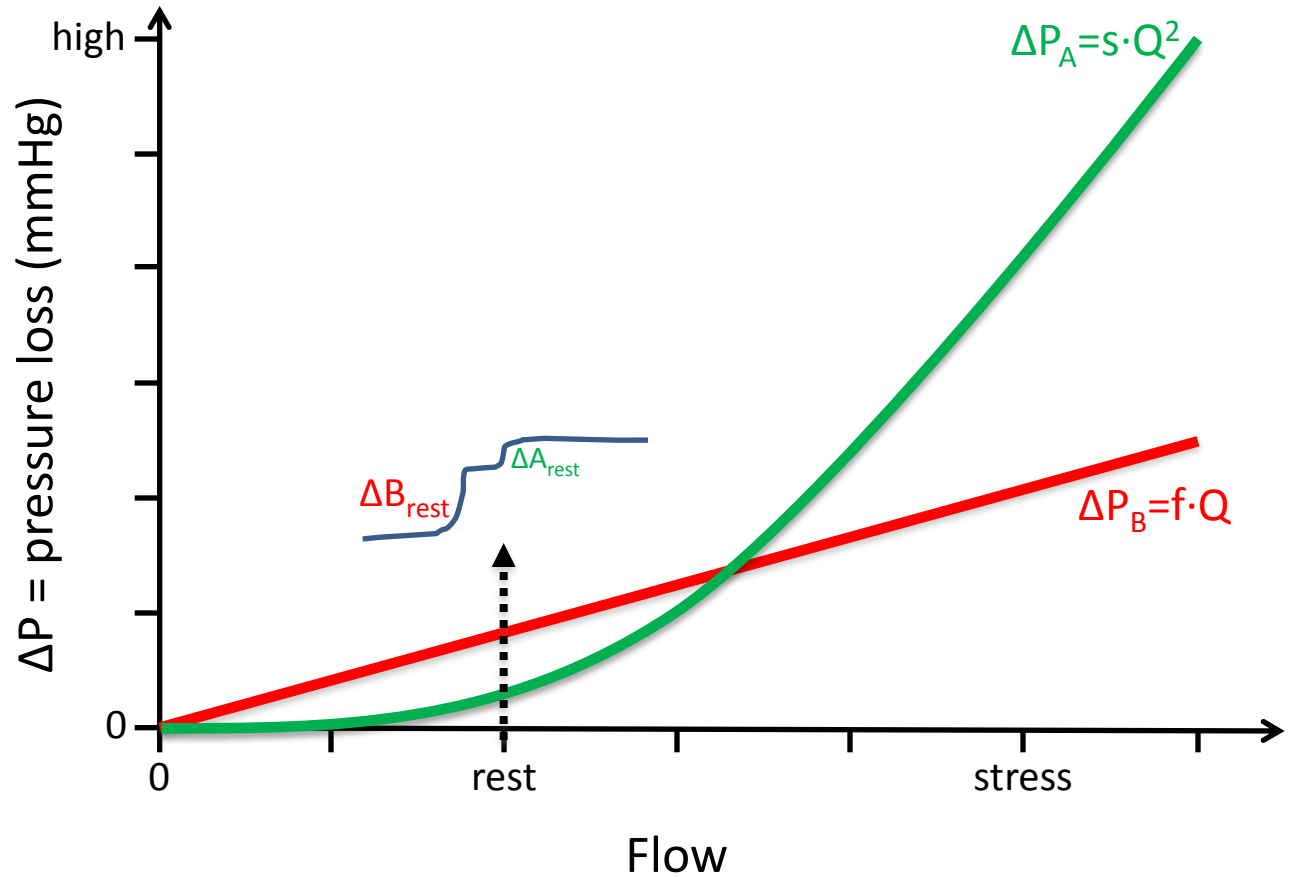
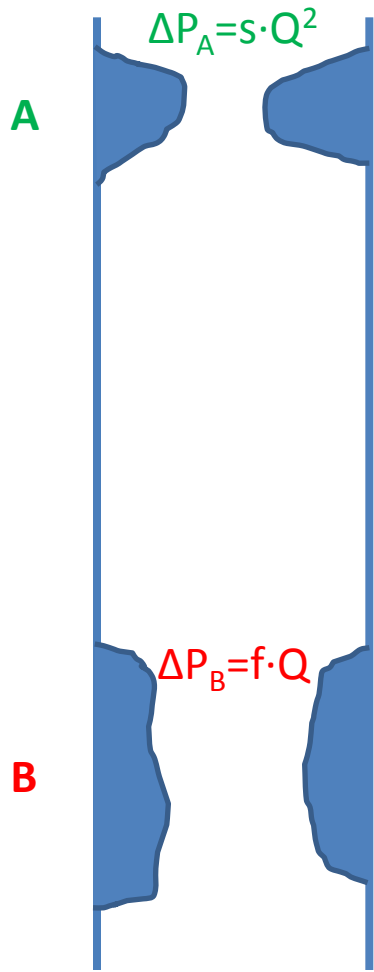
Limitations to prediction

- Produces false positives and negatives
- Requires measurement of wedge pressure
- Theory makes several assumptions
 - ✓ Neglects quadratic flow term
 - ✓ Does not allow branch between lesions
 - ✓ Assumes no coupling of flow profiles
 - ✓ Venous pressure ignored

Does resting physiology help?

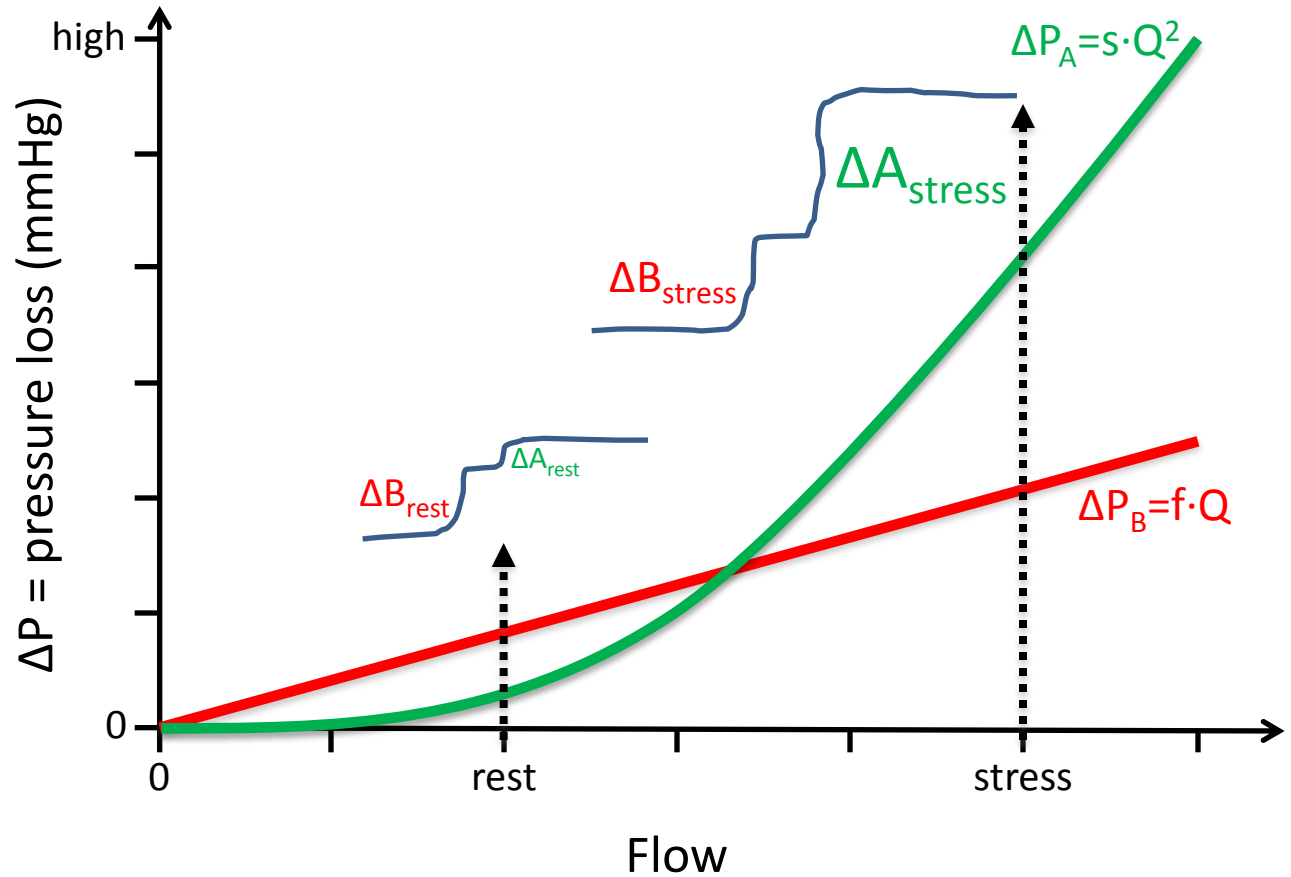
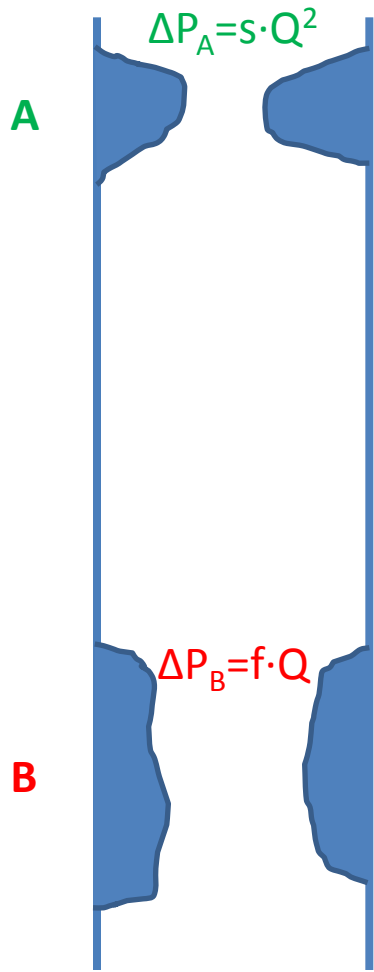


Does resting physiology help?



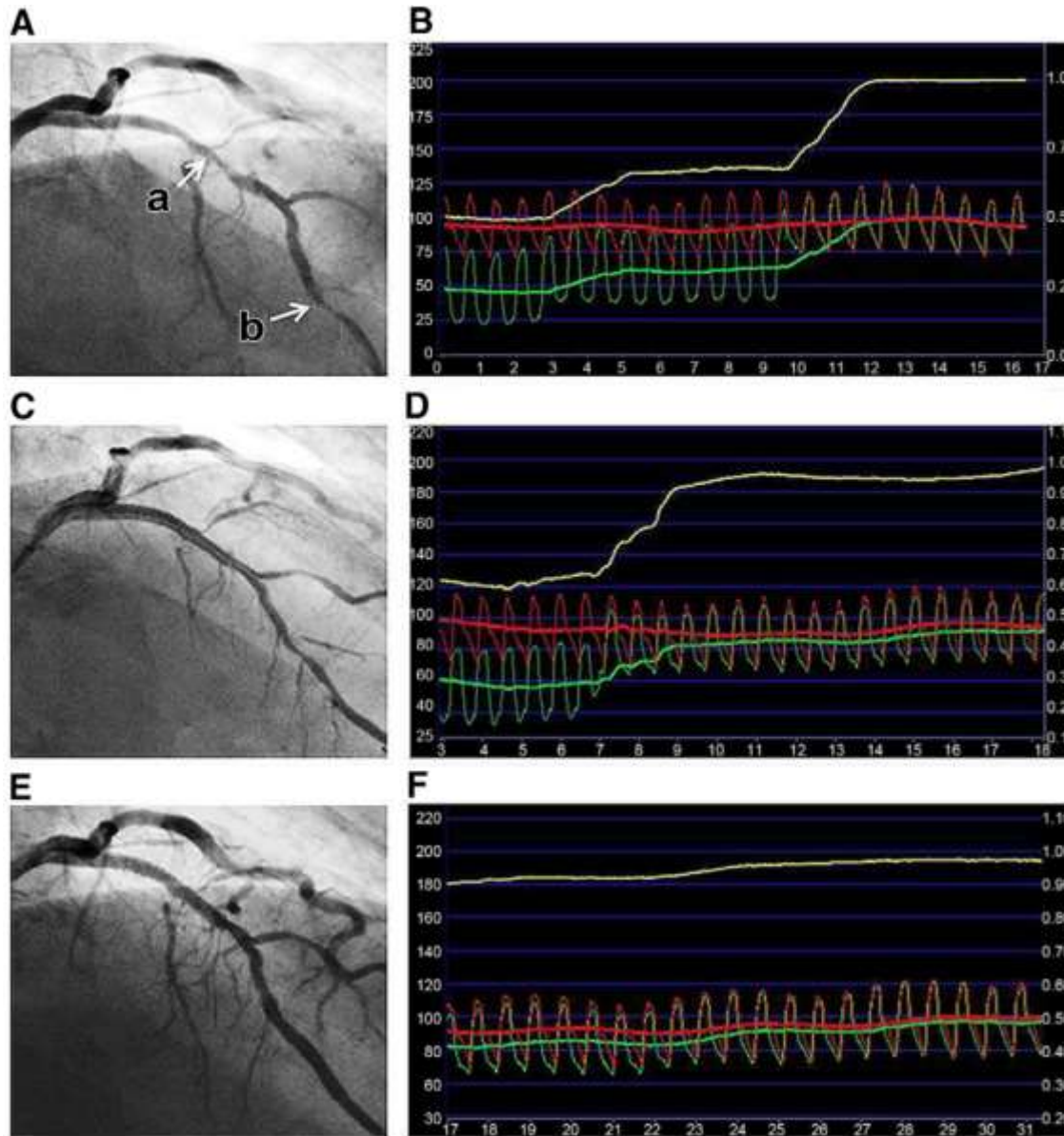
Rest conclusion: B worse than A

Does resting physiology help?



Stress conclusion: **A worse than B**
(also easier to measure!)

“Everyday” algorithm for serial lesions



pullback

treat worst
(if possible)
and remeasure
(\gg distal PCI 1st)

stop when
significant focal
($\Delta > 10$ mmHg)
is gone

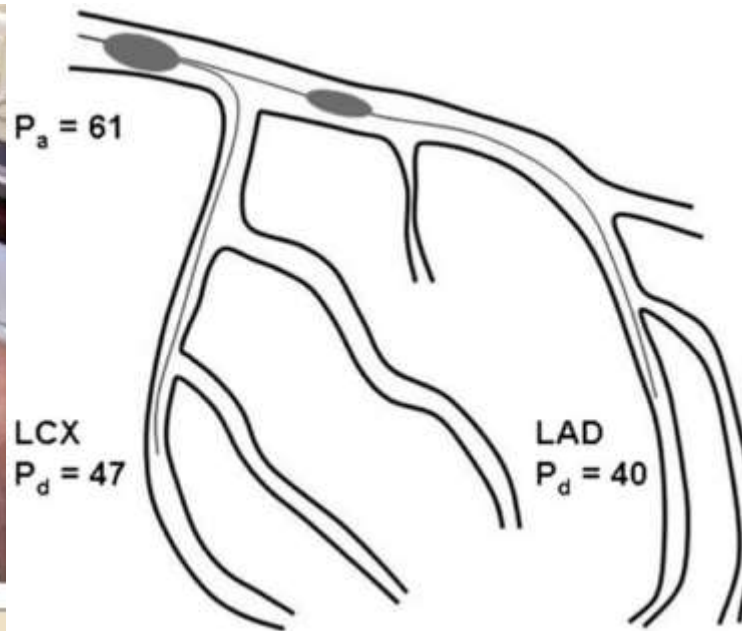
But what about the left main?

benchtop, 2012

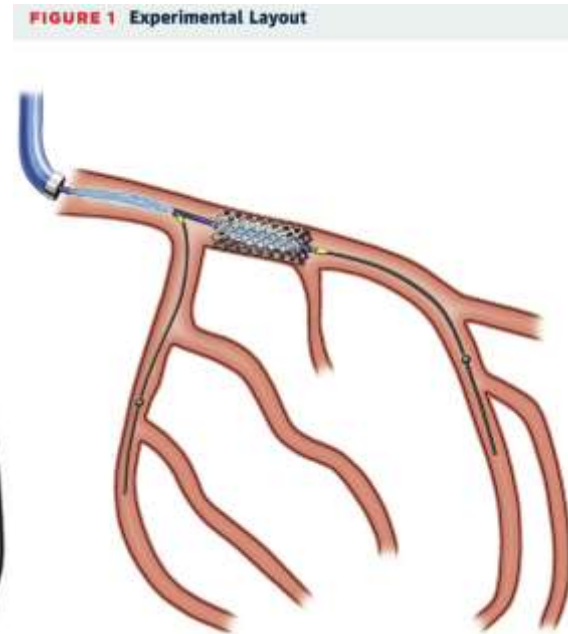


Figure 1. In Vitro Model of the Coronary Circulation

animals, 2013



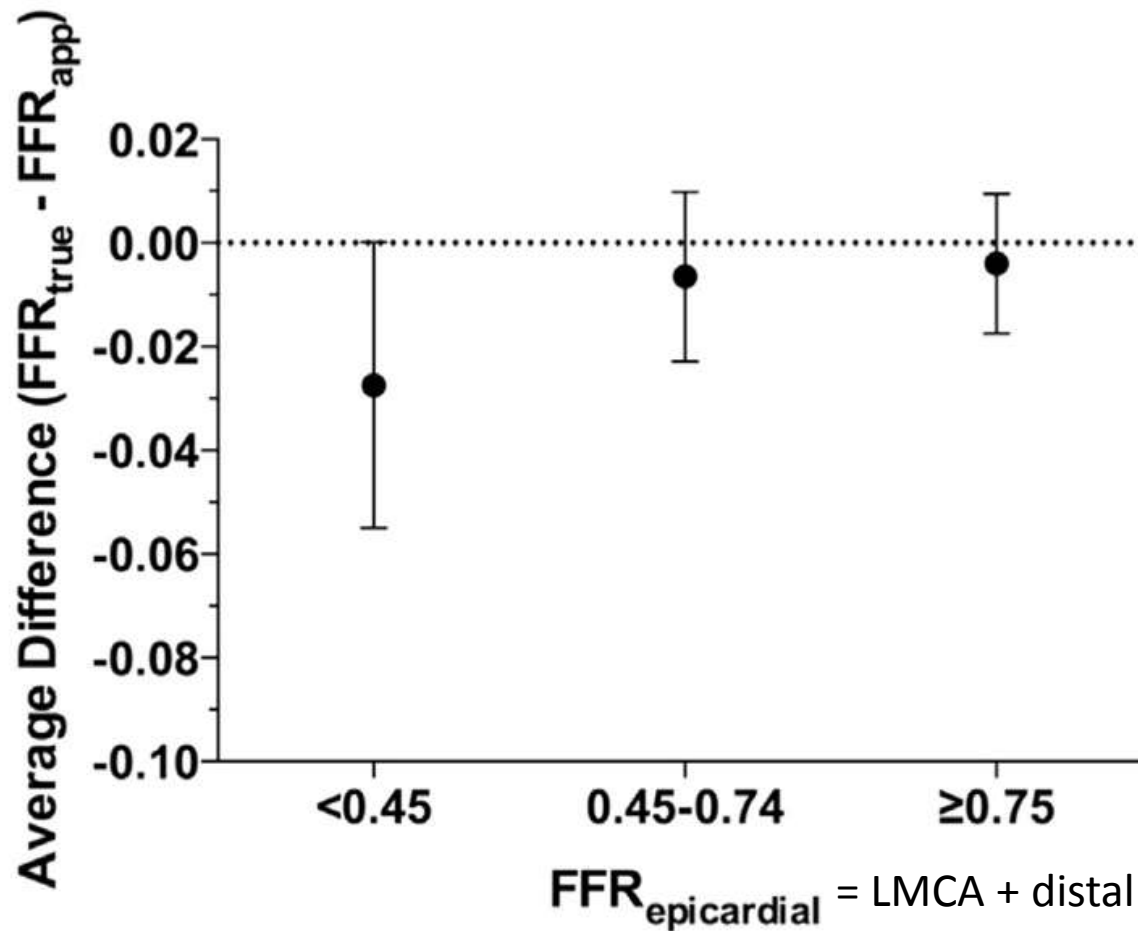
humans, 2015



left = Daniels DV. *JACC Cardiovasc Interv.* 2012 Oct;5(10):1021-5. (Figure 1)
middle = Yong AS. *Circ Cardiovasc Interv.* 2013 Apr;6(2):161-5. (Figure 1B)
right = Fearon WF. *JACC Cardiovasc Interv.* 2015 Mar;8(3):398-403. (Figure 1)

Only important with low distal FFR

FIGURE 7 Minimal Effect of Downstream Disease on LMCA FFR



Algorithm for LM serial/tandem lesion

- Measure FFR in LMCA + non-diseased vessel
 - ✓ $FFR \leq 0.80$ then LMCA significant
 - ✓ $FFR > 0.85$ then LMCA not significant
 - ✓ $FFR = 0.81$ to 0.85
 - Measure FFR = LMCA + diseased vessel
 - If $FFR_{\text{distal}} \leq 0.45$ then LMCA ?significant
 - If $FFR_{\text{distal}} > 0.45$ then LMCA likely OK

Practice and Potential Pitfalls of Coronary Pressure Measurement

**Nico H.J. Pijls,^{1*} MD, PhD, Morton J. Kern,^{2†} MD, Paul G. Yock,^{3†} MD
and Bernard De Bruyne⁴ MD, PhD**

In summary, a pullback curve at maximum hyperemia is the most accurate, most convincing, and most reliable way to study the functional status of every part of a coronary artery. **The pullback curve under hyperemia never lies.**