ABC OF CORONARY PHYSIOLOGY AND FFR FOR THE INTERVENTIONALIST

TCT ASIA
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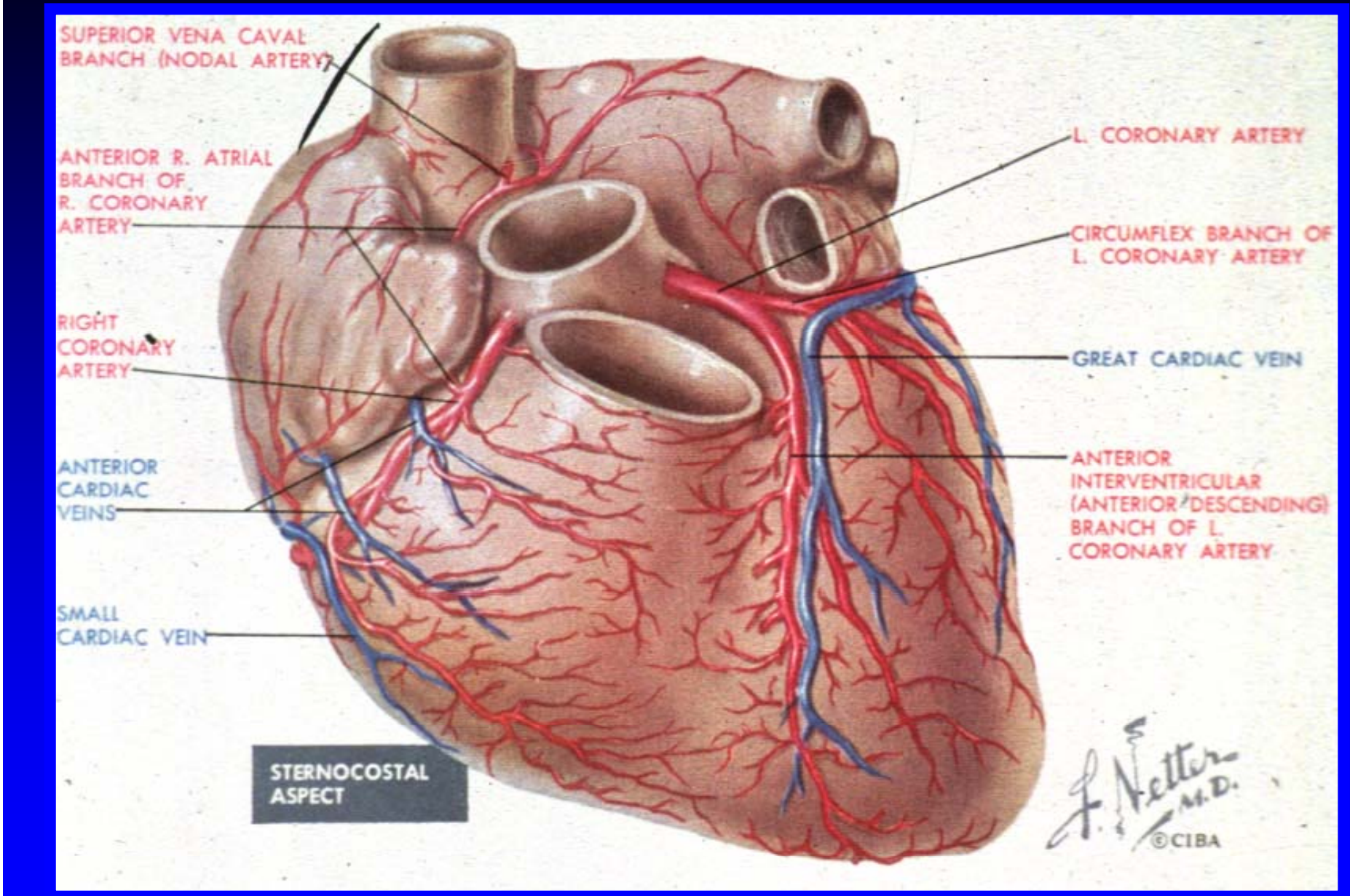
ABC OF CORONARY PHYSIOLOGY FOR THE INTERVENTIONALIST

1. Regulation of coronary blood flow
2. Autoregulation
3. Fluid Dynamics Equation and Necessity of Maximum Hyperemia
4. Fractional Flow Reserve


SUPERIOR VENA CAVAL BRANCH (NODAL ARTERY)
ANTERIOR R. ATRIAL BRANCH OF R. CORONARY ARTERY
RIGHT CORONARY ARTERY
ANTERIOR CARDIAC VEINS
SMALL CARDIAC VEIN
L. CORONARY ARTERY
CIRCUMFLEX BRANCH OF L. CORONARY ARTERY
GREAT CARDIAC VEIN
ANTERIOR INTERVENTRICULAR (ANTERIOR DESCENDING) BRANCH OF L. CORONARY ARTERY
STERNOCOSTAL ASPECT
F. Netter M.D.
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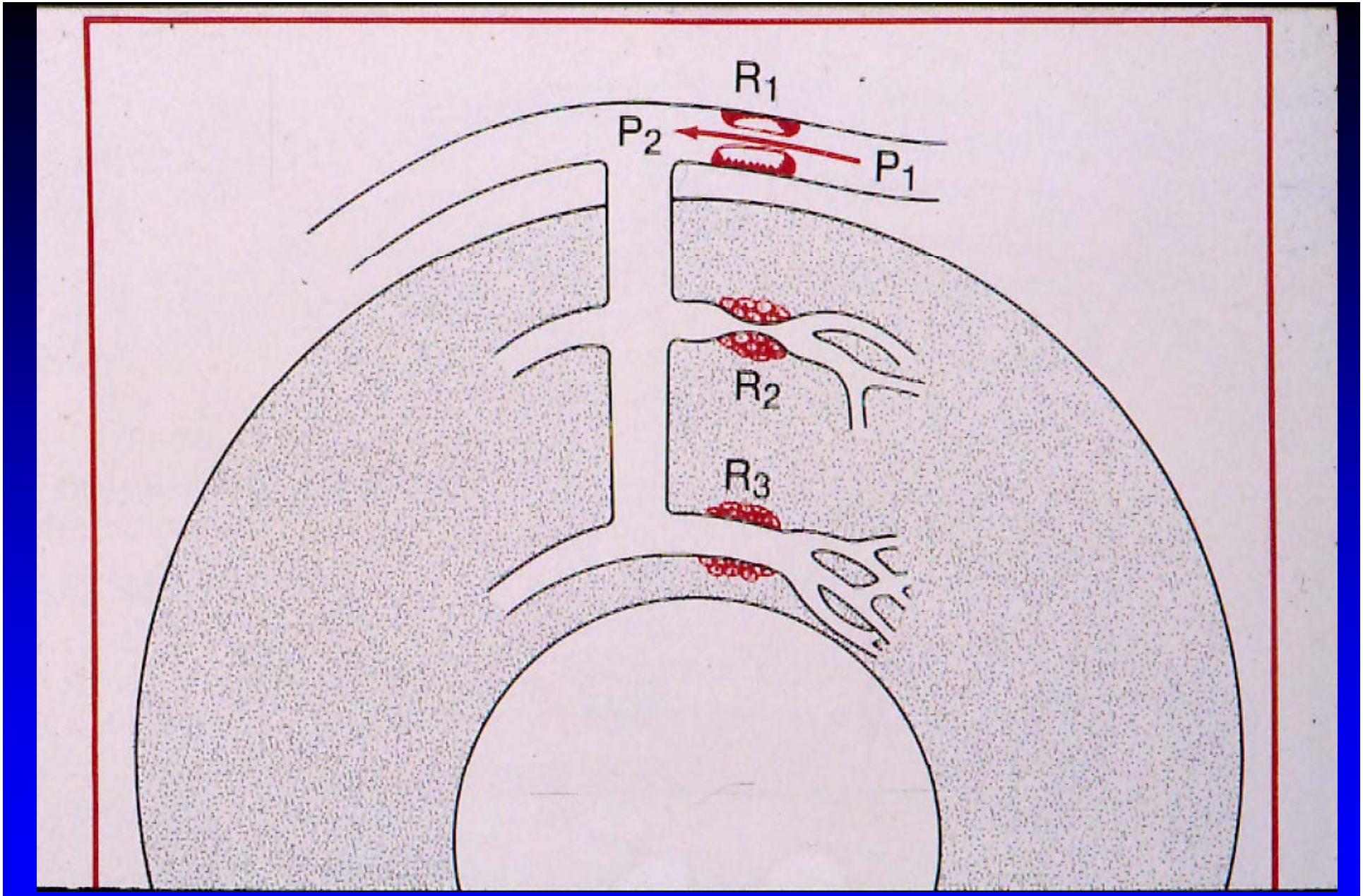
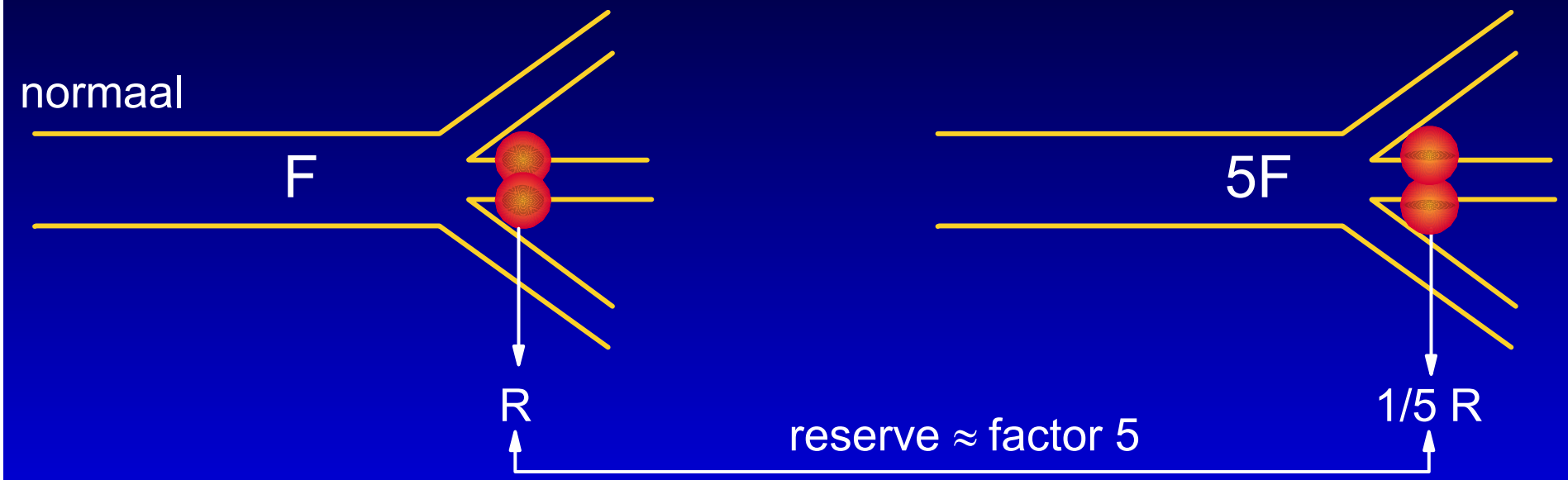


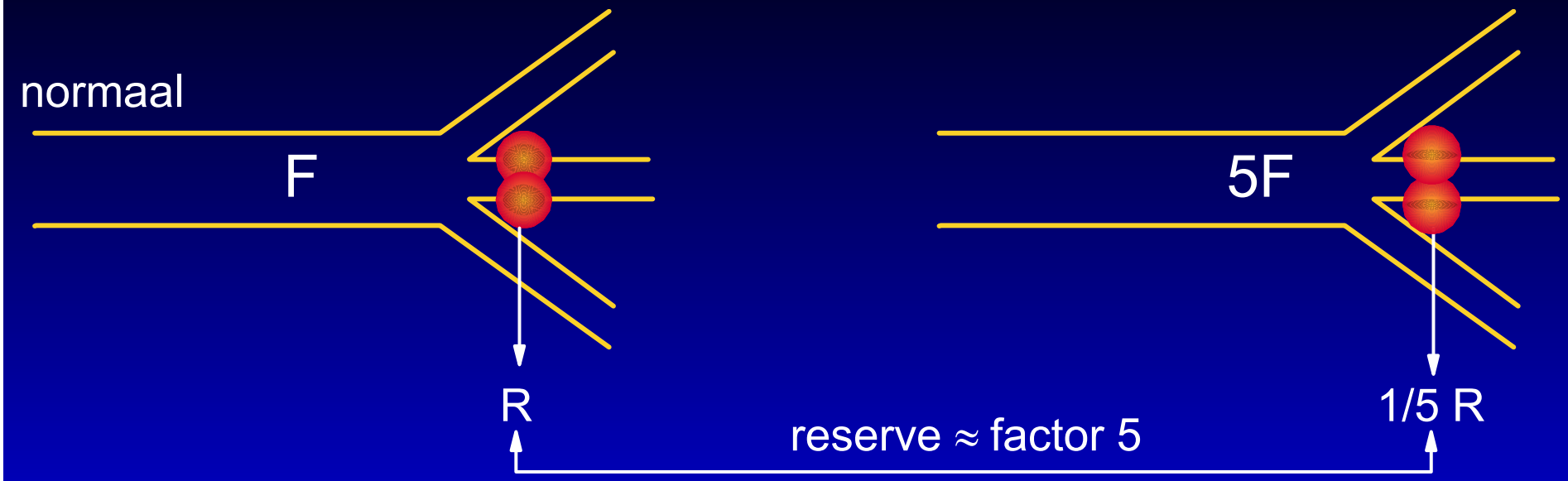
03 cc/schema Braunwald

Regulation of coronary blood flow by arteriolar vasodilation



RUST **MAX. VASODILATATIE**

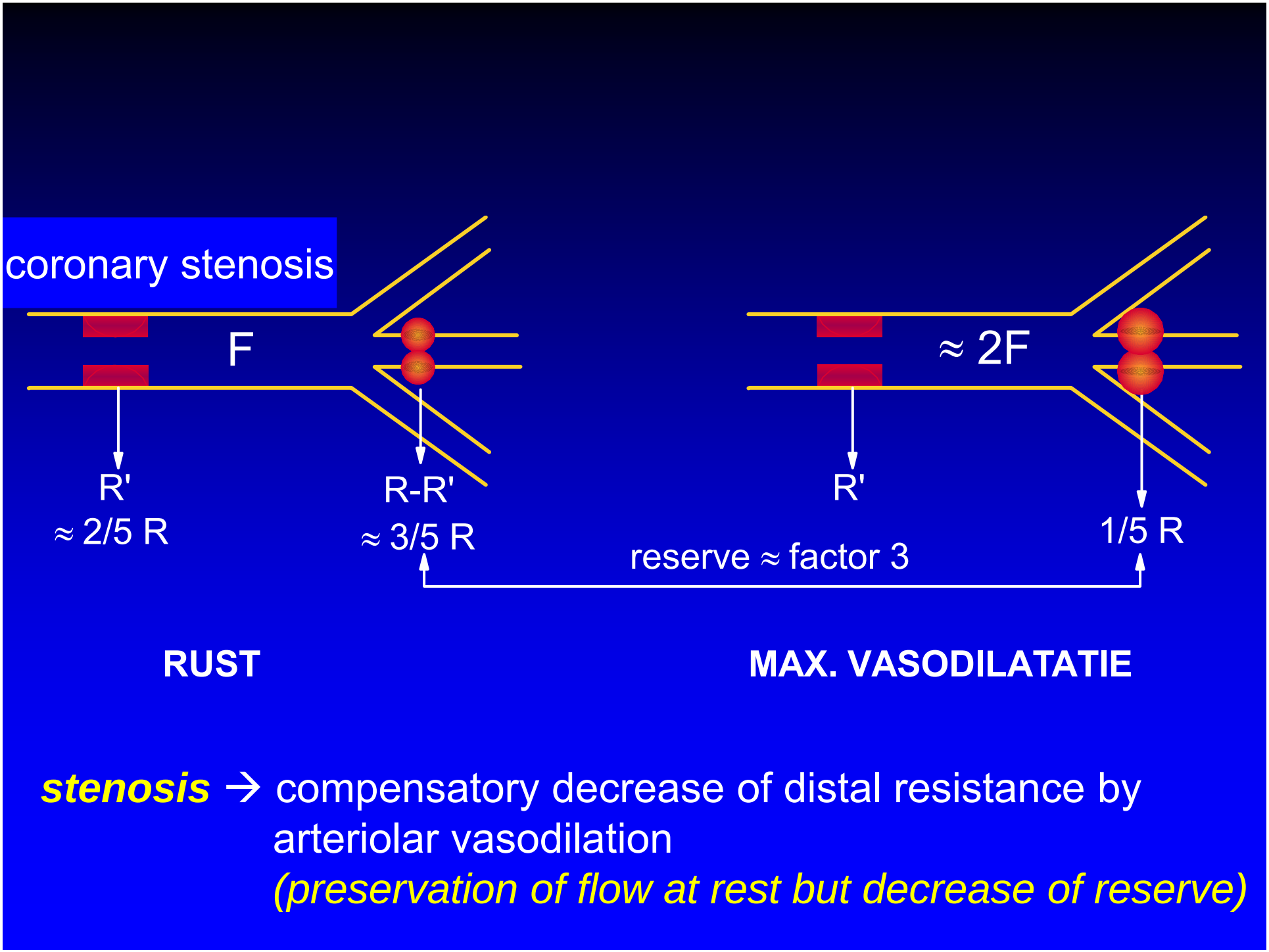
exercise → increased oxygen demand → vasodilation



RUST **MAX. VASODILATATIE**

decrease of blood pressure → arteriolar vasodilation
increase of blood pressure → arteriolar constriction

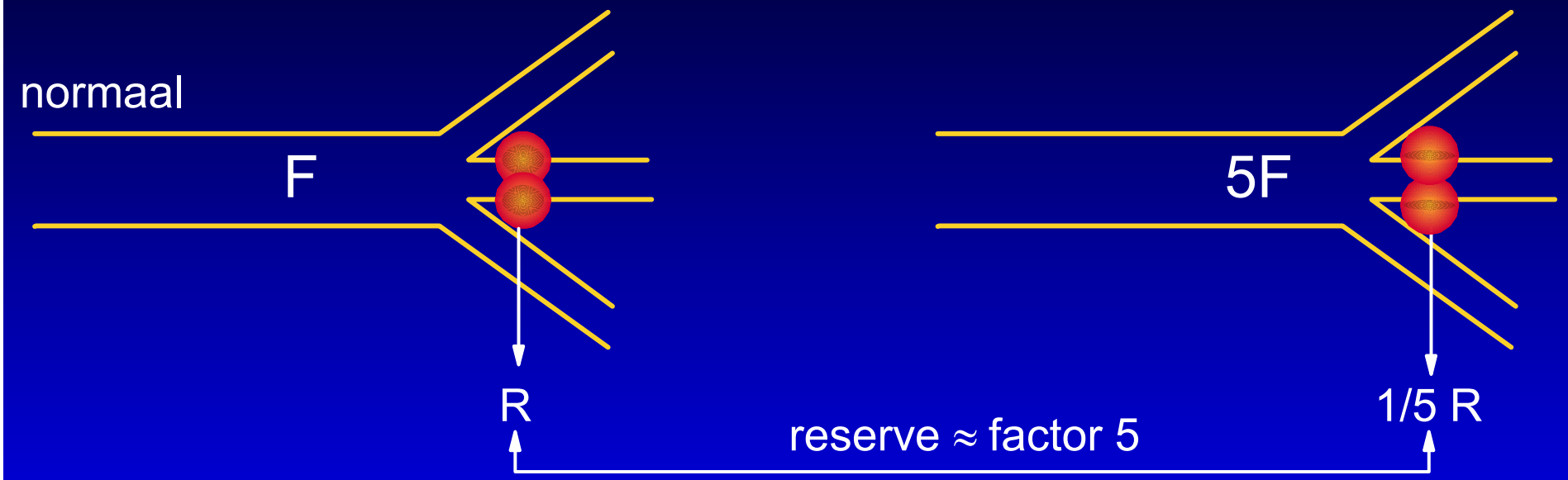
→ **coronary flow remains (almost) equal over wide range of perfusion pressure and matched to O2 demand (autoregulation)**



stenosis → compensatory decrease of distal resistance by arteriolar vasodilation

(preservation of flow at rest but decrease of reserve)

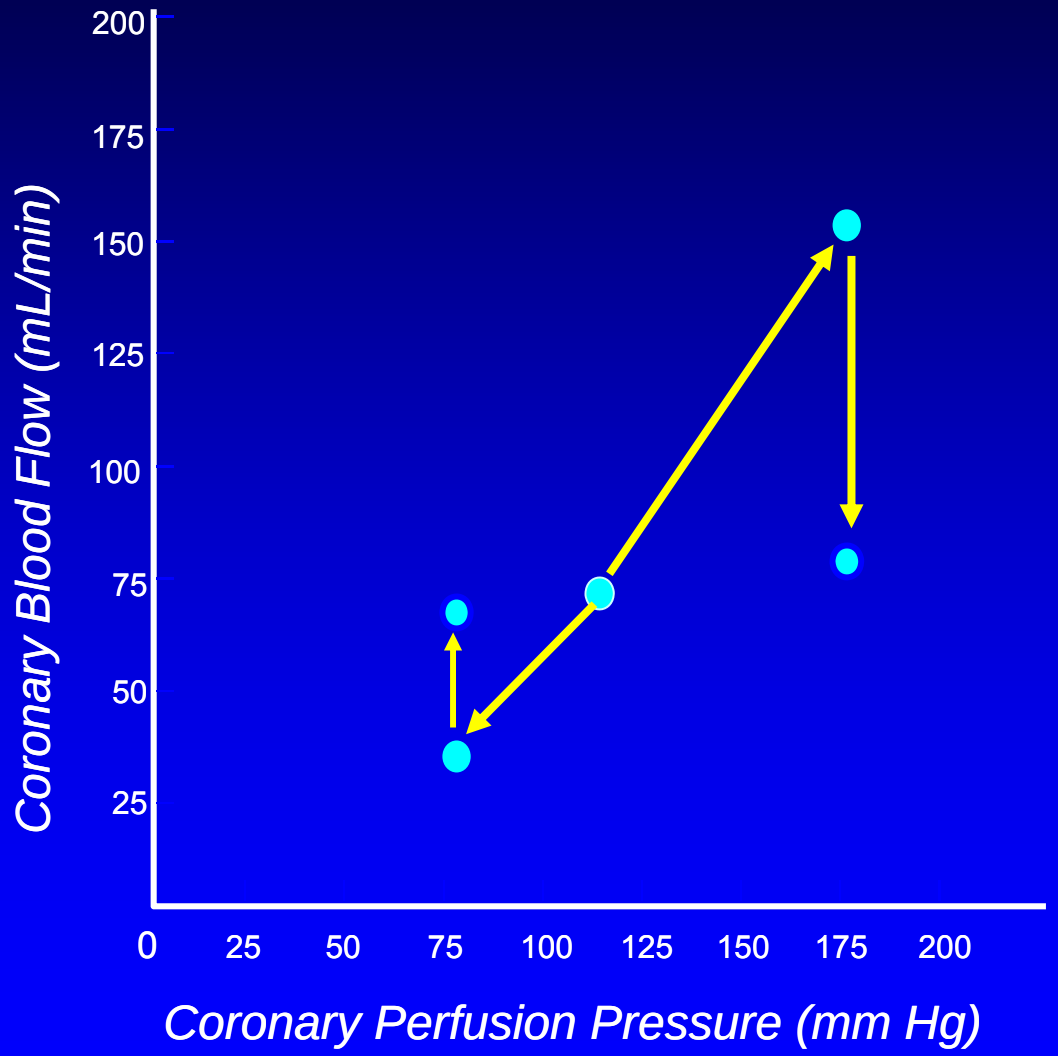
Regulation of coronary blood flow by arteriolar vasodilation



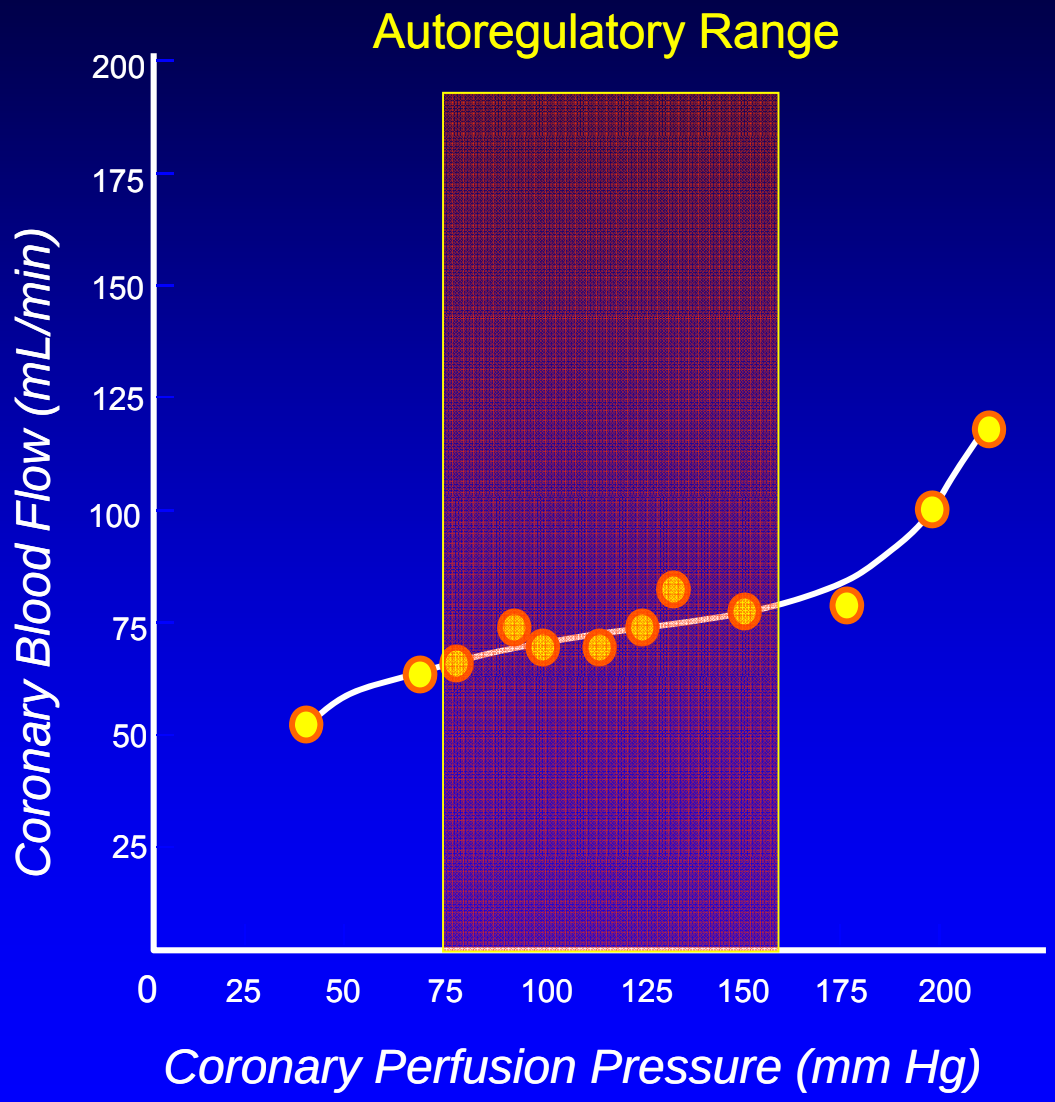
RUST **MAX. VASODILATATIE**

IF *we like to relate perfusion pressure to blood flow, resistance should be excluded as a variable.*
This can only be achieved by ***maximum arteriolar vasodilation***

Autoregulation

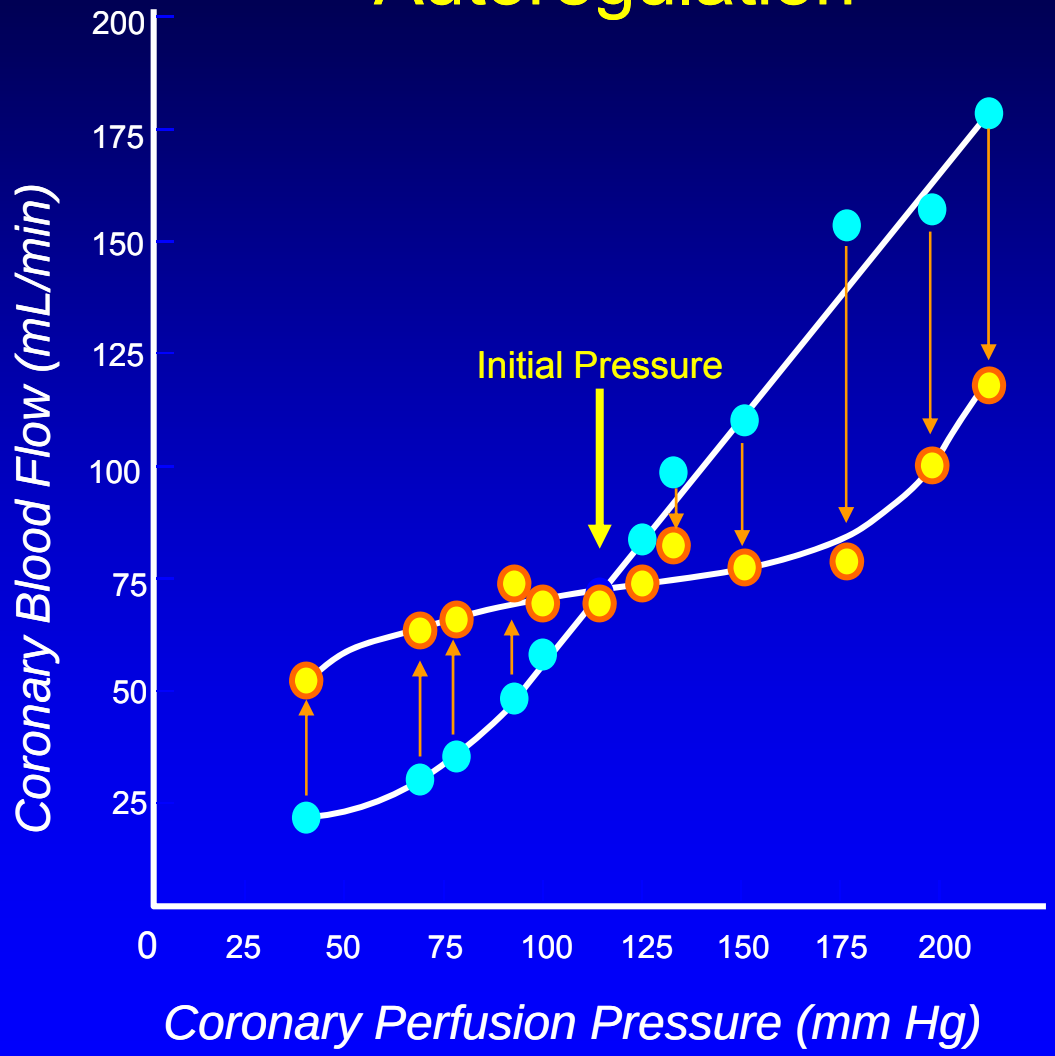


Rubio and Berne, Prog CV Disease 1975



Rubio and Berne, Prog CV Disease 1975

Autoregulation

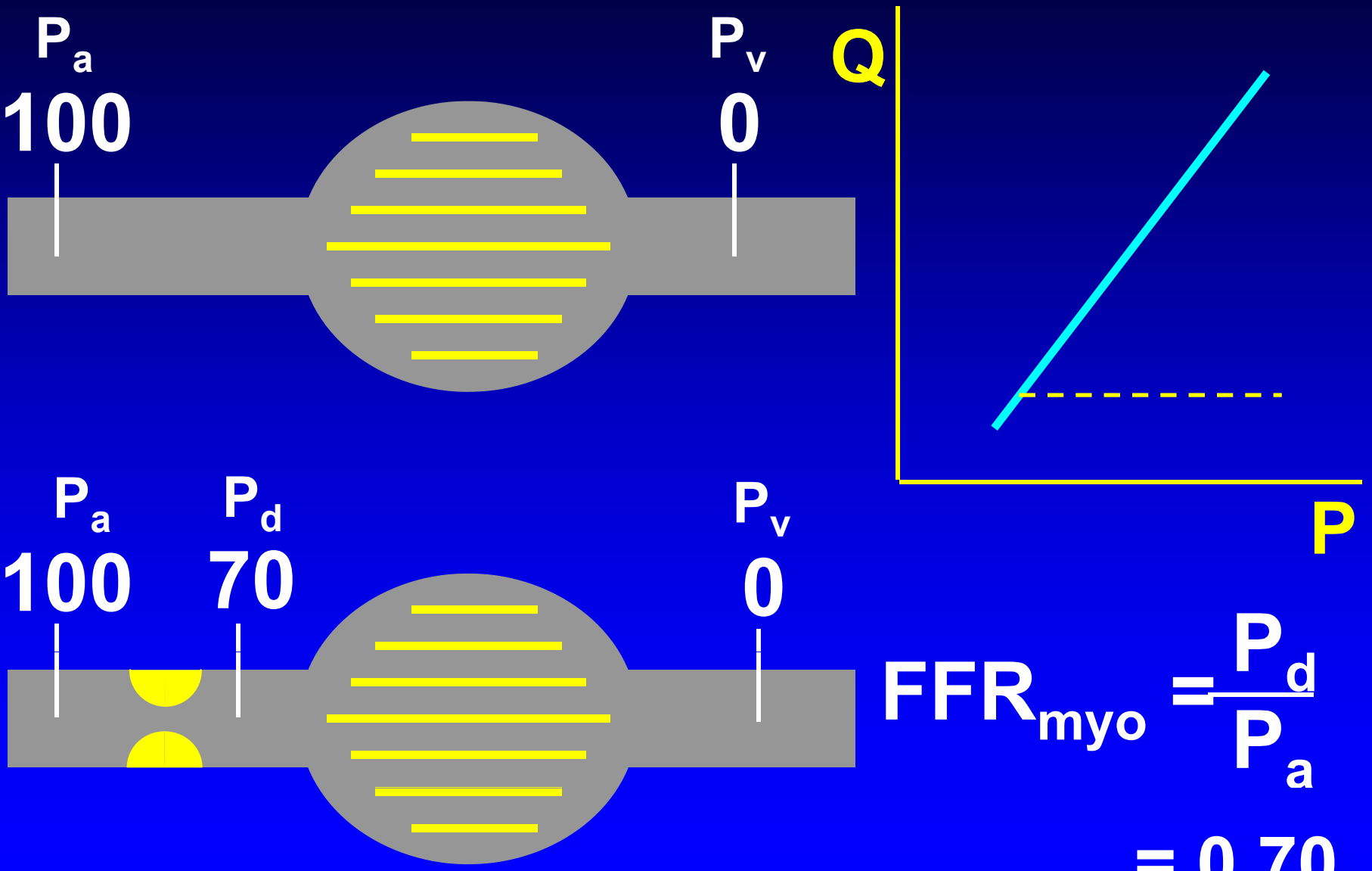


Rubio and Berne, Prog CV Disease 1975

Only During Maximal Vasodilatation (corresponding with minimum myocardial resistance), the ratio of pressures reflects a ratio of flows

→ **Basis of FFR**

During Maximal Vasodilatation



Is hyperemia truly mandatory?

recent ***hypothetical*** claim that “minimum resistance” is present during part of heart cycle ***at rest*** (*so-called wave free period ~ 75 % of diastole*)

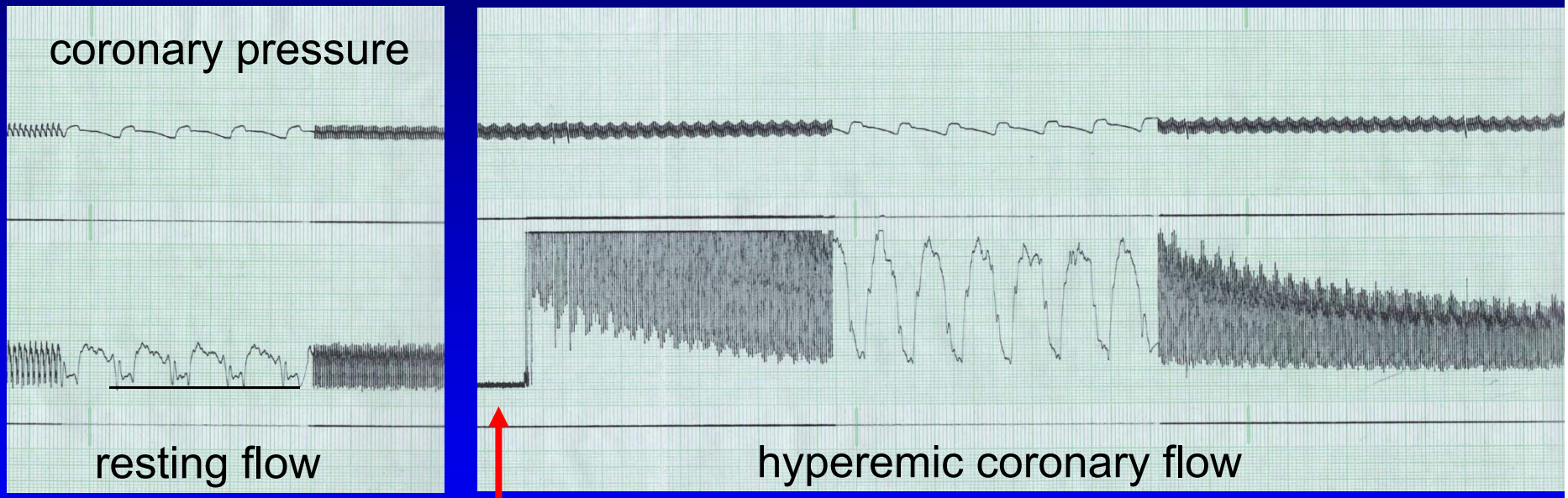
Not any experimental validation or evidence

Despite very complex background theory, the index “iFR” is simply calculated as a ratio of 2 pressures ***averaged*** over ~ 75 % of diastole at rest $(P_d / P_a)_{rest}$

So, in contrast to what the name suggests, there is *nothing “instantaneous” in this index*

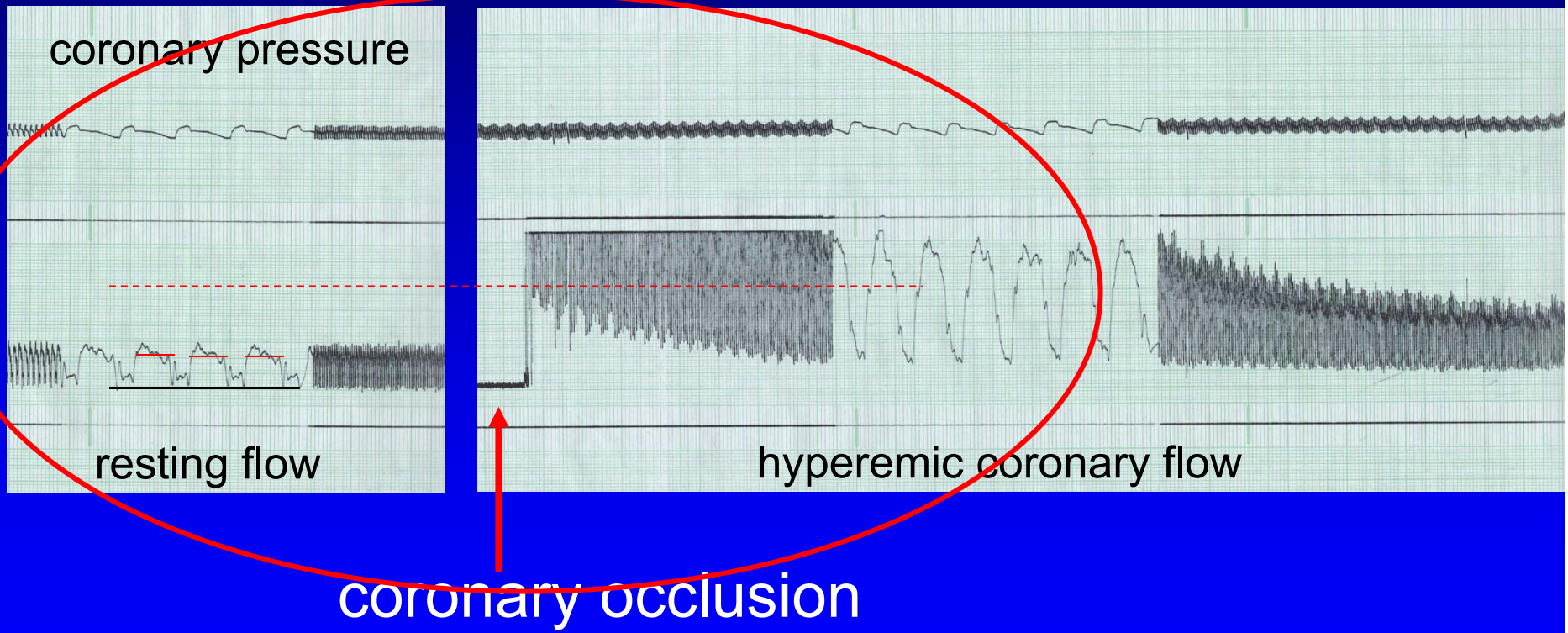
Neither is it “hyperemia-free” *(independent of hyperemia)*

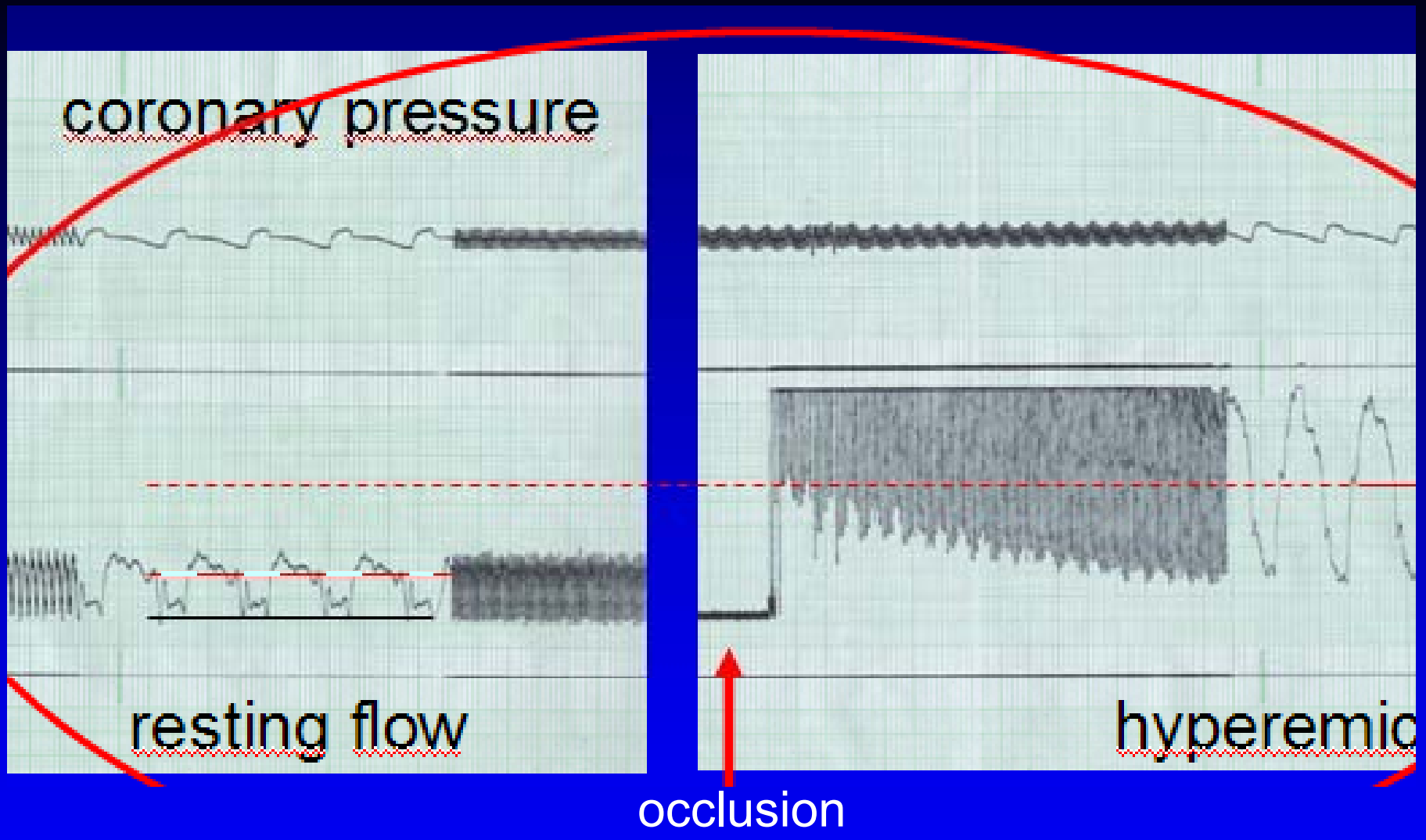
simultaneous recording of coronary pressure and absolute volumetric coronary blood flow at baseline and during maximum hyperemia in a normal coronary artery in a dog



In the presence of constant coronary pressure

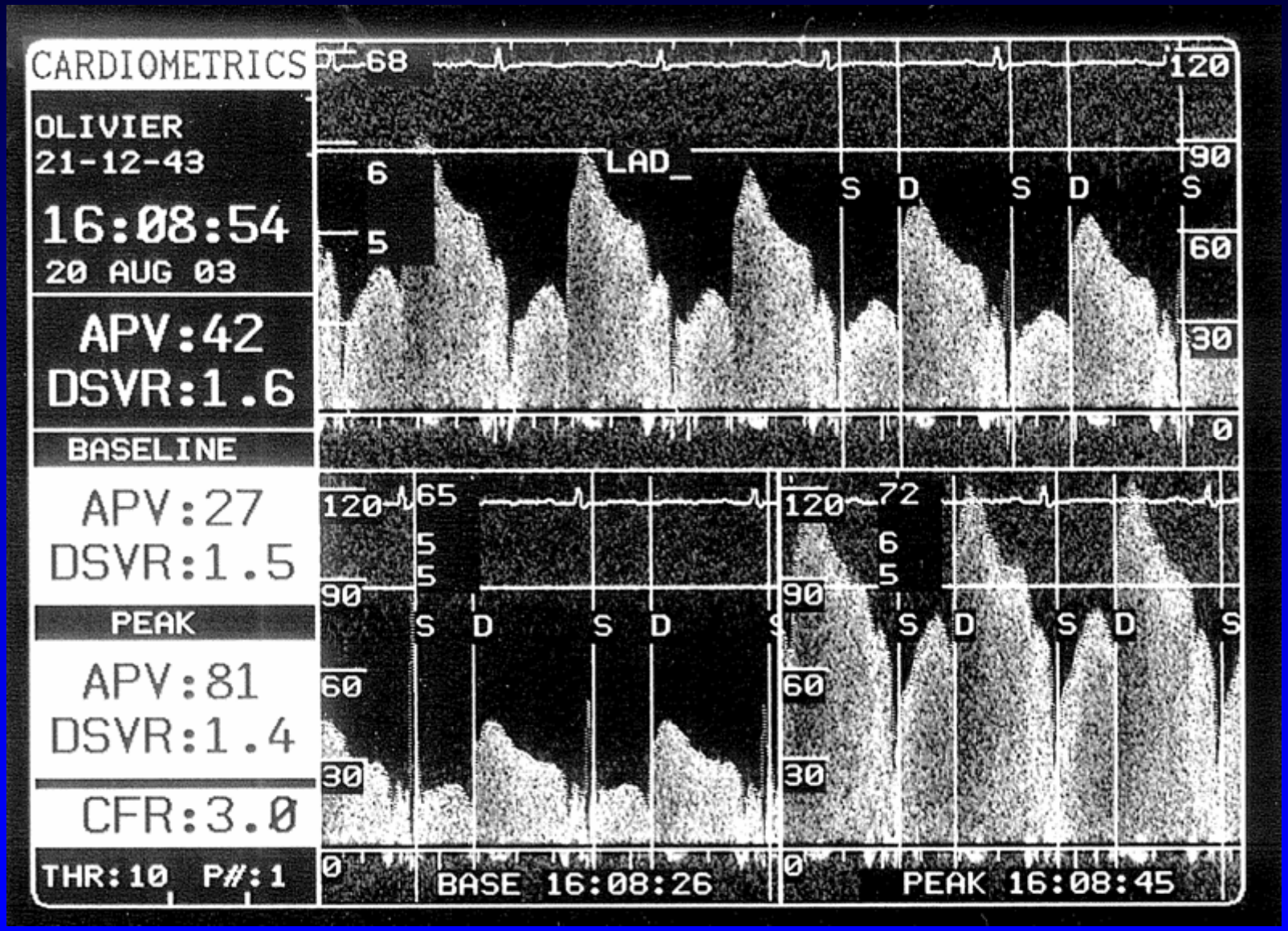
⟶ R ~ 1 / Flow





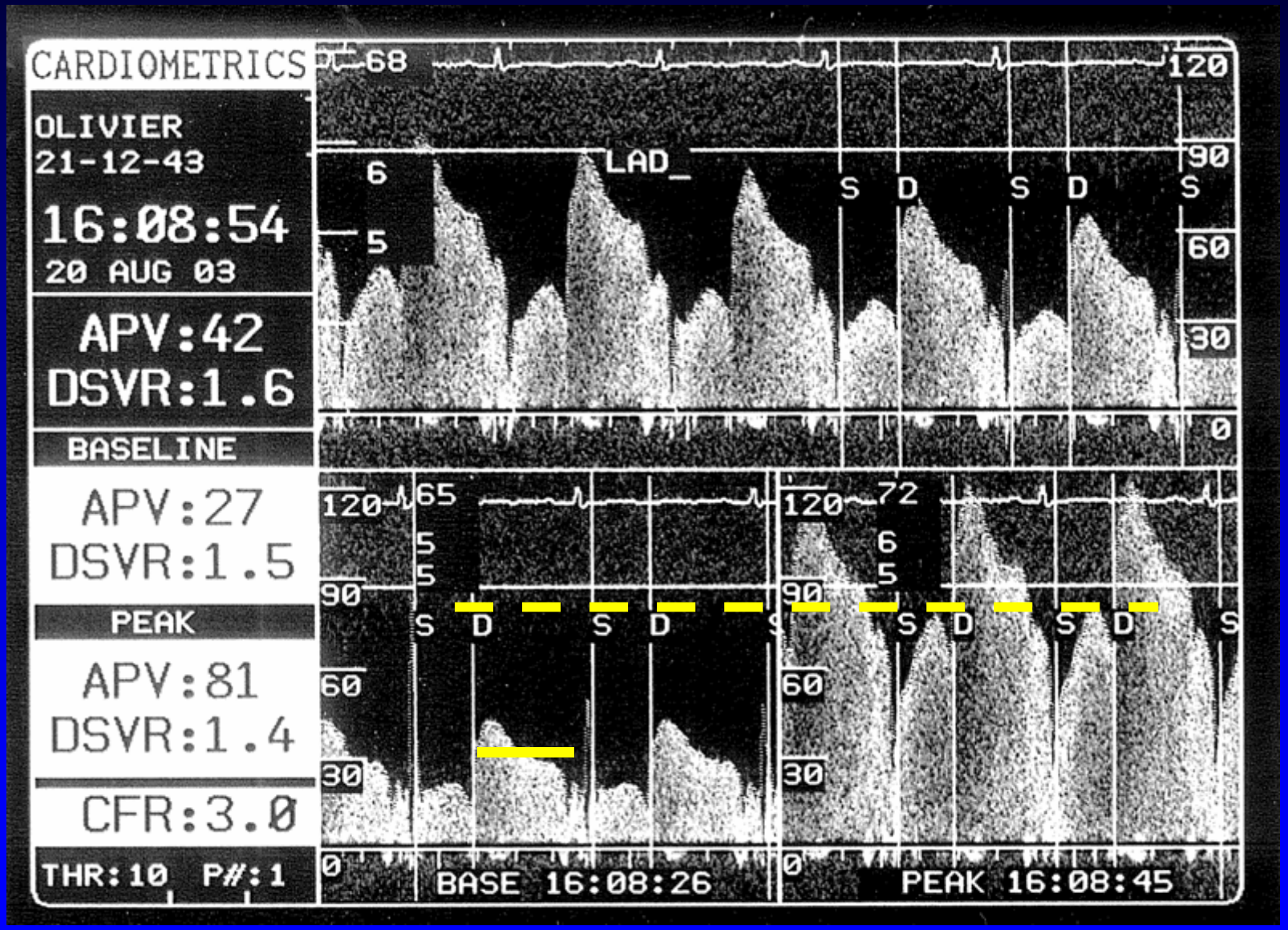
Even during any part of diastole (or “wave-free period”) ***at rest***, resistance is 3x higher than average hyperemic resistance

Doppler flow velocity recording in a human coronary artery



resting **hyperemia (adenosine)**

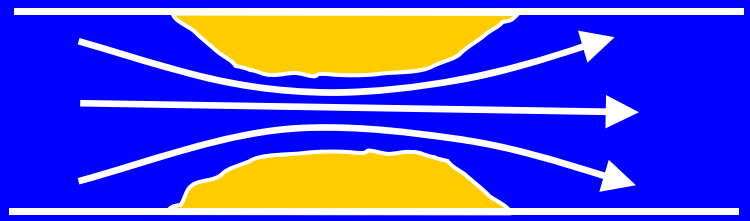
Doppler flow velocity recording in a human coronary artery
(constant pressure ⟶ R ~ 1 / Flow)



resting **hyperemia (adenosine)**

$\Delta P = f.Q + s.Q^2$

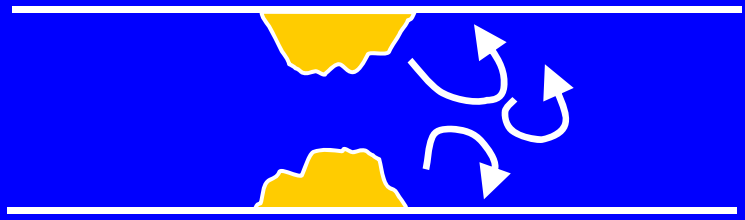
f = friction coefficient



Moderate gradient at rest

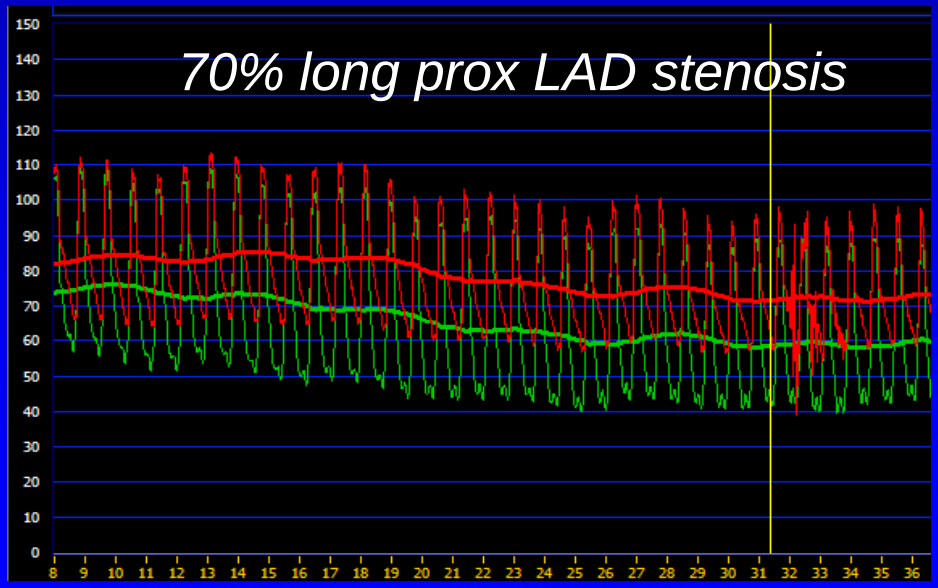
Moderate increment at hyperemia

S = separation coefficient

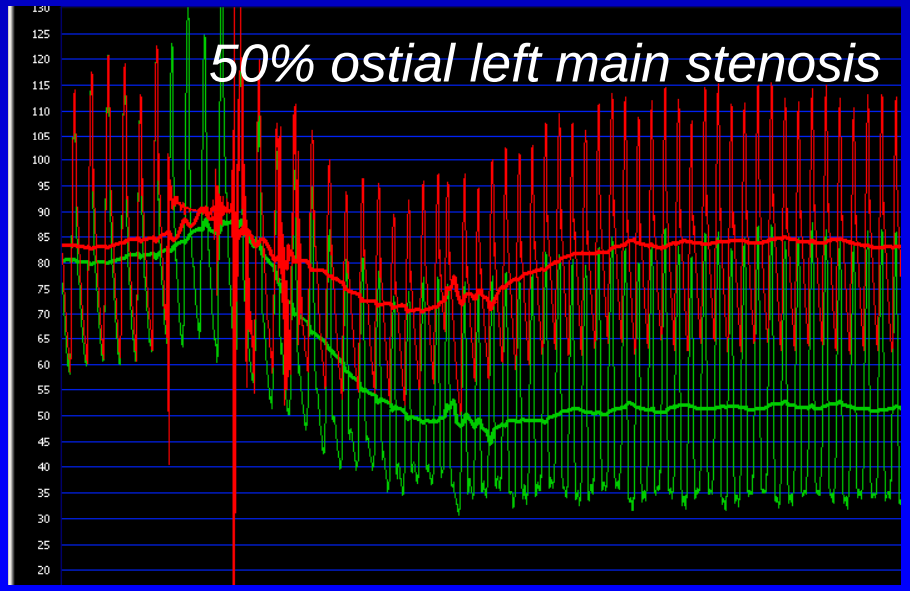


Small gradient at rest

Large gradient at hyperemia



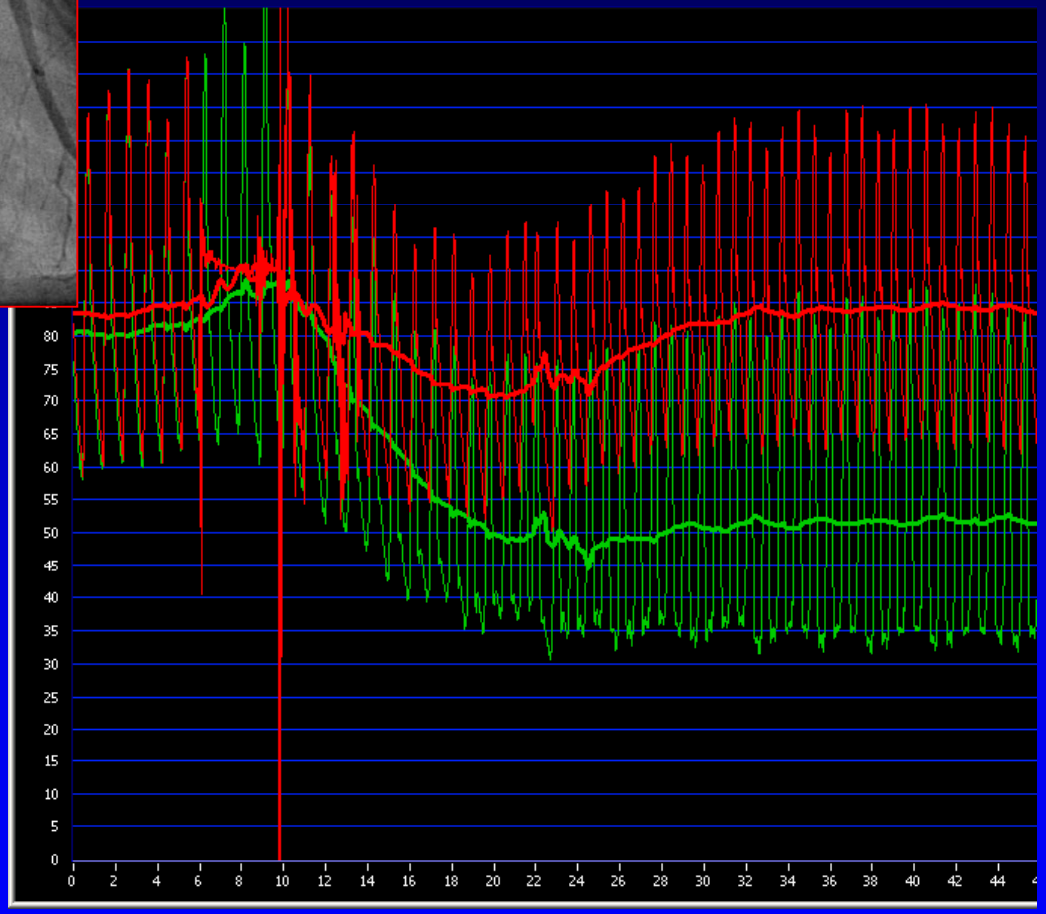
iFR = 0.89 FFR = 0.85



iFR = 0.94 FFR = 0.57

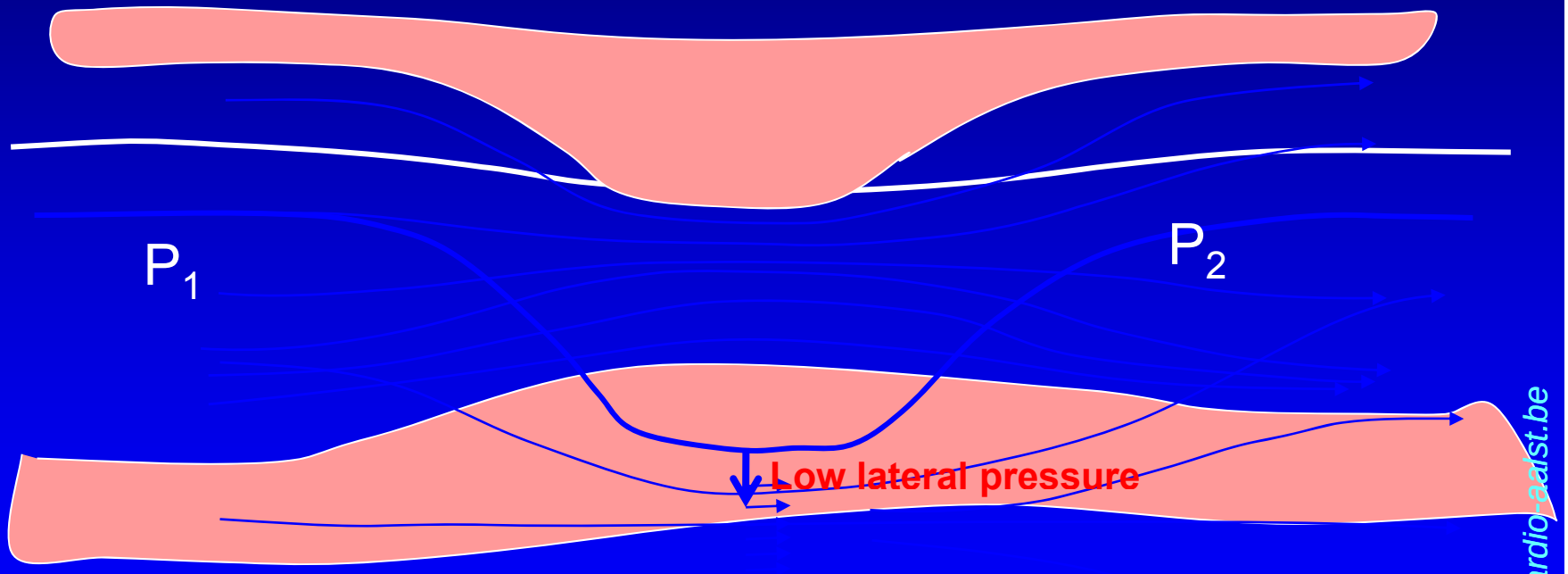
CONCLUSIONS:

- Physiologic assessment of coronary artery stenosis is important and has significant implications for outcome
- Therefore, some knowledge and insights in coronary physiology is mandatory
- Fractional Flow Reserve (FFR) is an easy technique with a sound physiologic basis, well validated both experimentally and clinically vs a true gold standard
- Maximum hyperemia is paramount to avoid “guessing” and to take the correct decisions with respect to decision-making on revascularization



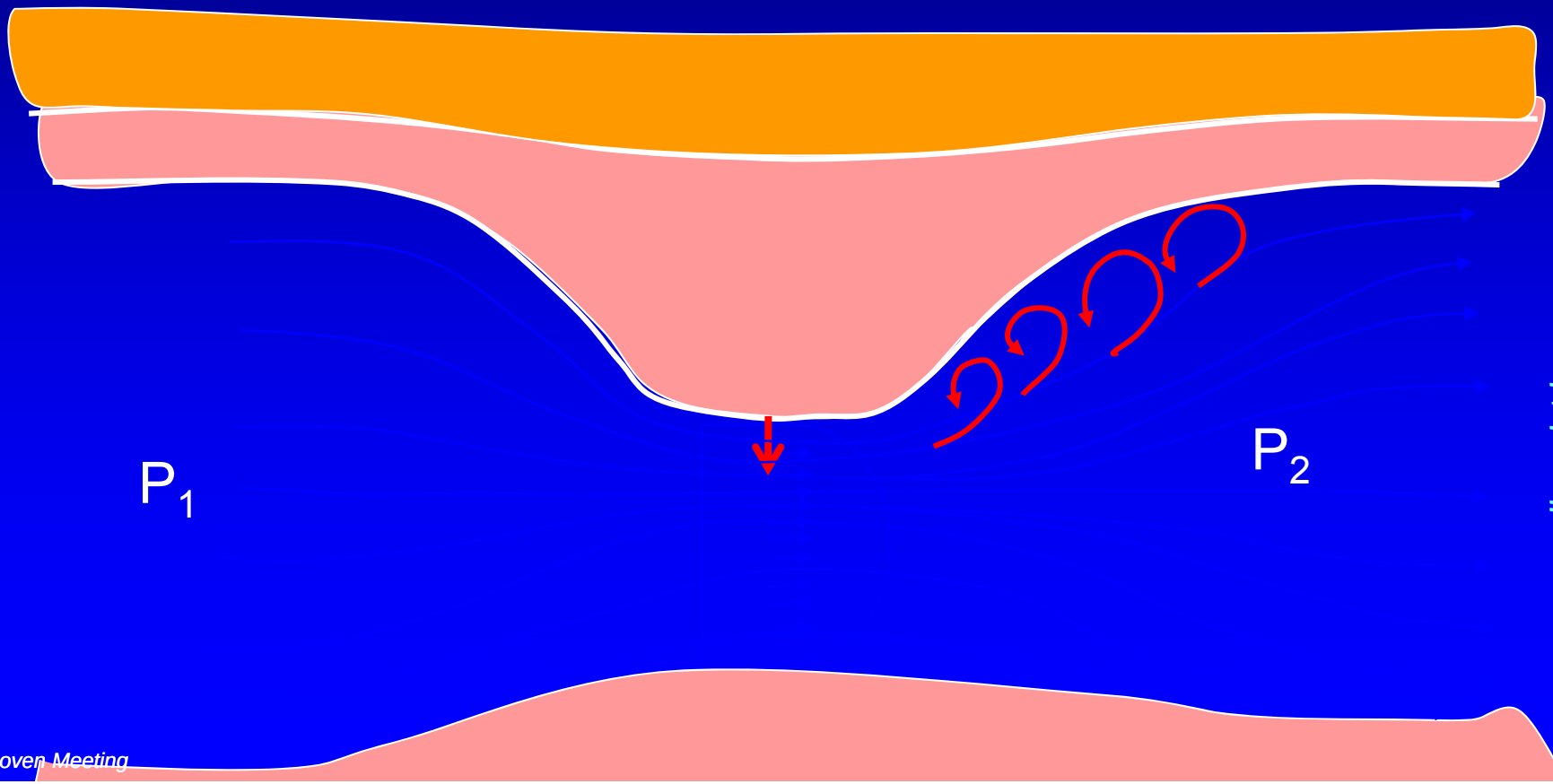
Mechanical constraints on coronary stenoses

Decreased lateral pressure (Venturi)

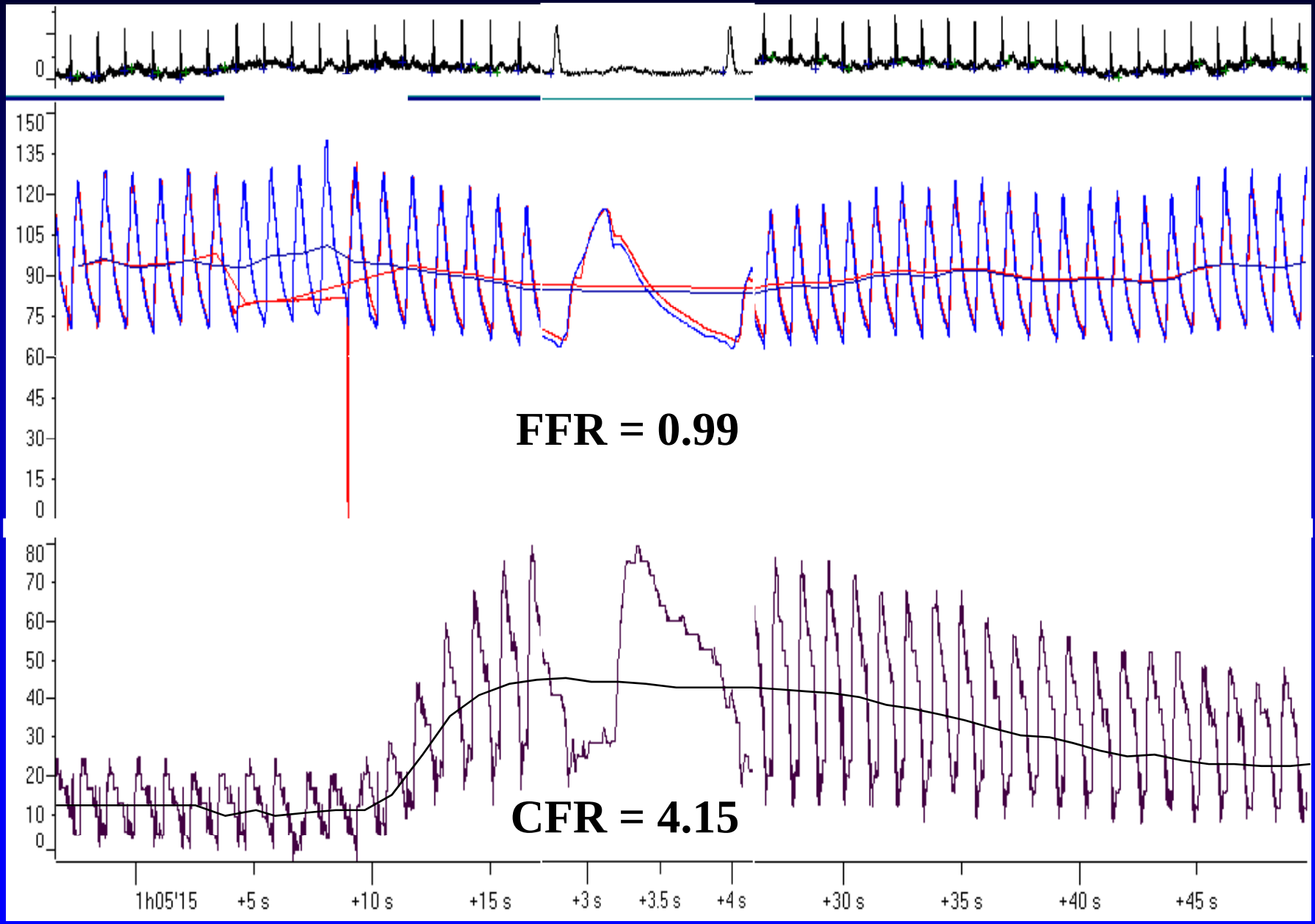


Mechanical constraints on coronary stenoses

Turbulences = unfavorable rheologic conditions

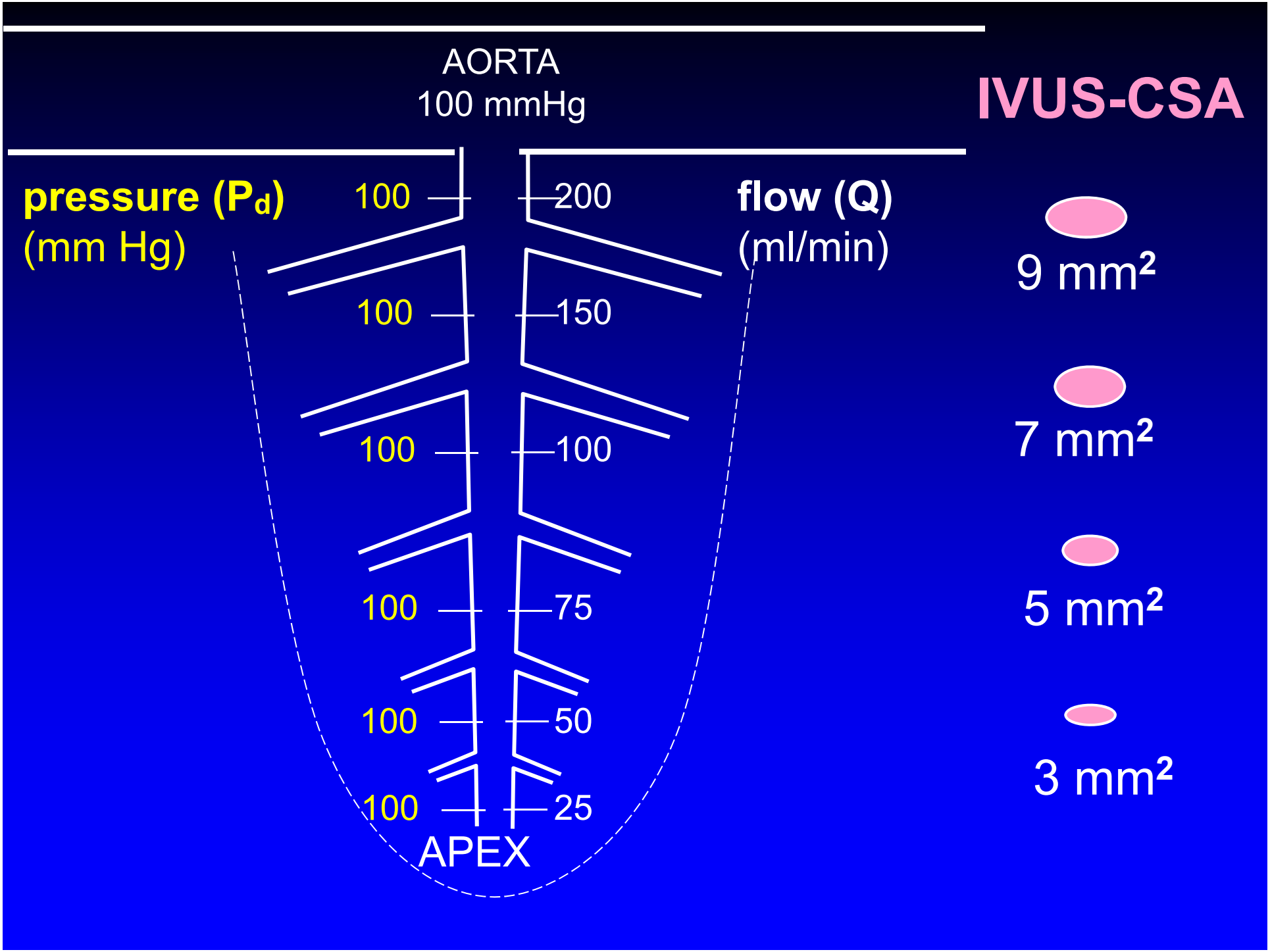


Normal Coronary Artery



Tekstdia over noodzaak hyperemia

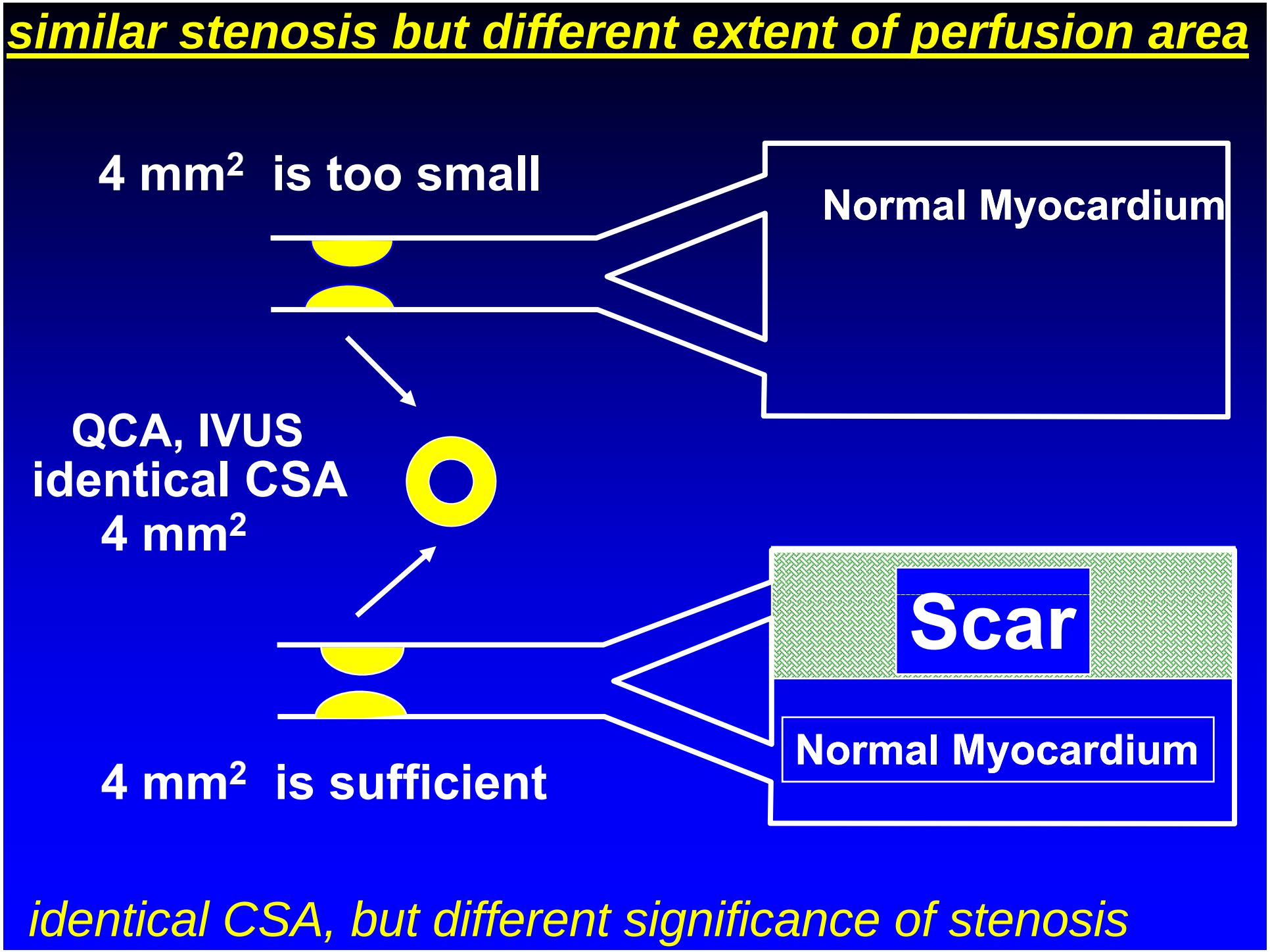
+ evt 1-2 dia’s uit iFR-FFR
(result + ifrrestvshyp)


AORTA
100 mmHg
IVUS-CSA
pressure (P_d)
(mm Hg)
flow (Q)
(ml/min)
100
200
100
150
100
100
100
75
100
50
100
25
APEX
9 mm²
7 mm²
5 mm²
3 mm²

! We cannot understand the physiologic significance of a stenosis without taking into account the extent of the distal perfusion territory !

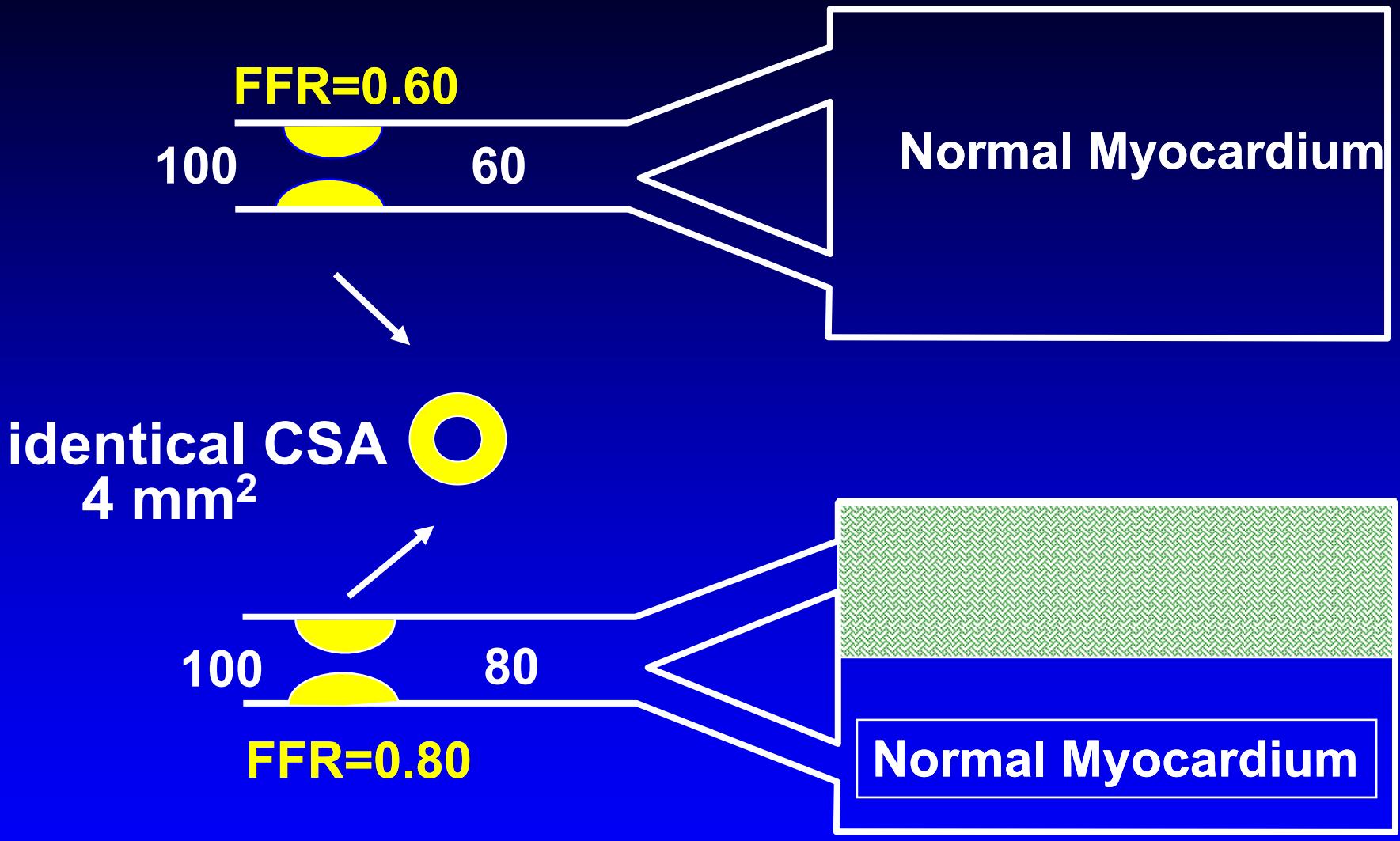
……*especially not under pathologic conditions, when the "physiologic match" between vessel size and perfusion area has been lost*

similar stenosis but different extent of perfusion area



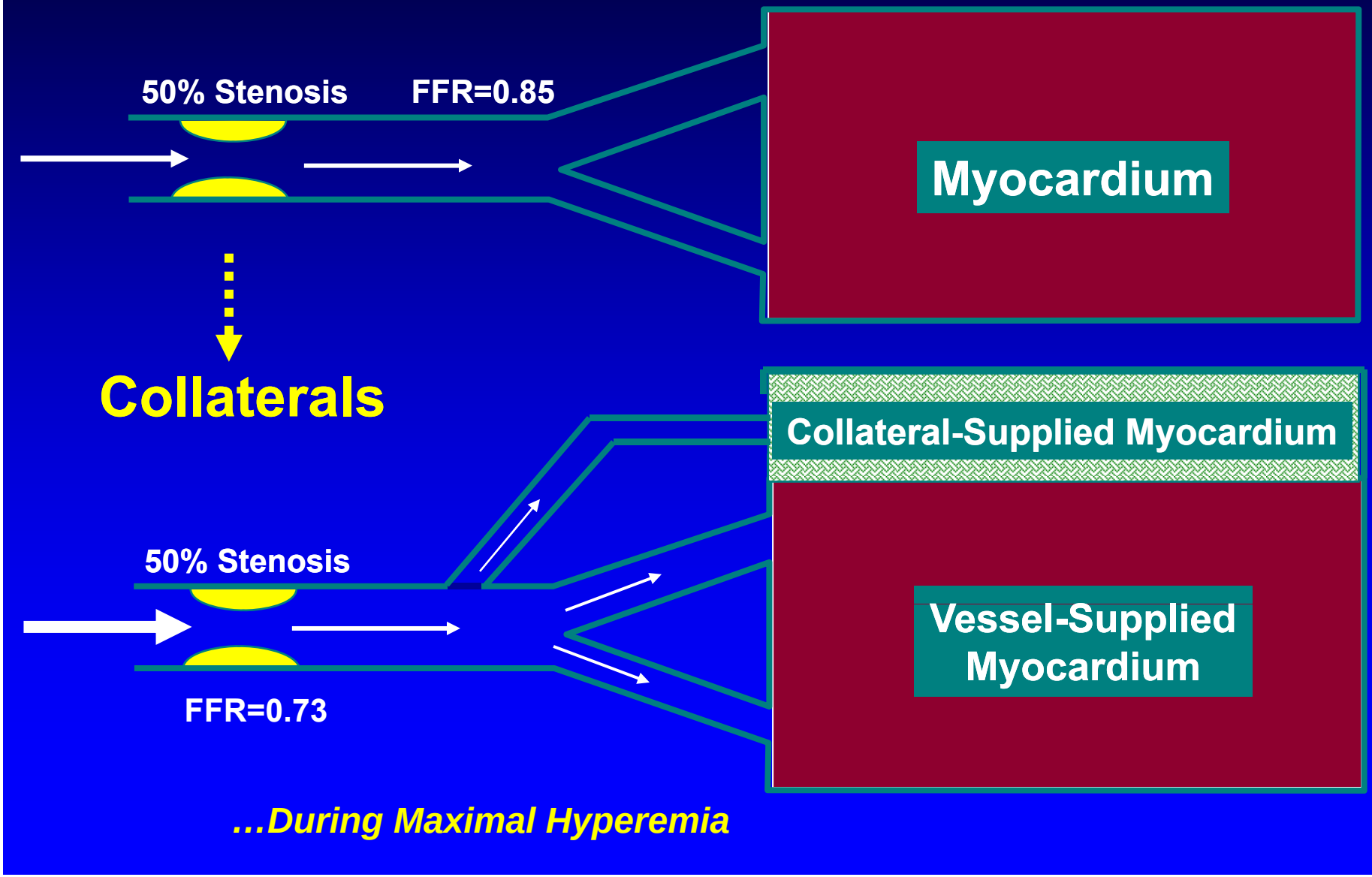
identical CSA, but different significance of stenosis

vessel size and myocardial mass after myocardial infarction:



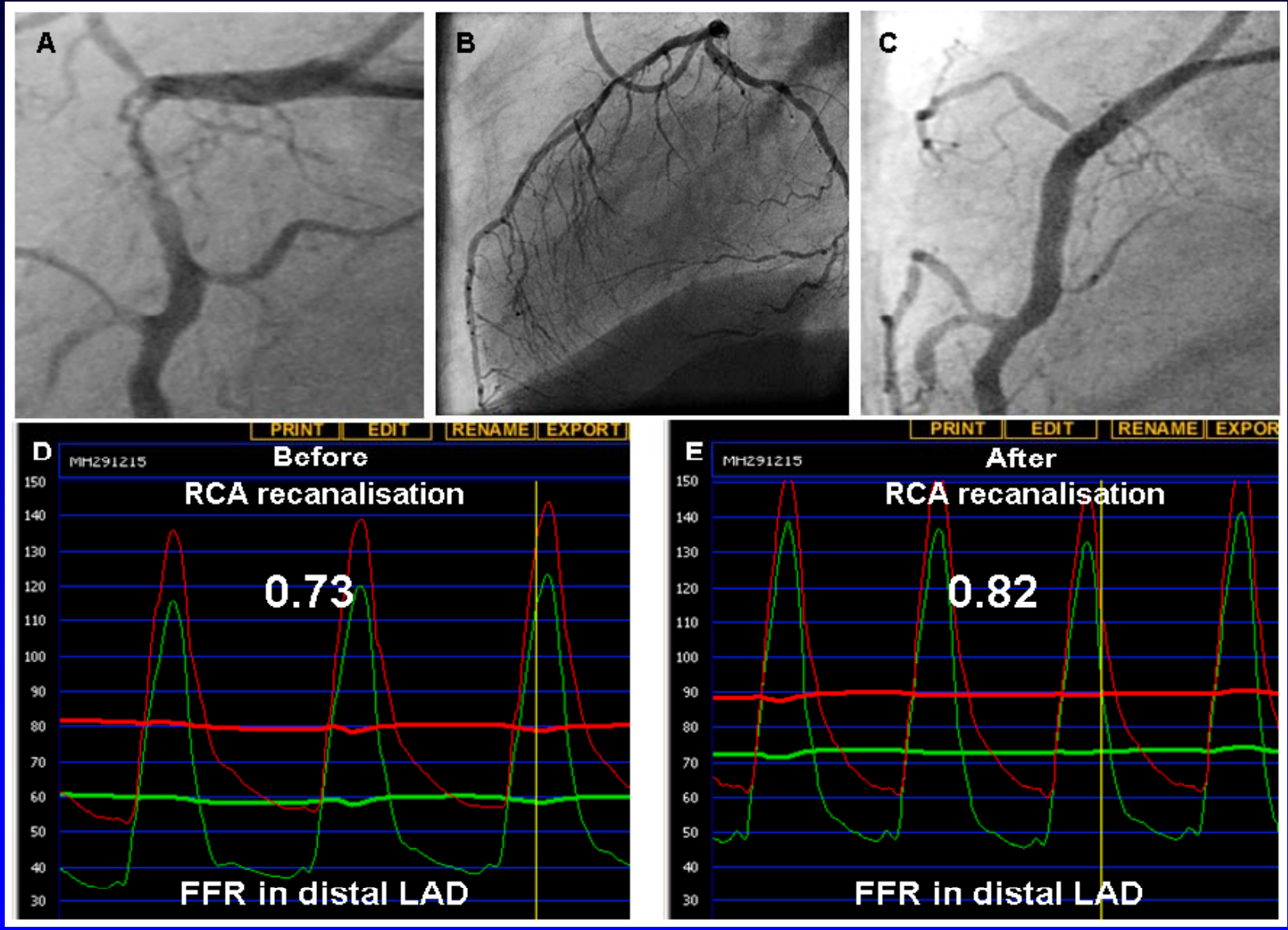
Anatomic stenosis severity is similar but Physiologic severity is different
→ reflected by FFR

Disconnect between Anatomy and Physiology



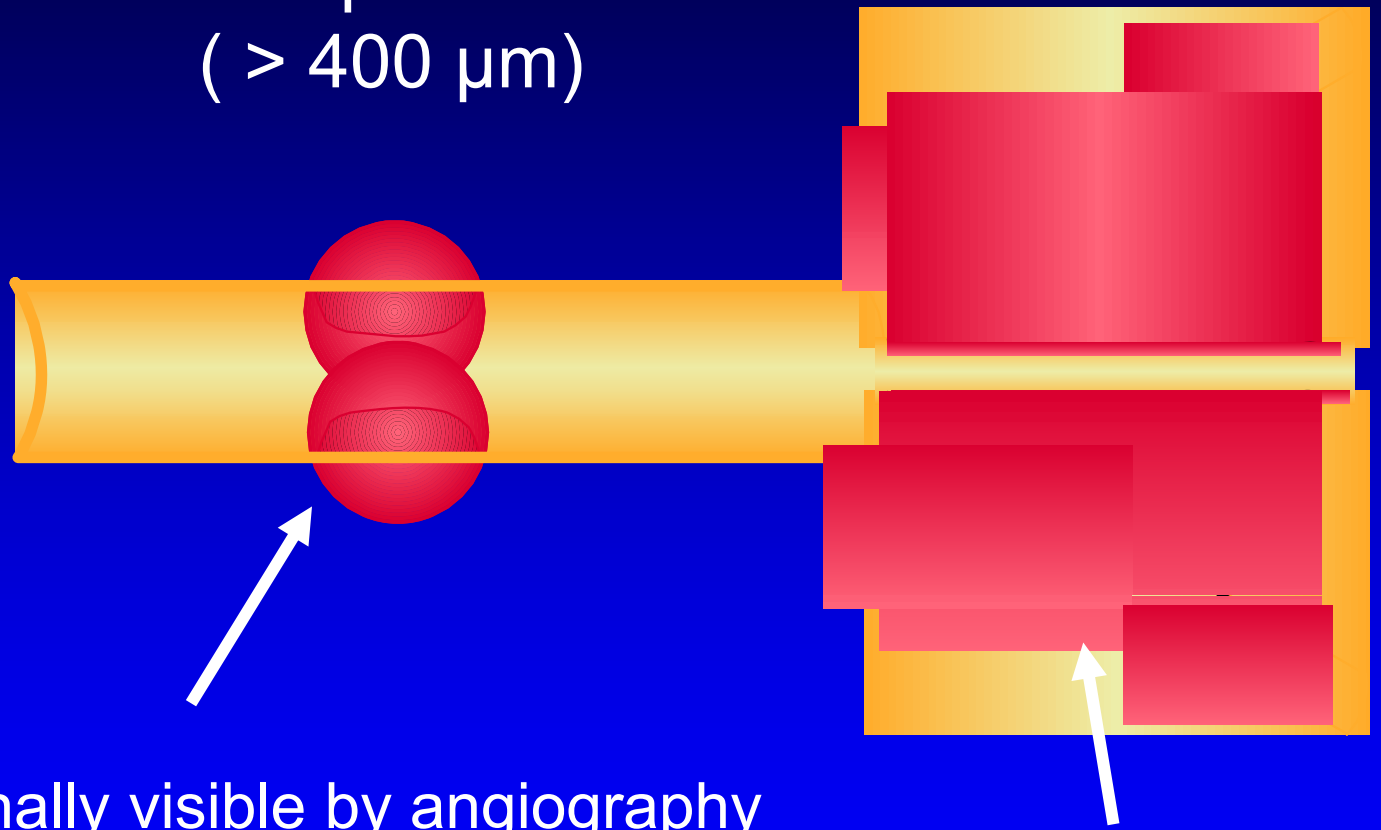
…During Maximal Hyperemia

FFR in the distal LAD before and After recanalization of the RCA



epicardial
compartment
(> 400 µm)

microvascular
compartment

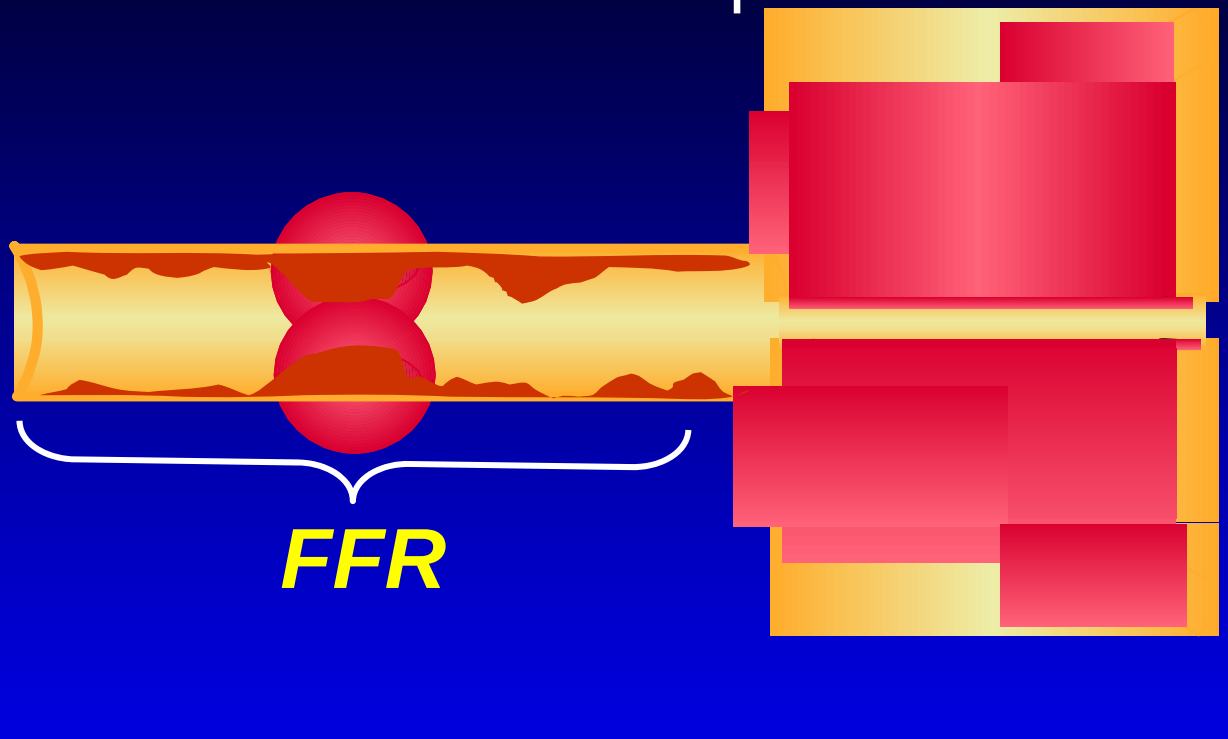


traditionally visible by angiography
and more recently by many invasive
and non-invasive imaging methods

Black box
(until recently)

focal ***and*** diffuse
epicardial disease

microvascular
compartment



hard to distinguish by
traditional methods,
but easily assessed
and quantified by FFR

The 3rd compartment:

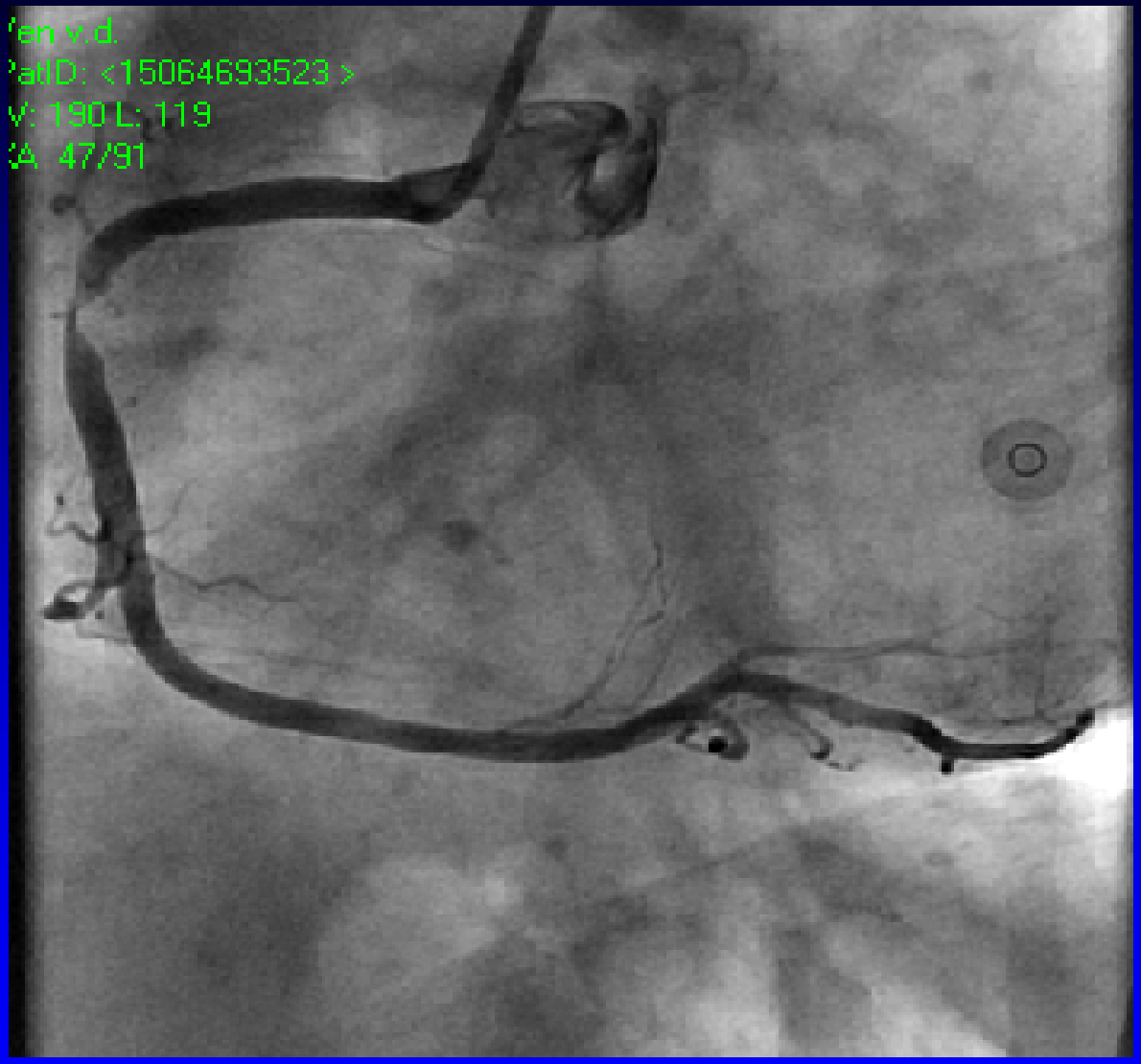
Diffuse epicardial coronary disease
(Dr Fearon)

→ **easily evaluable by FFR**
(pressure pull-back recording)

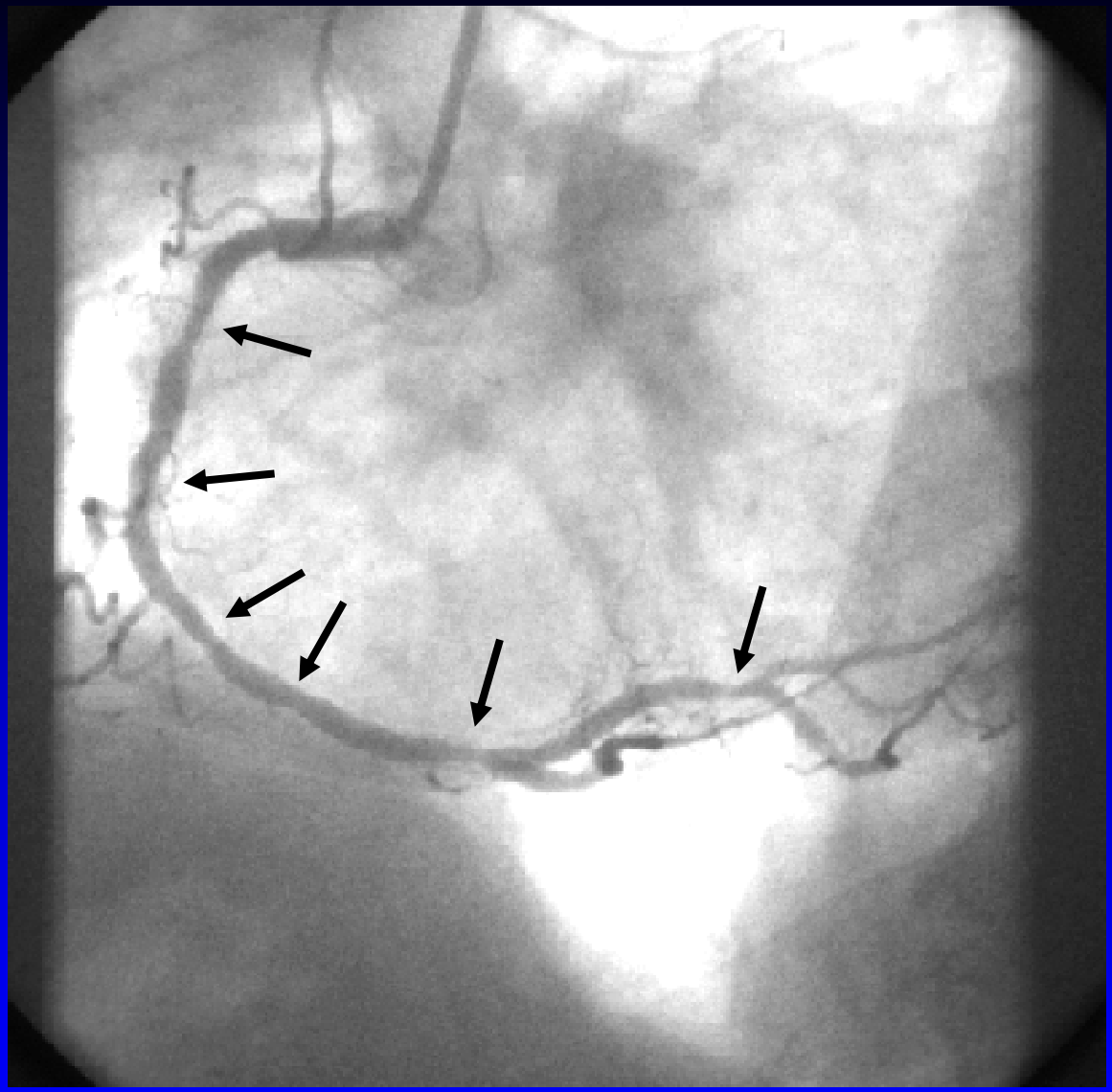
↓

important consequence for treatment
(interventional or medical)

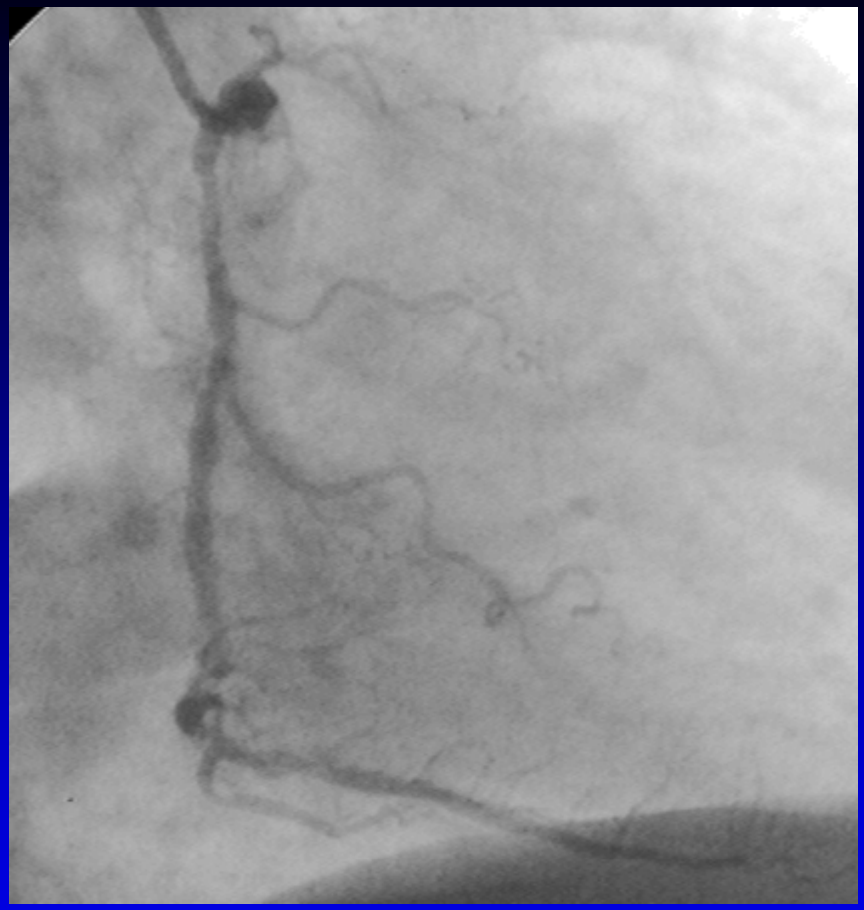
Male 58-y-old



Typical chest pain; positive MIBI-Spect inferior wall

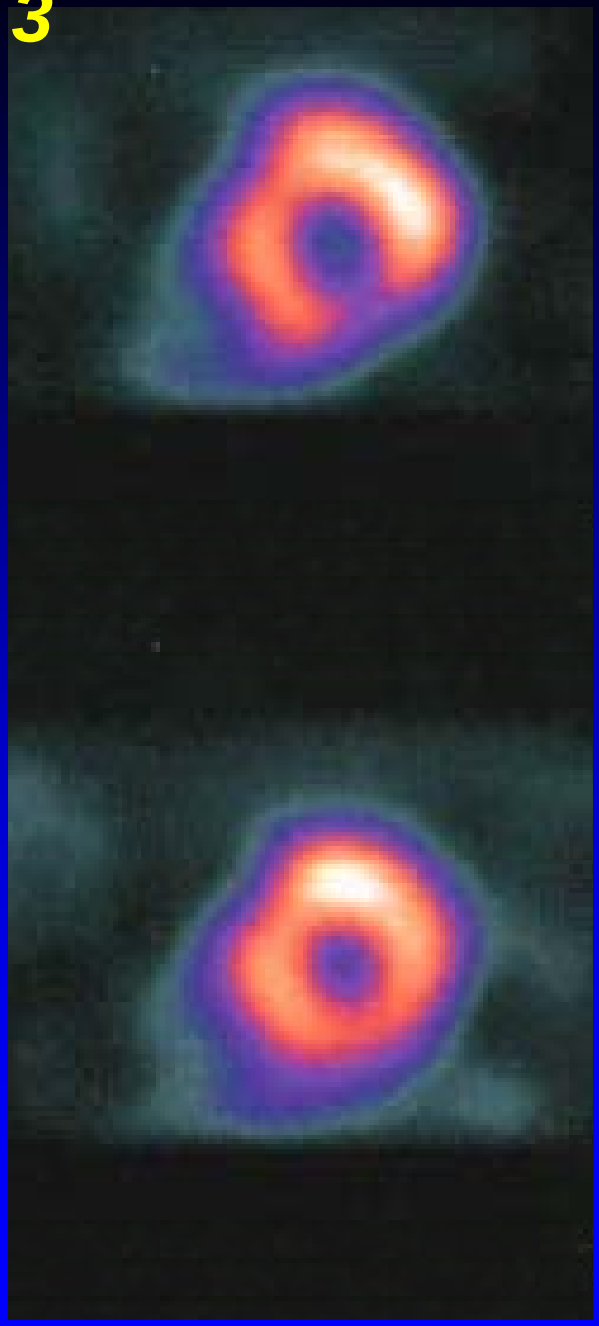


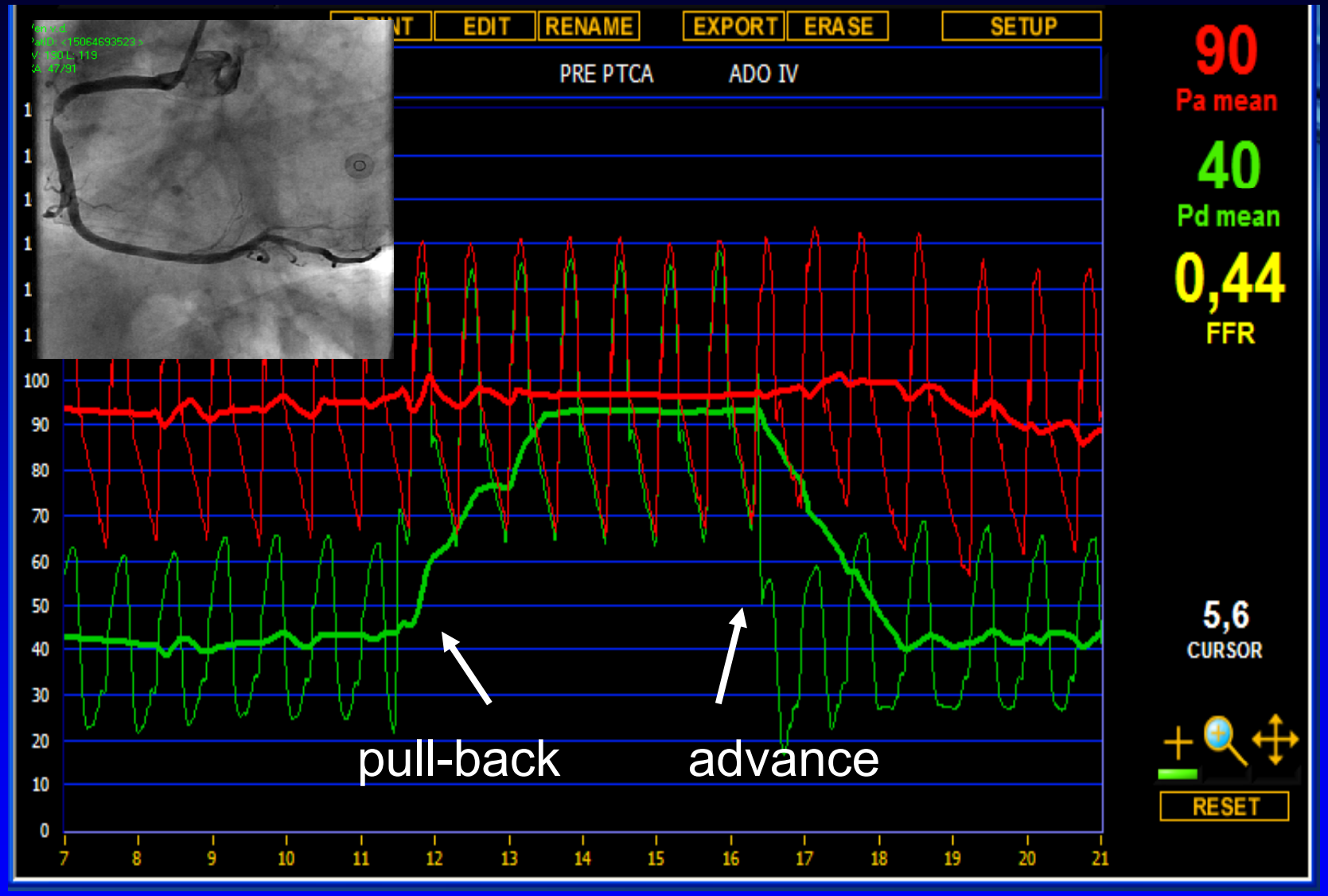
Typical chest pain; positive MIBI-Spect inferior wall

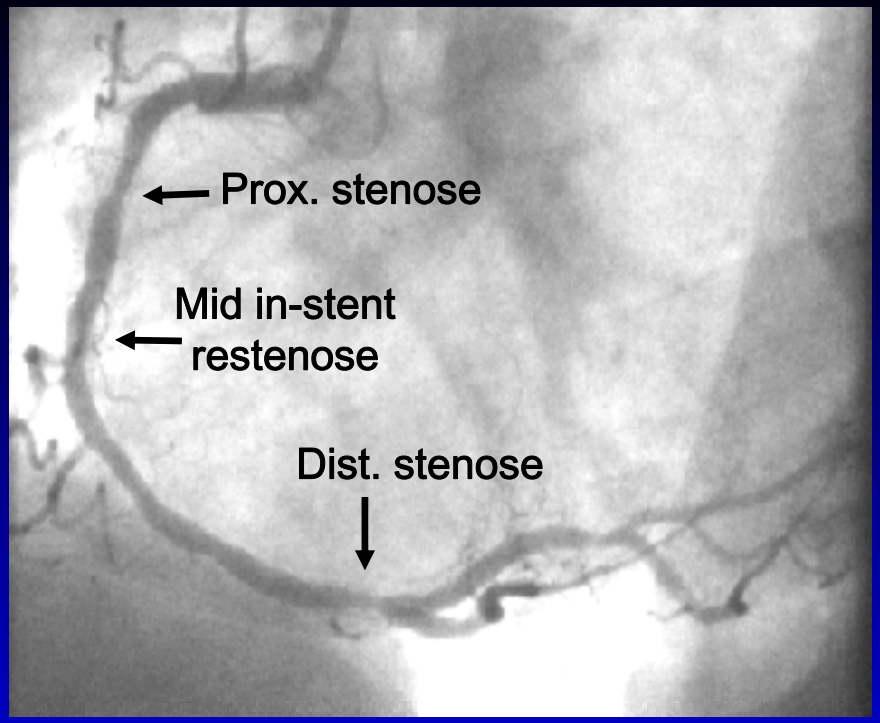


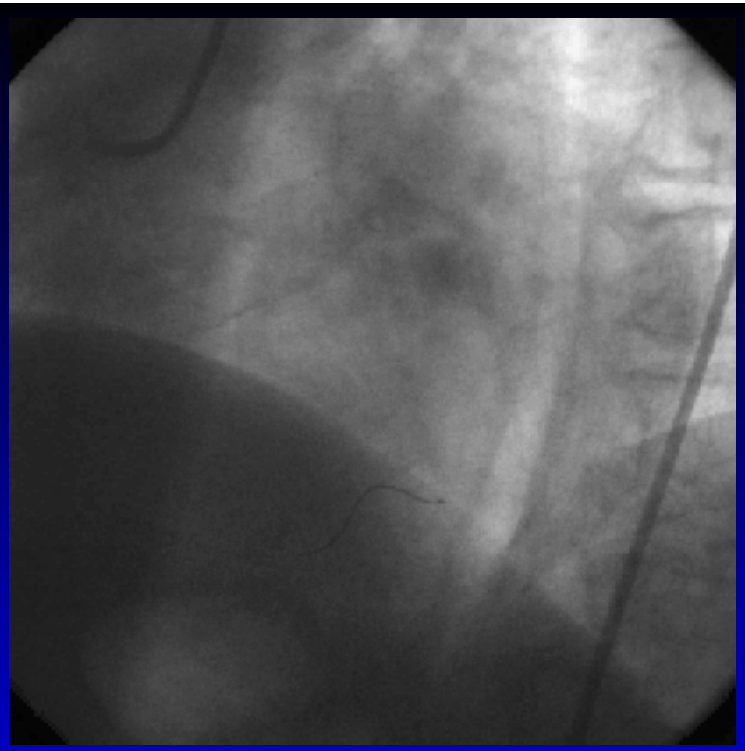
Typical chest pain; positive MIBI-Spect inferior wall

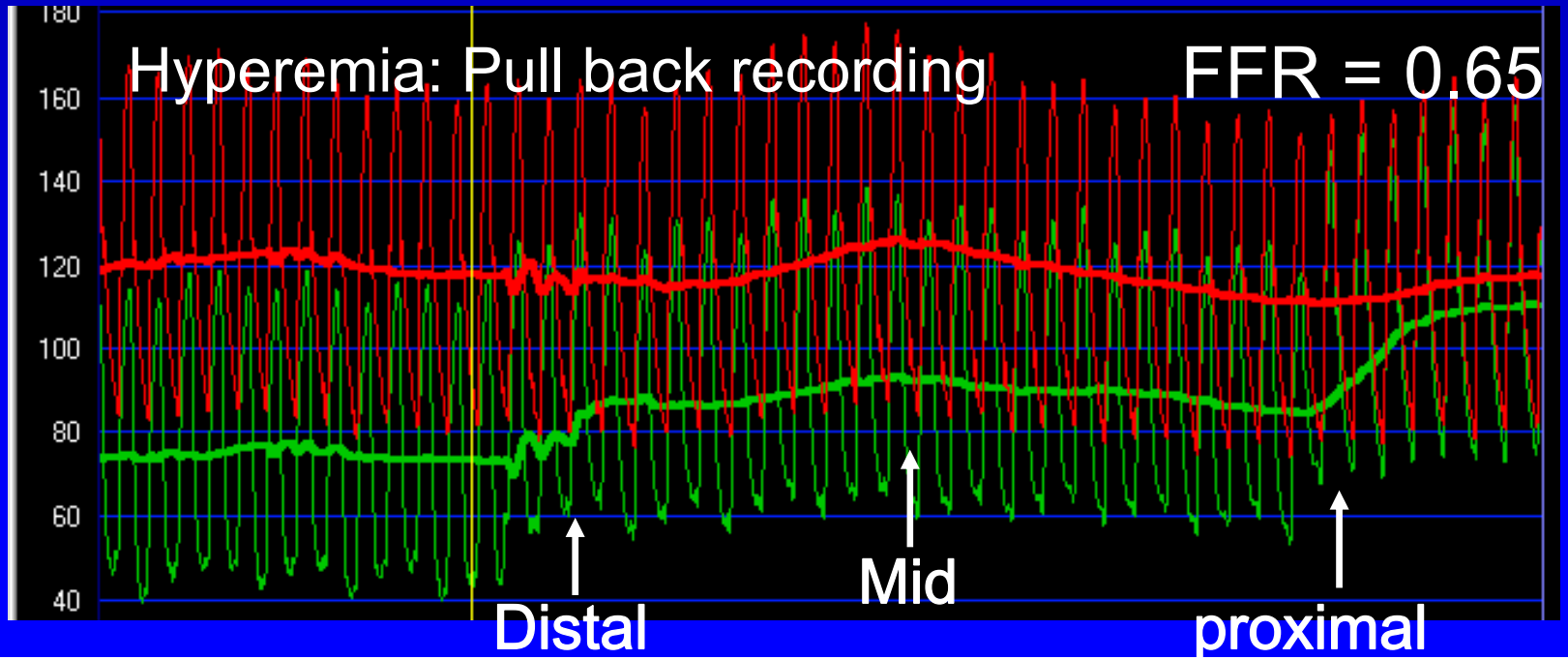
CASE # 3

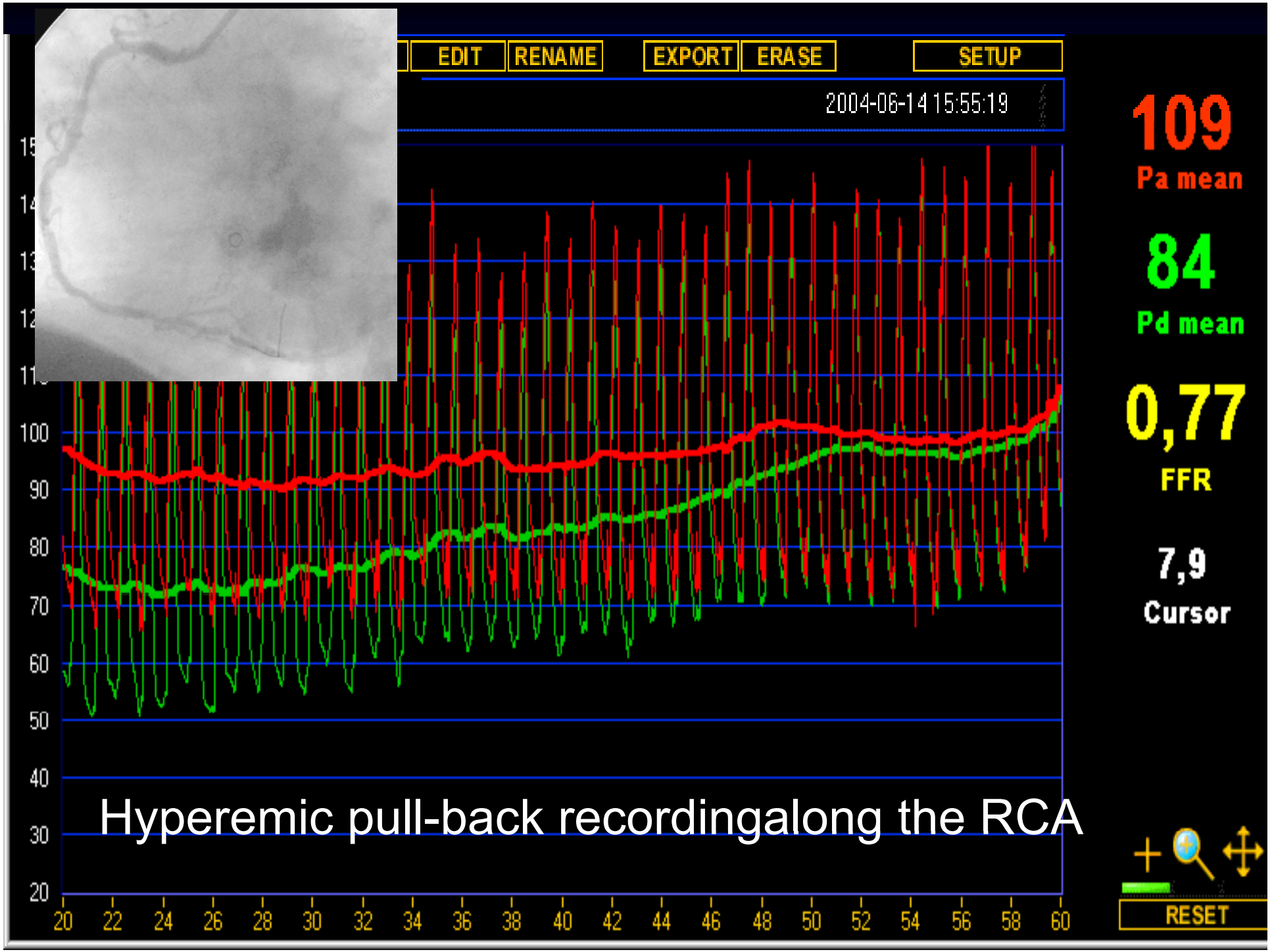



EDIT
RENAME
EXPORT
ERASE
SETUP
PRE PTCA
ADO IV
90
Pa mean
40
Pd mean
0,44
FFR
5,6
CURSOR
RESET
pull-back
advance
100
90
80
70
60
50
40
30
20
10
0
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21


Prox. stenose
Mid in-stent restenose
Dist. stenose

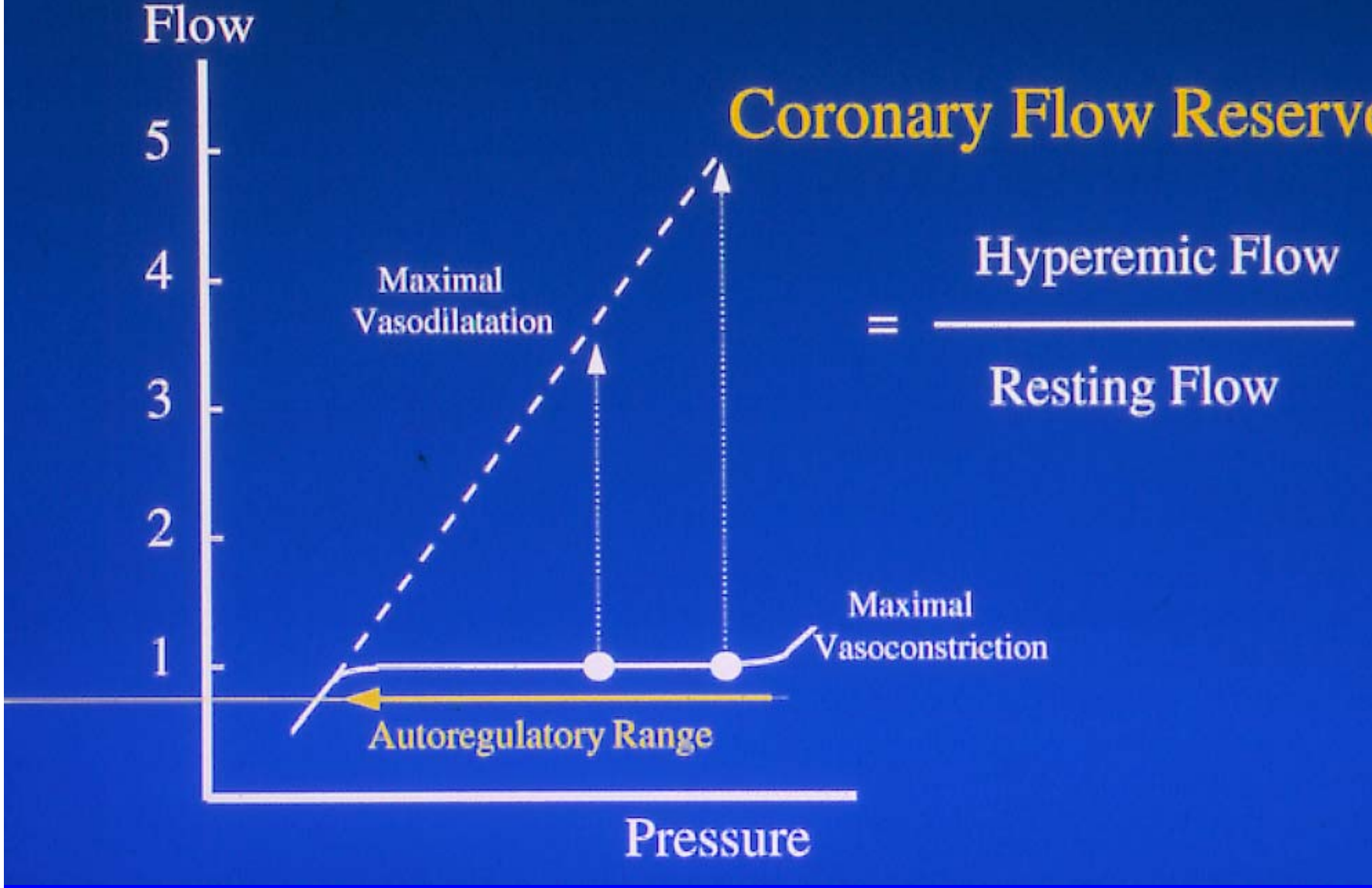



Hyperemia: Pull back recording
FFR = 0.65
180
160
140
120
100
80
60
40
Distal
Mid
proximal


EDIT
RENAME
EXPORT
ERASE
SETUP
2004-06-14 15:55:19
109
Pa mean
84
Pd mean
0,77
FFR
7,9
Cursor
RESET
Hyperemic pull-back recordingalong the RCA

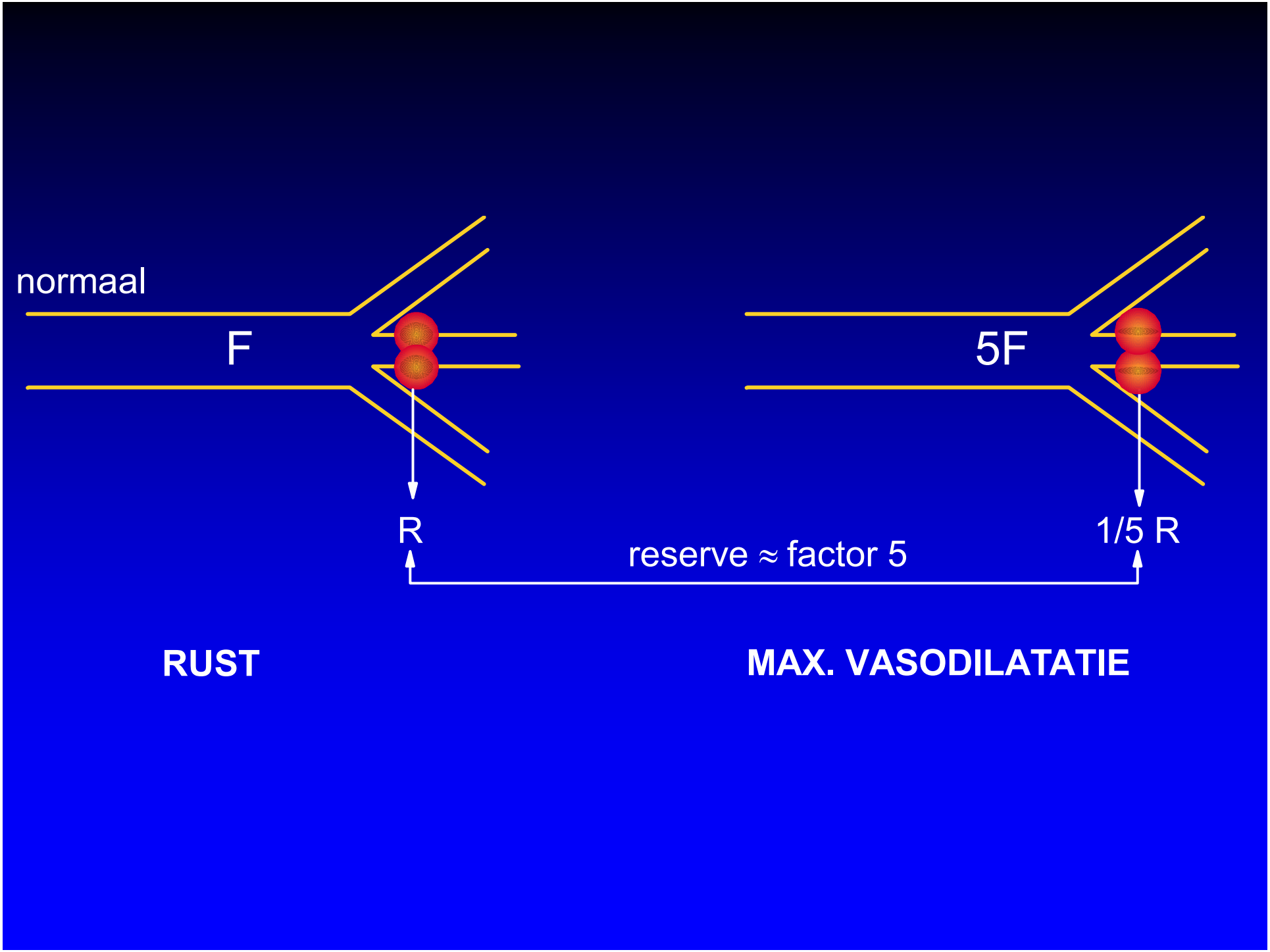
Soort conclusiedia

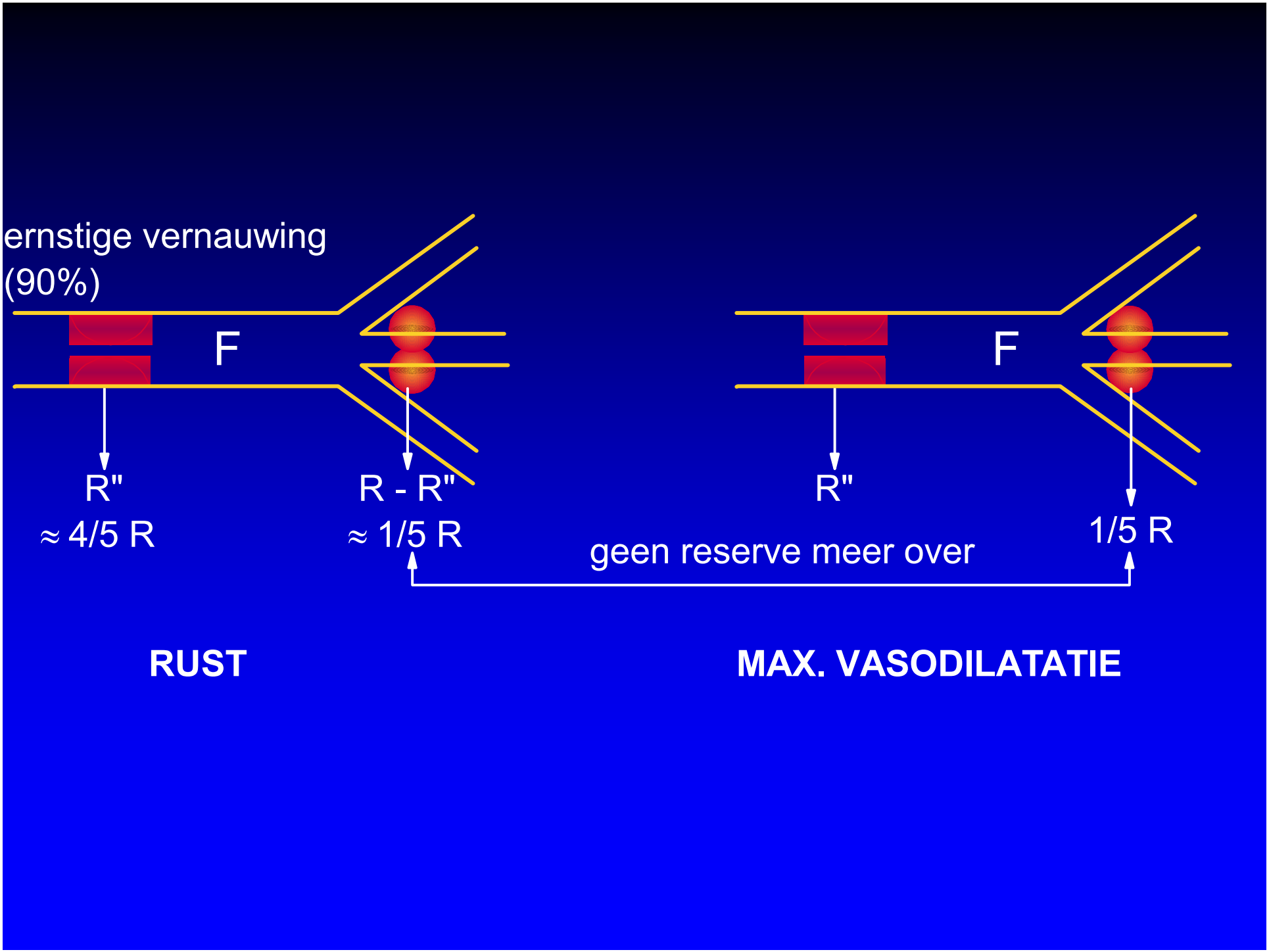
The Concept of Coronary Flow Reserve

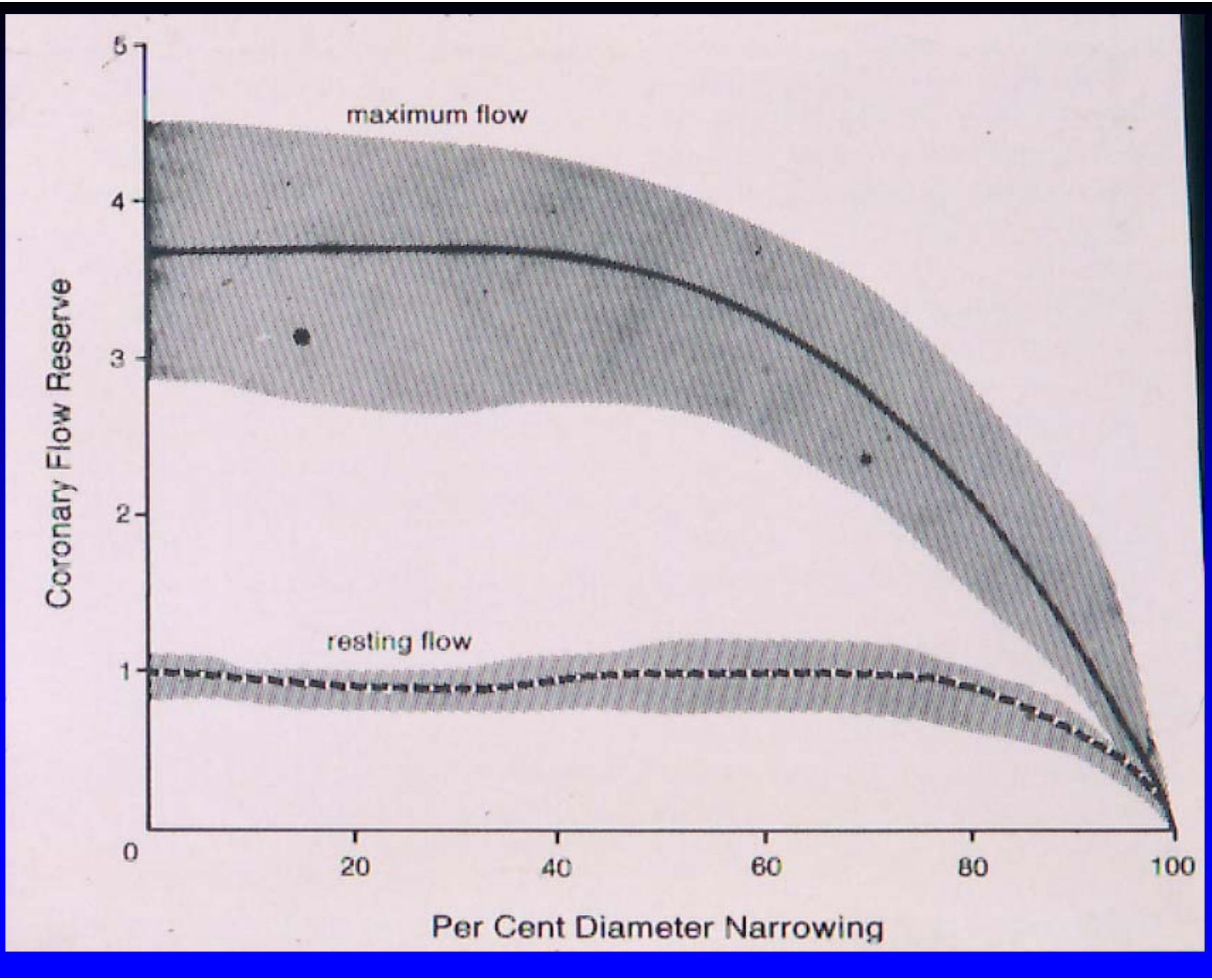


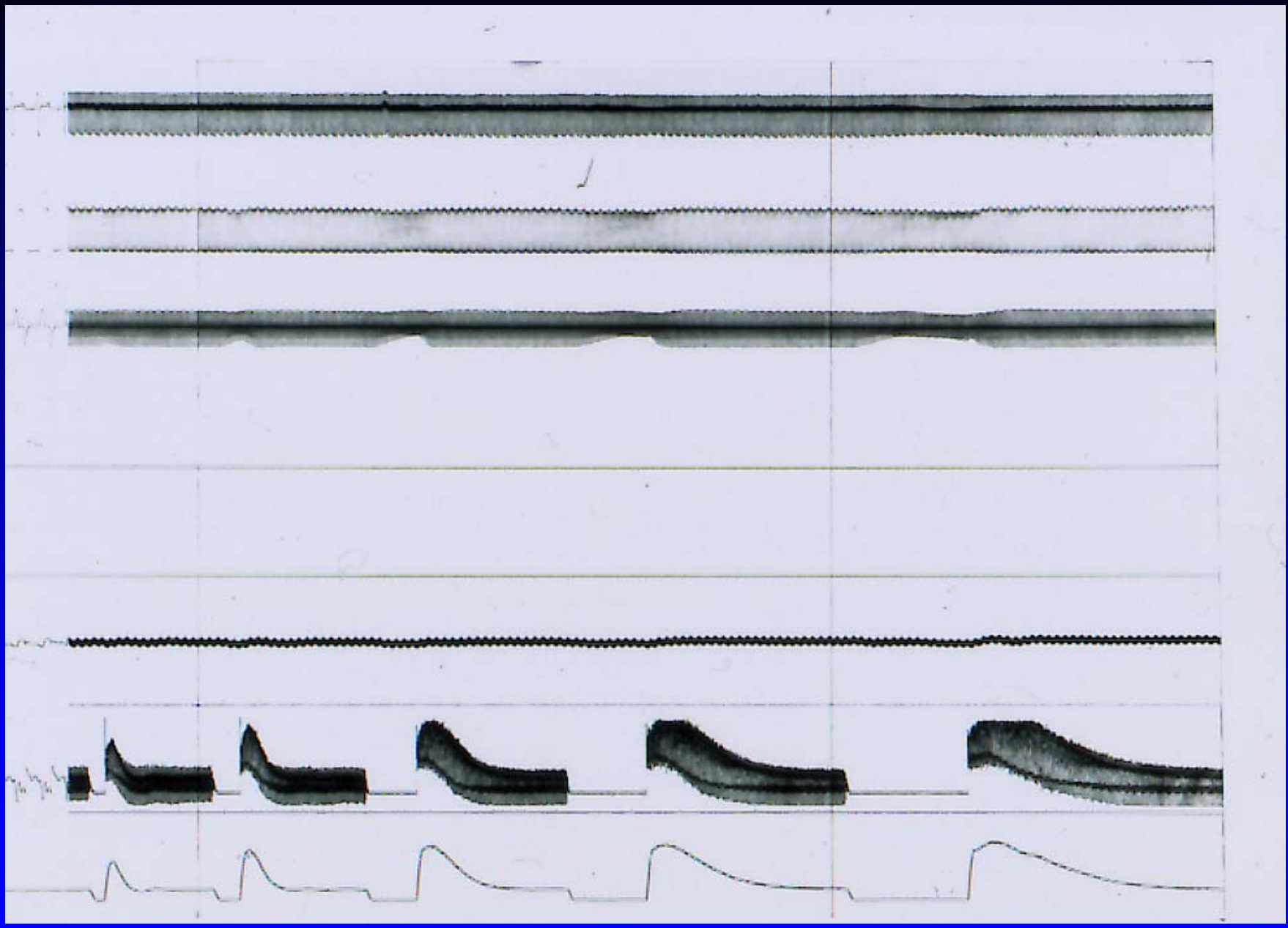
Coronary Flow Reserve

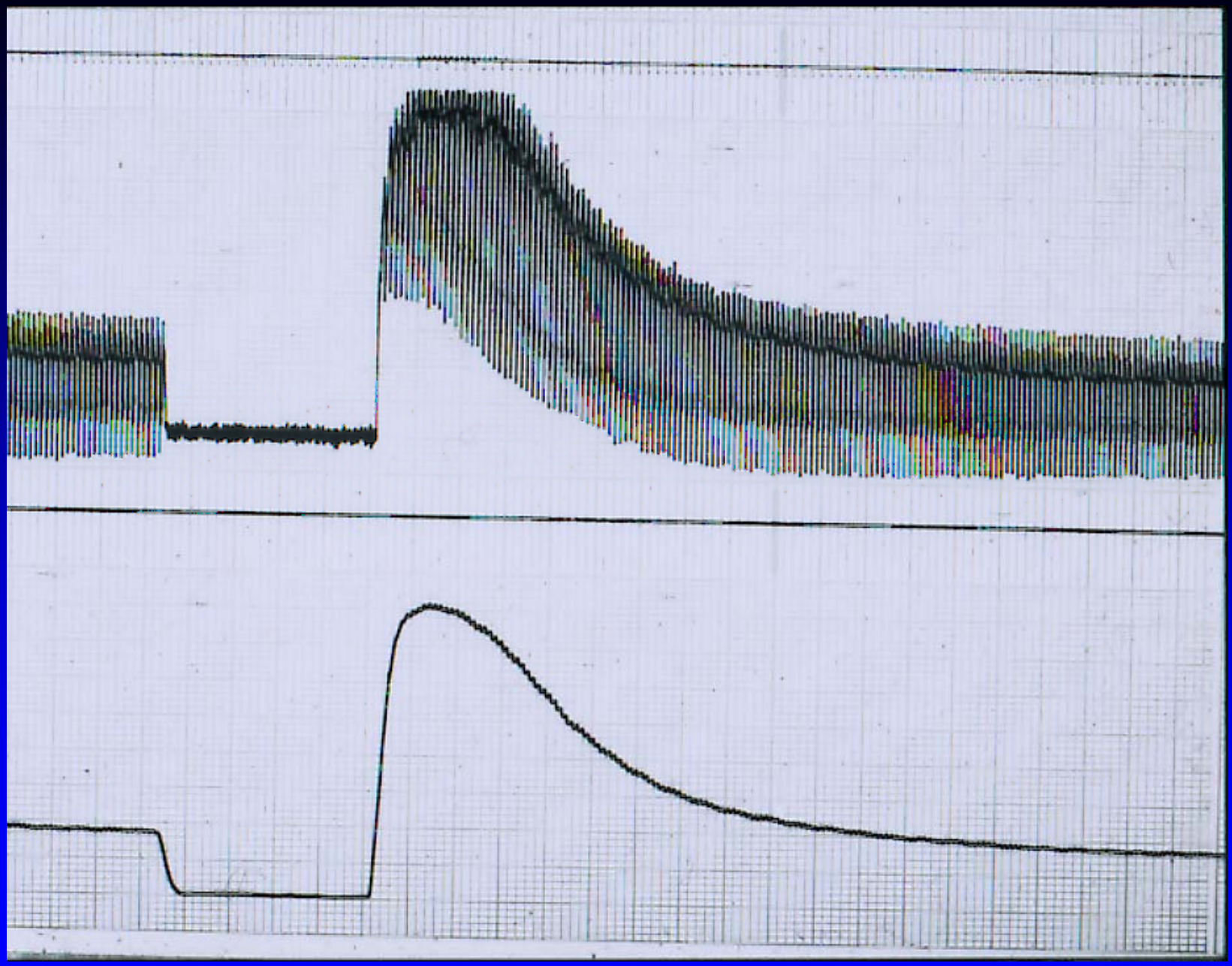
$$= \frac{\text{Hyperemic Flow}}{\text{Resting Flow}}$$

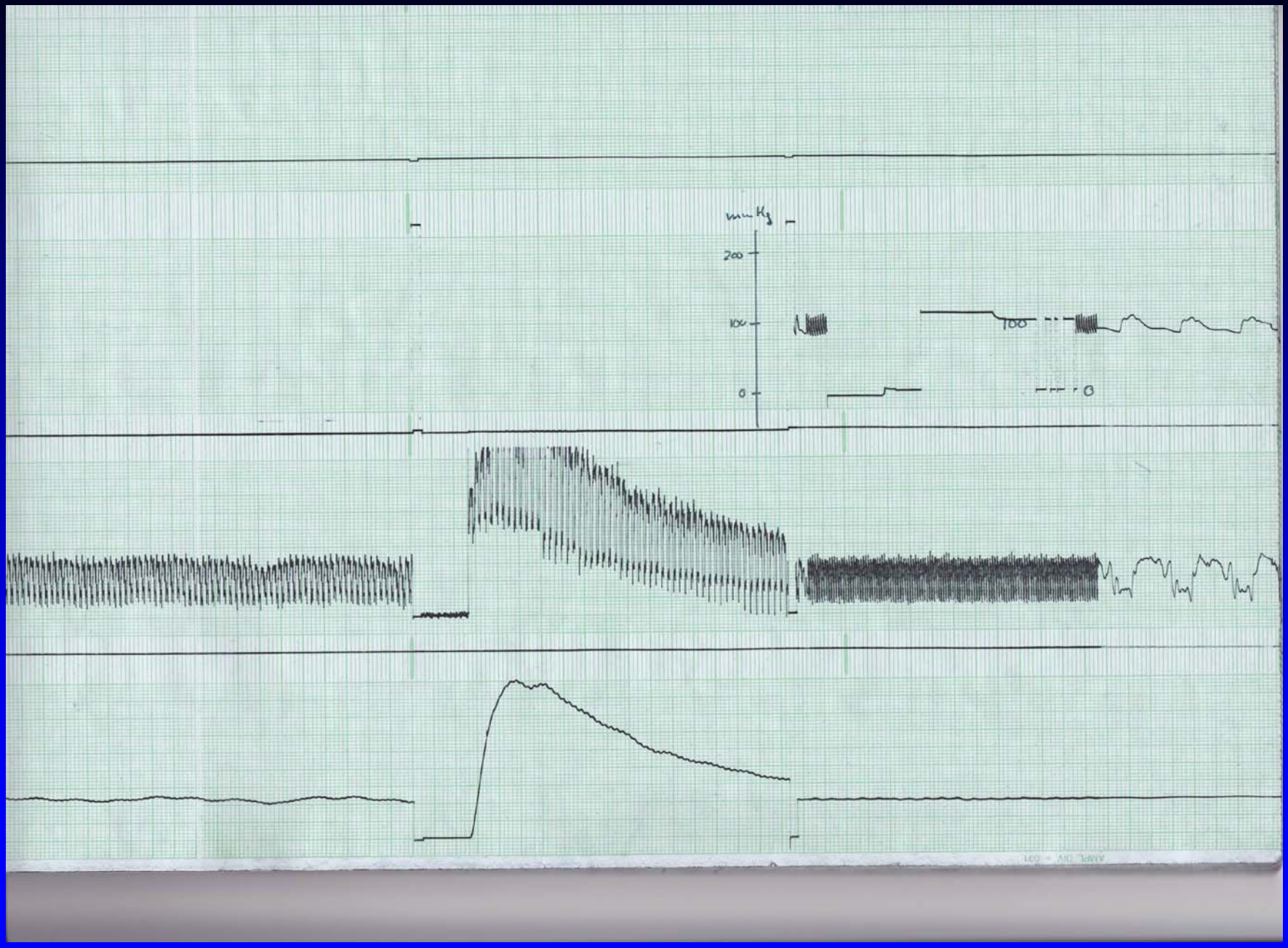

normaal
F
5F
R
1/5 R
reserve ≈ factor 5
RUST
MAX. VASODILATATIE


ernstige vernauwing
(90%)
F
R"
≈ 4/5 R
R - R"
≈ 1/5 R
F
R"
1/5 R
geen reserve meer over
RUST
MAX. VASODILATATIE


maximum flow
resting flow
Coronary Flow Reserve
5
4
3
2
1
0
20
40
60
80
100
Per Cent Diameter Narrowing






mm Hg
200
100
0
100
0

FLUID DYNAMIC EQUATION:

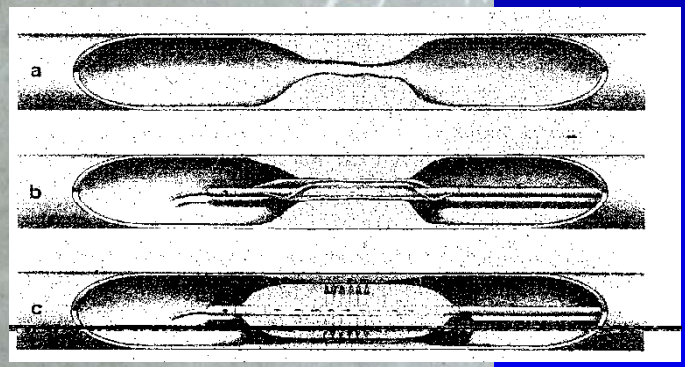
$$\Delta P = f.Q + s.Q^2$$

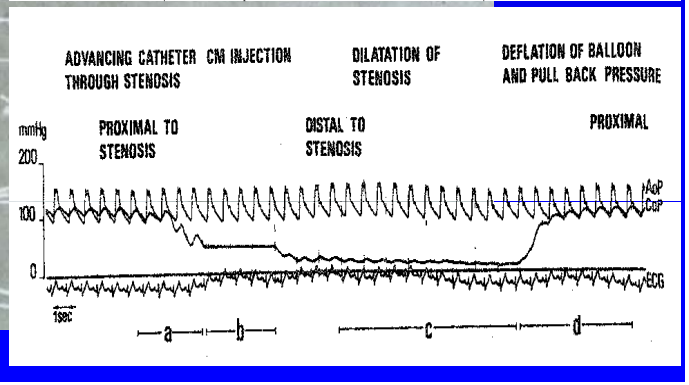
Hier tekening afmaken

→ Resting gradient cannot predict hyperemic gradient becaus
Friction coefficient (f) and separation coefficient (s) are grea
Different from one stenosis to another

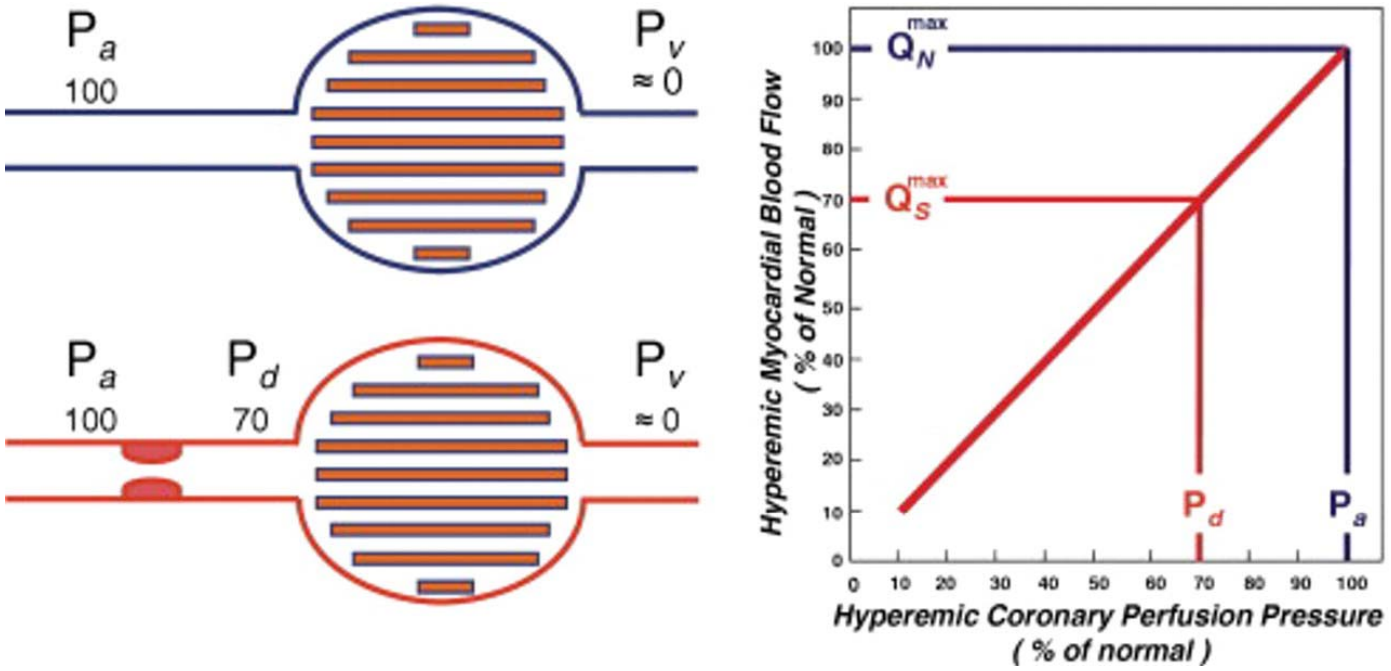
→ Functional assessment without hyperemia is a
Windtunnel without wind

“The resting gradient is not nearly enough but it’s all I have now”.



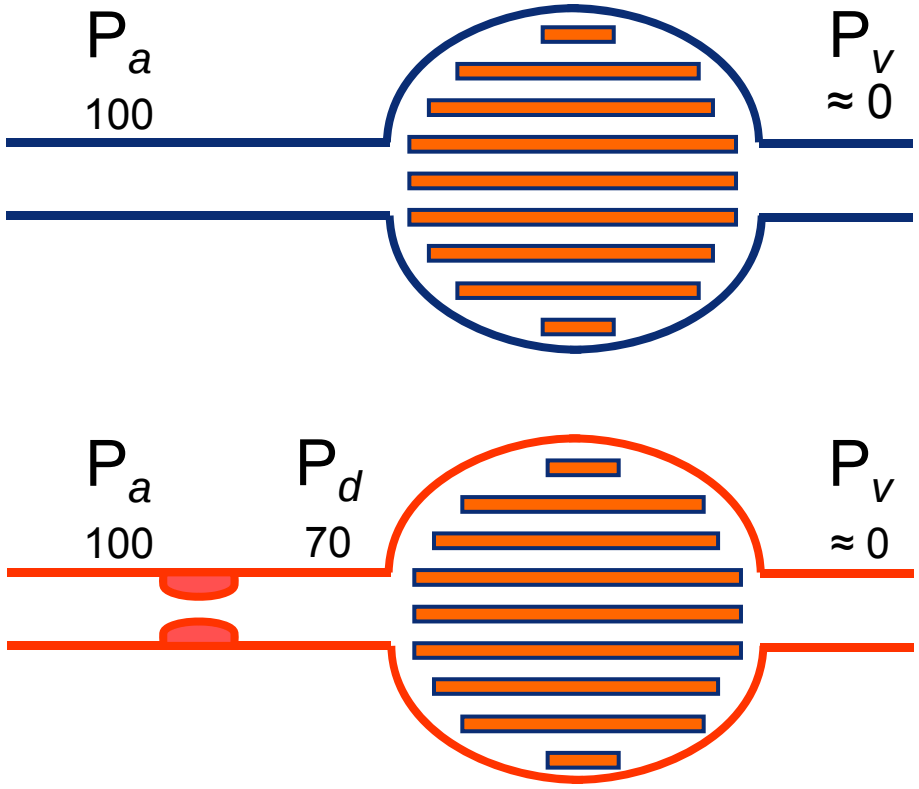


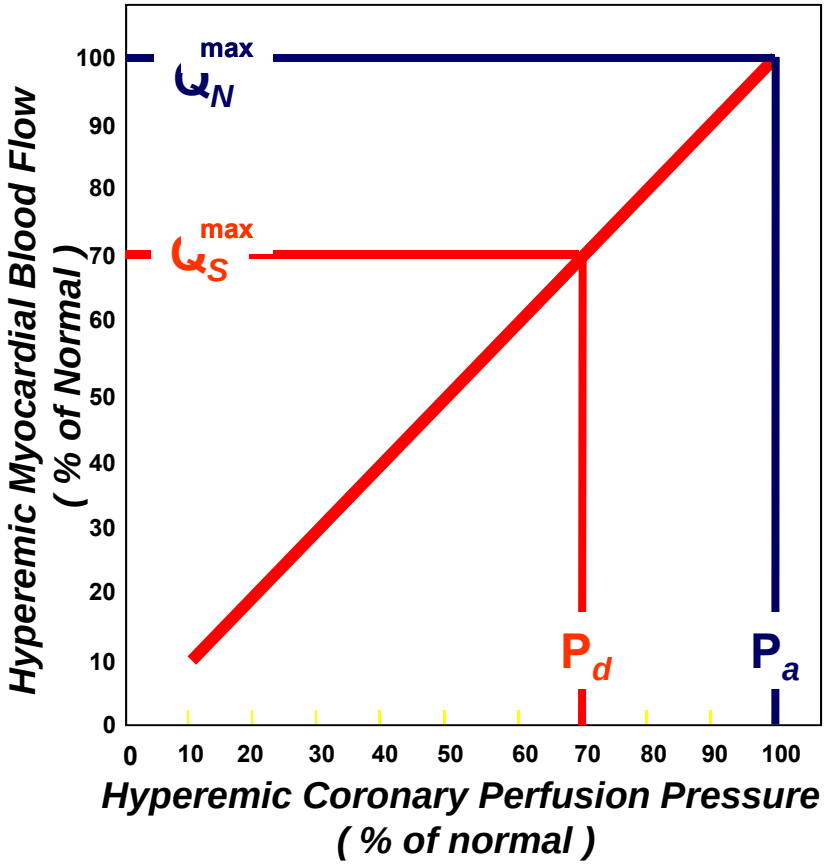
Concept of Fractional Flow Reserve Measurements



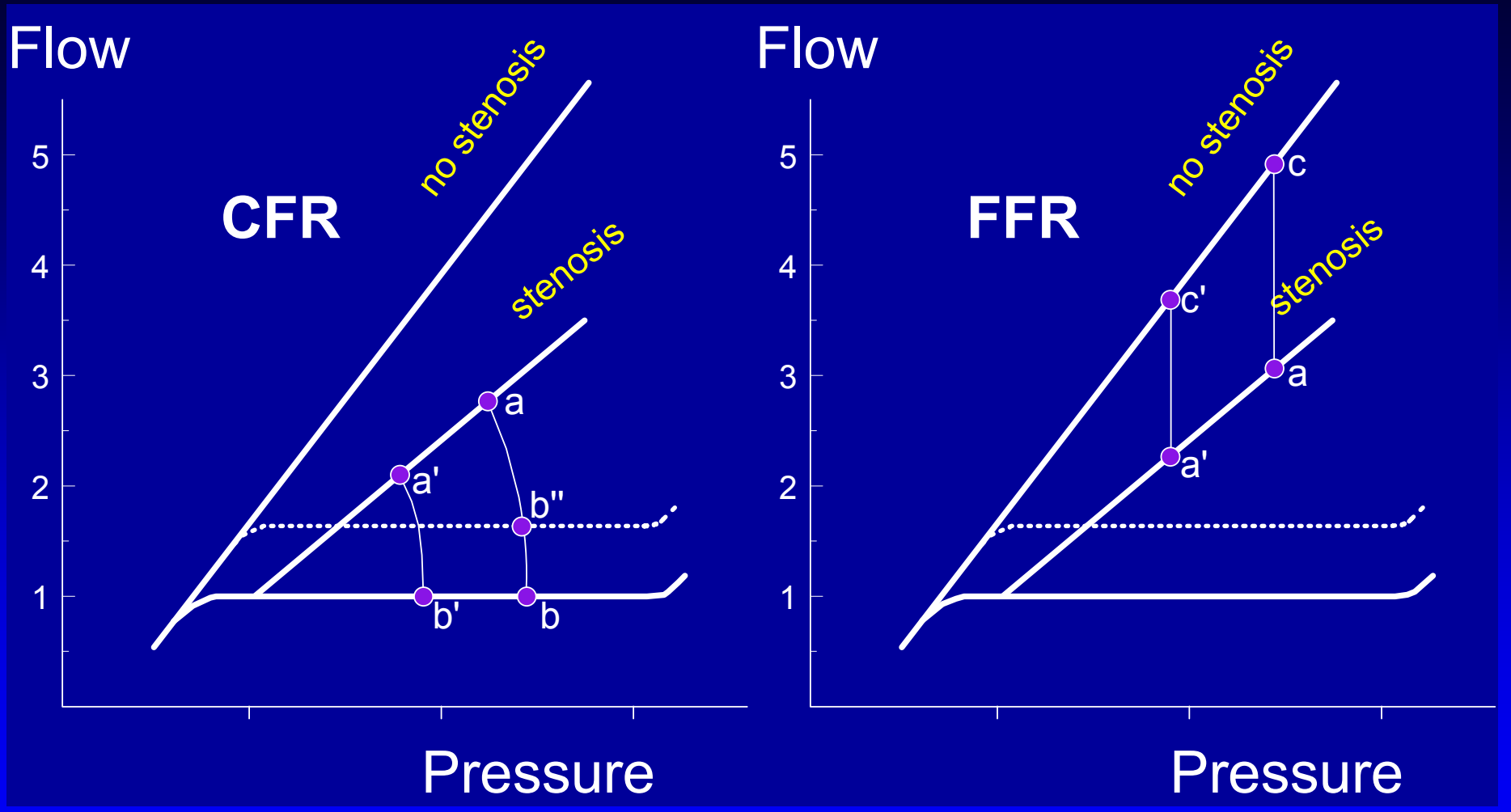
Pijls, N. H. J. et al. J Am Coll Cardiol 2012;59:1045-1057

The concept of Fractional Flow Reserve





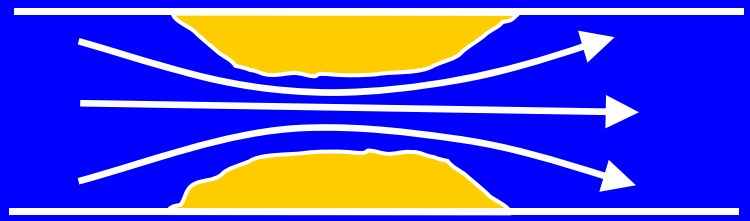
Pijls et al, JACC 2012;59:1045-1057



Deze dia er uit

$\Delta P = f.Q + s.Q^2$

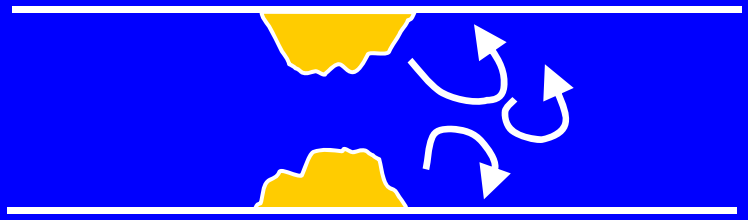
f = friction coefficient



Moderate gradient at rest

Moderate increment at hyperemia

s = separation coefficient



Small gradient at rest

Large gradient at hyperemia