

CORONARY ARTERY DISEASE AND CORONARY INTERVENTIONS

Aniff YEAROO

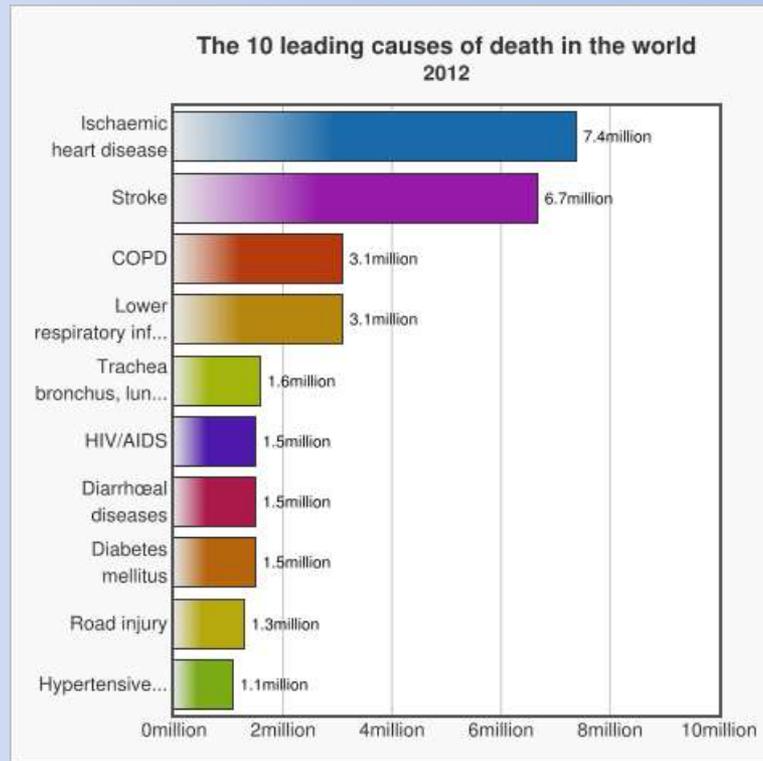
Interventional Cardiologist

Wednesday 29/01/2020

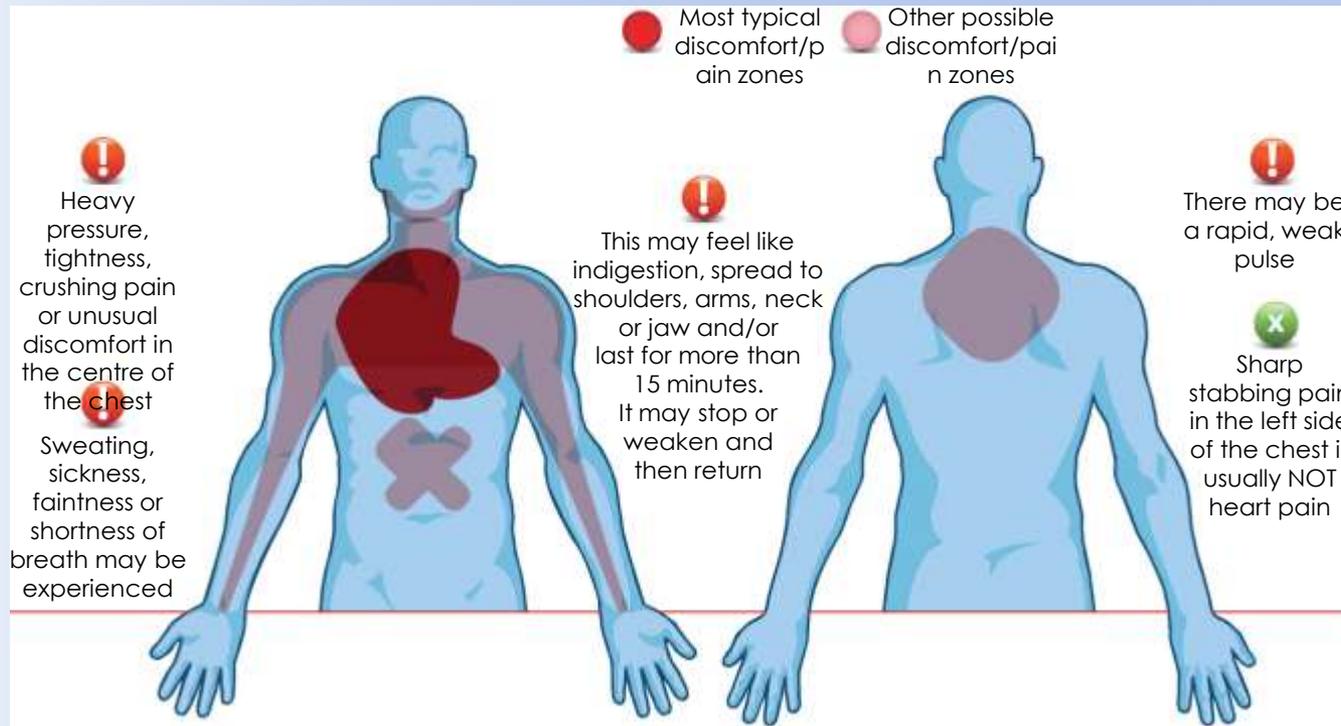
INTRODUCTION

- Ischemic Heart Disease (IHD) manifests as a spectrum of presentations ranging from asymptomatic states, chronic stable IHD, Acute Coronary Syndrome and Sudden death.
- Presentation: Exertional angina pectoris, atypical chest pain, dyspnea, fatigue, effort intolerance or Acute Chest Pain suggestive of ACS.
- Abnormal ECG findings, CT Scan or Exercise ECG.

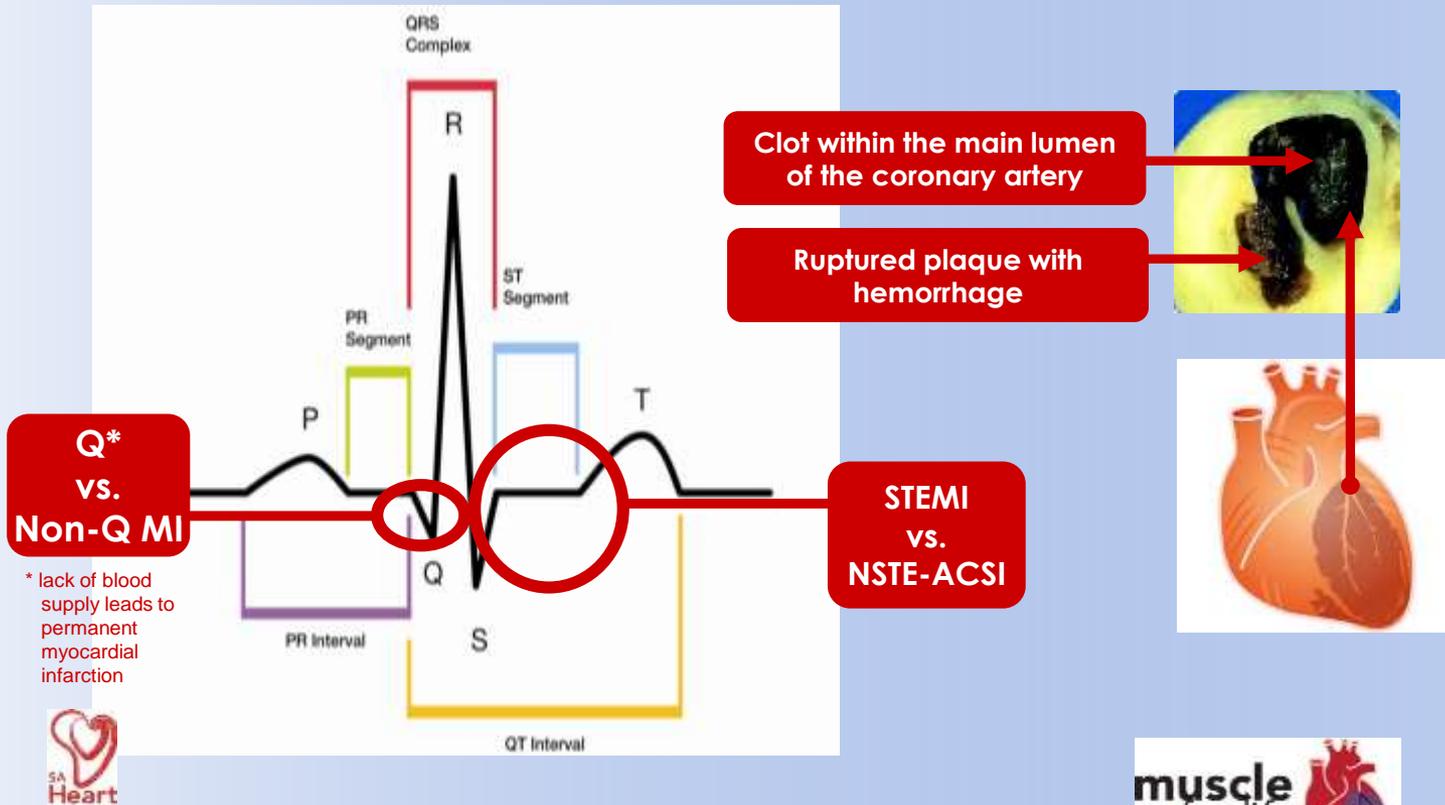
IHD leading cause of death globally



Symptoms of Ischemic Heart Disease

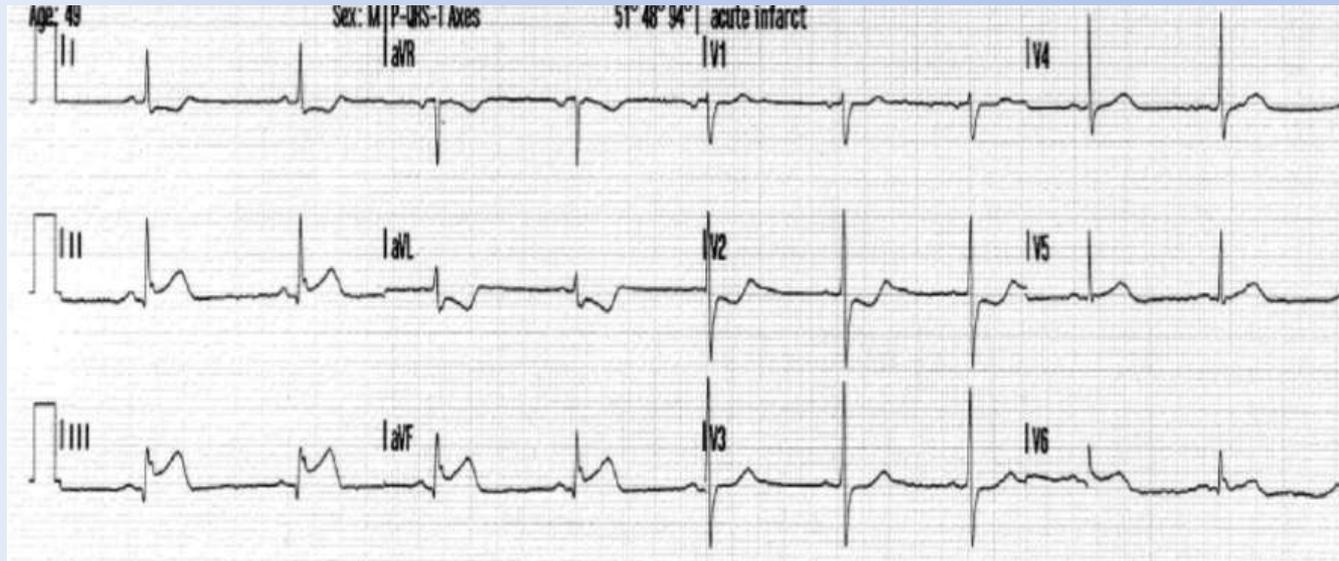


ECG Diagnosis



Adapted from J Davies (pathological specimen)

ECG

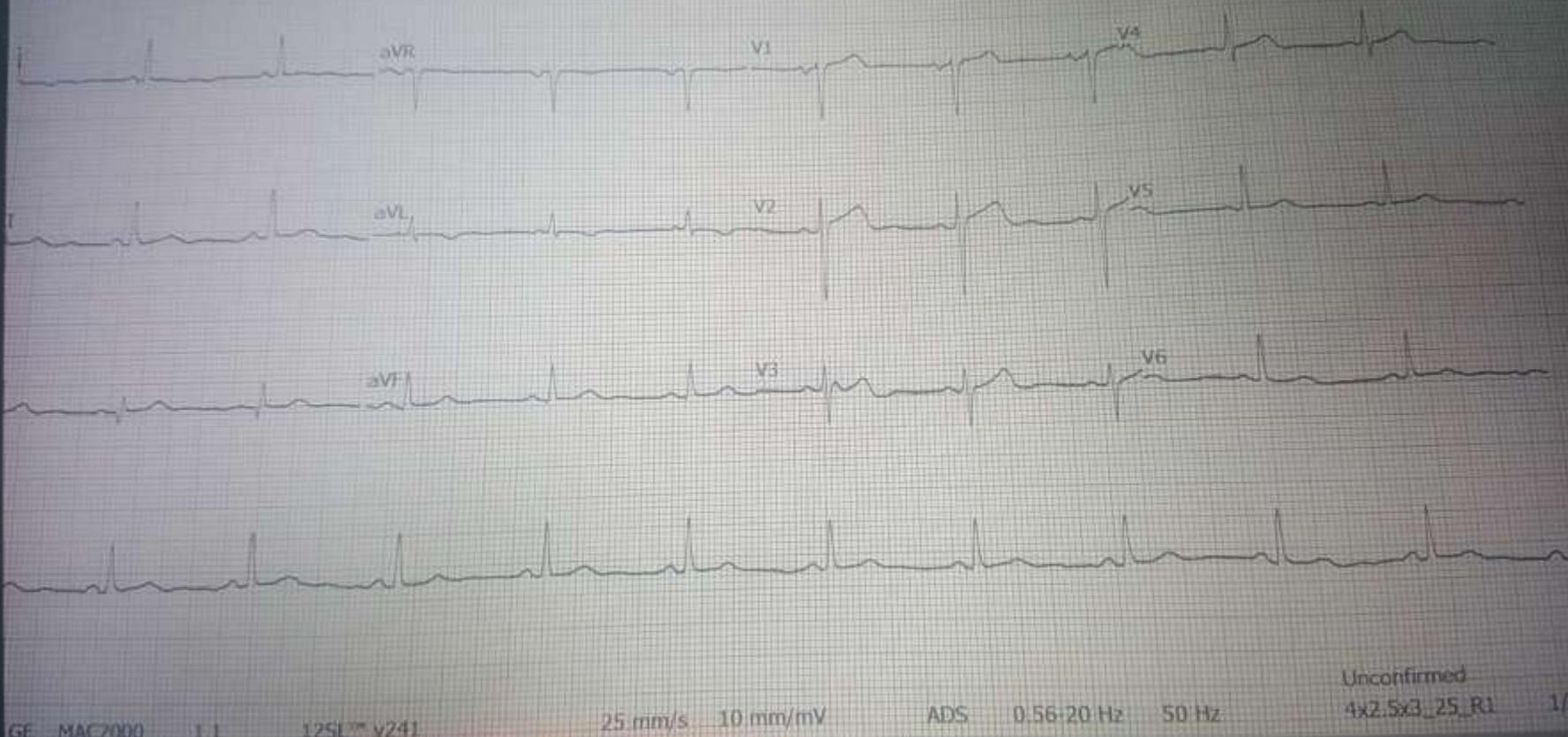


Test	Utility	Indication
Blood exams	Control disease progression and ischemia triggers	Every patient, every year
ECG	Discover ischemic signs and prognosis	Every patient, every year
Echocardiography	Exclude other cardiopathy and evaluate ejection fraction	Every patient
Stress test	Diagnosis and stratification	Diagnosis and follow-up of high risk patients
Angiography	Revascularization or anatomy investigation	Uncontrolled symptoms or possible complex lesions or high risk patients

Main tests in stable Ischemic Heart Disease

Non-invasive test	High-risk outcome
Exercise Treadmill	>2 mm of ST depression at low workload
	Exercise-induced ST elevation
	Exercise-induced ventricular tachycardia/fibrillation
	Failure to increase blood pressure >120 mmHg or sustained decrease >10 mmHg during exercise
Myocardial perfusion imaging	Resting perfusion abnormalities >10% of the myocardium
	Stress-induced perfusion abnormalities >10% of the myocardium or indicating multiple coronary obstruction
	Severe stress-induced left ventricular dysfunction
Stress echocardiography	Inducible kinetic abnormalities involving >2 coronary beds
	Kinetic abnormalities developing at low dose of dobutamine
Coronary computed tomographic angiography	Multi-vessel or left main stenosis

Stratification of risk outcome



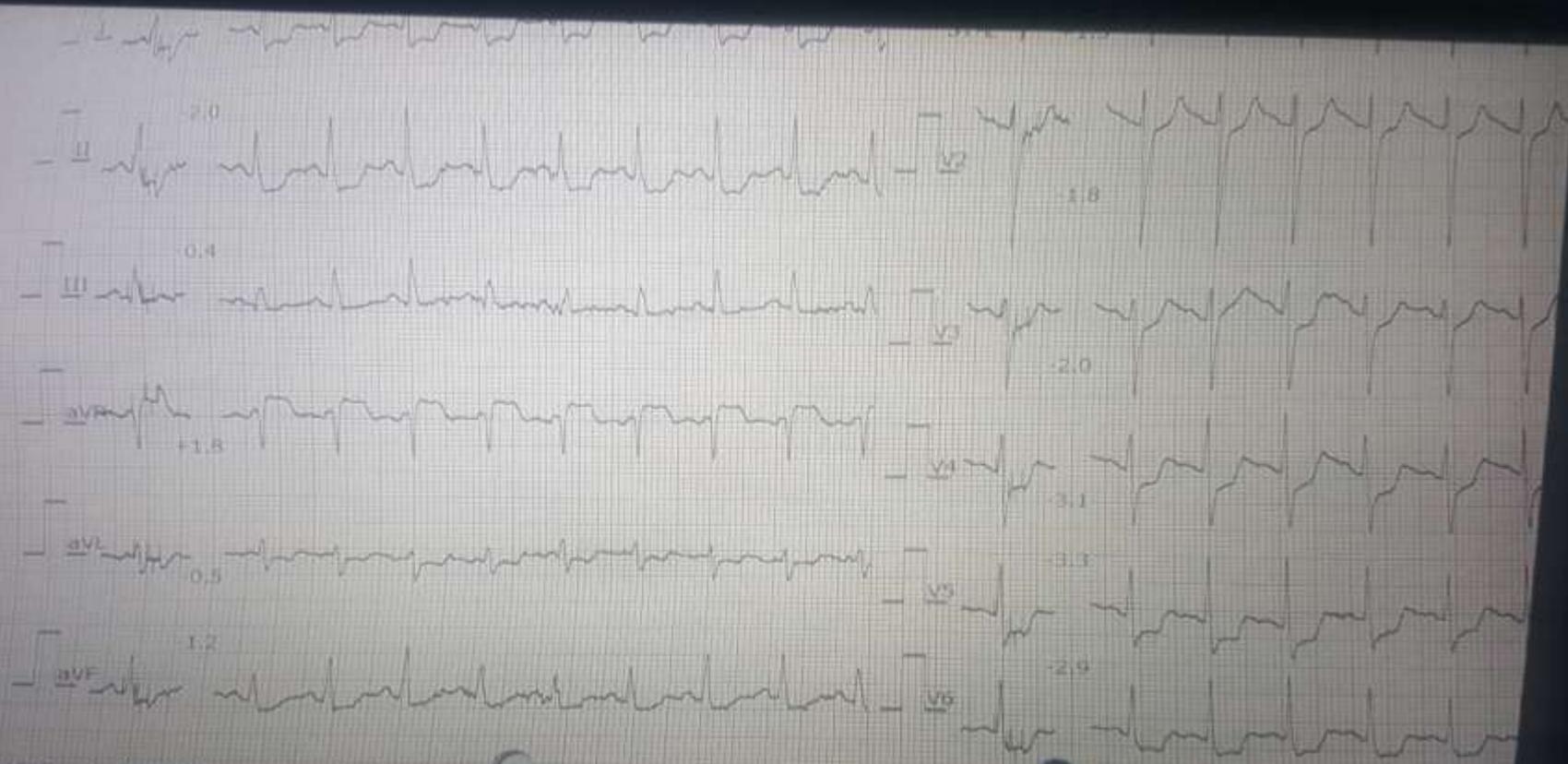
GE MAC2000 1.1 12SL™ v241

25 mm/s 10 mm/mV

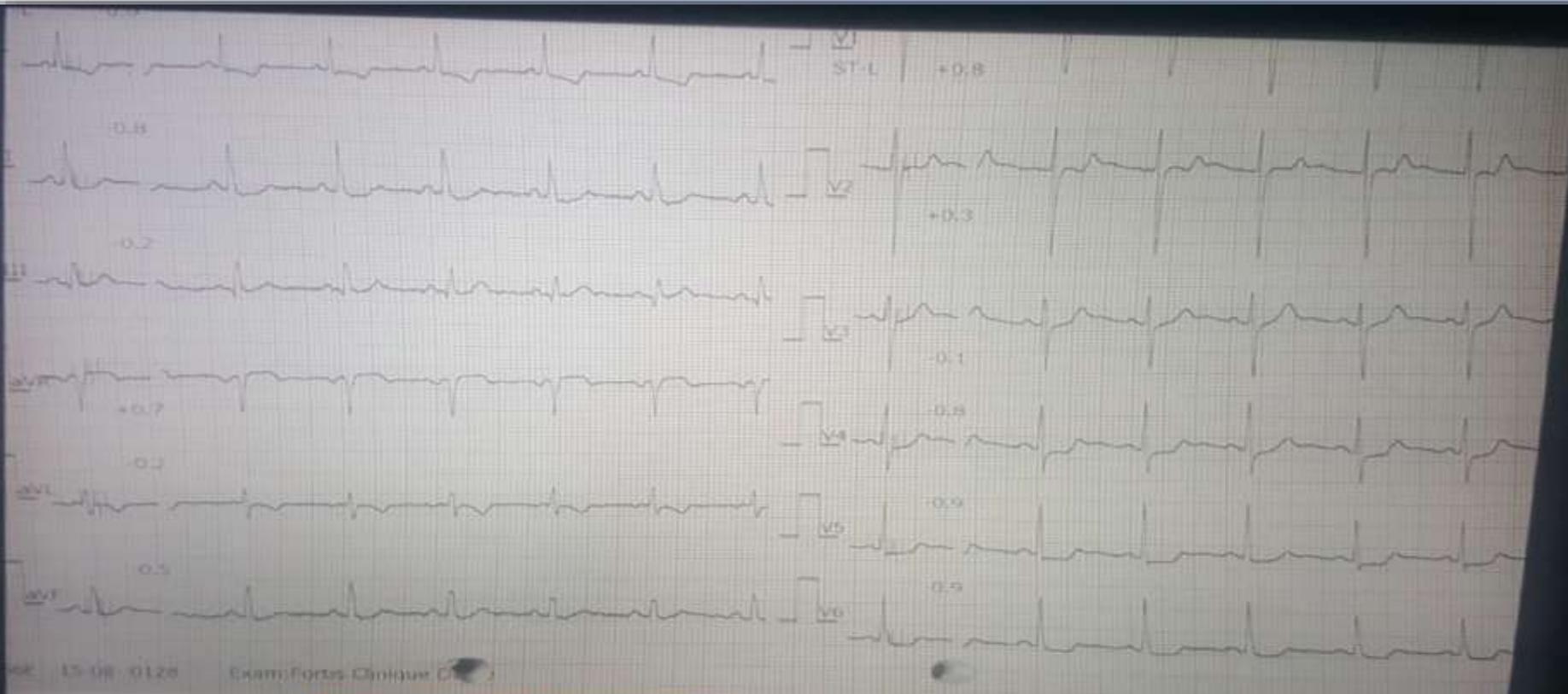
ADS 0.56-20 Hz 50 Hz

Unconfirmed
4x2.5x3_25_R1 1/1

ECG REST



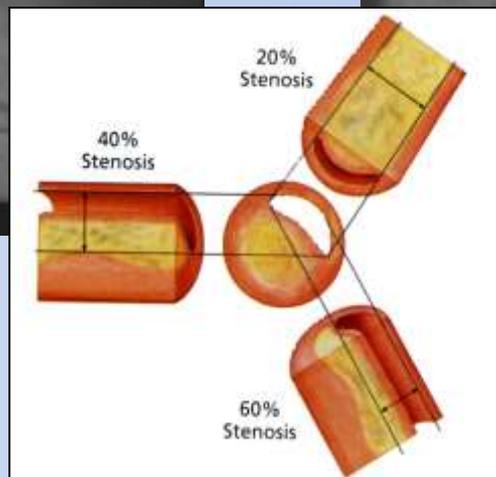
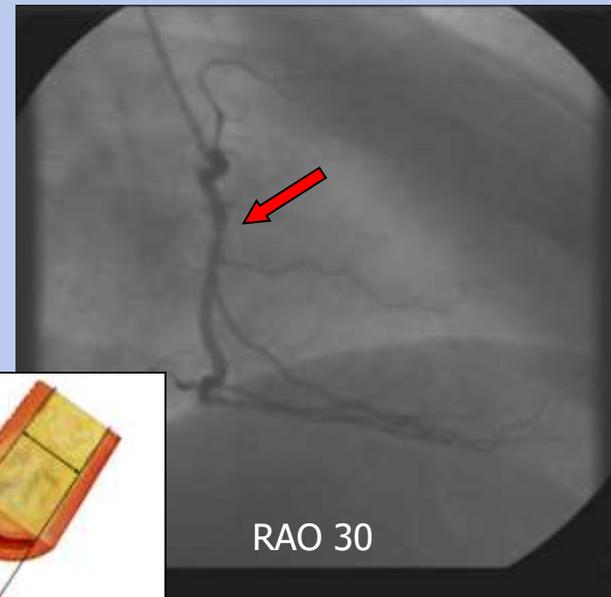
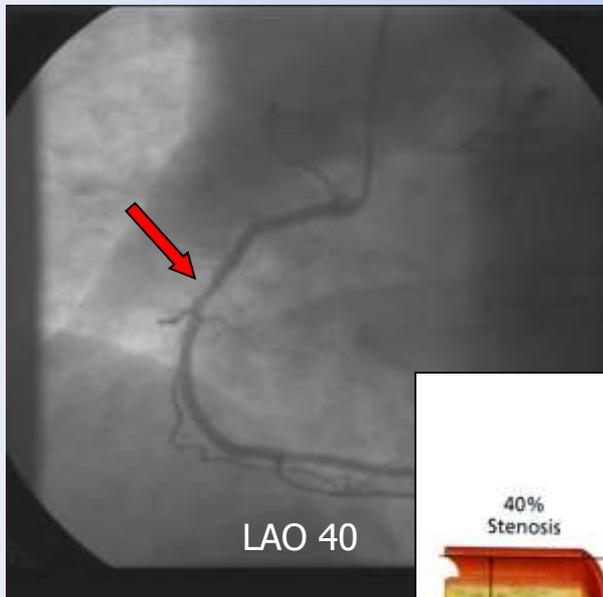
156E 15-08 0128 Exam: Fortis Clinique 



15-08-0128 Exam: Fortis Clinique

POST-EXERCISE ECG

RCA Angiogram



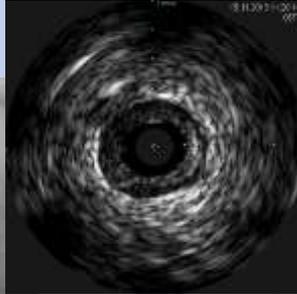
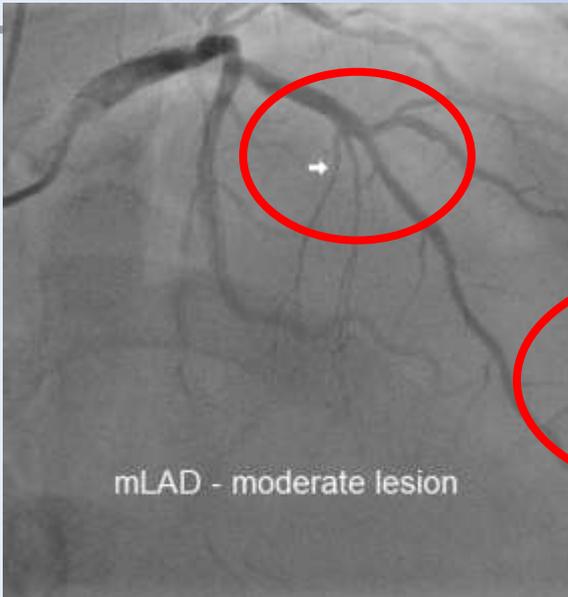
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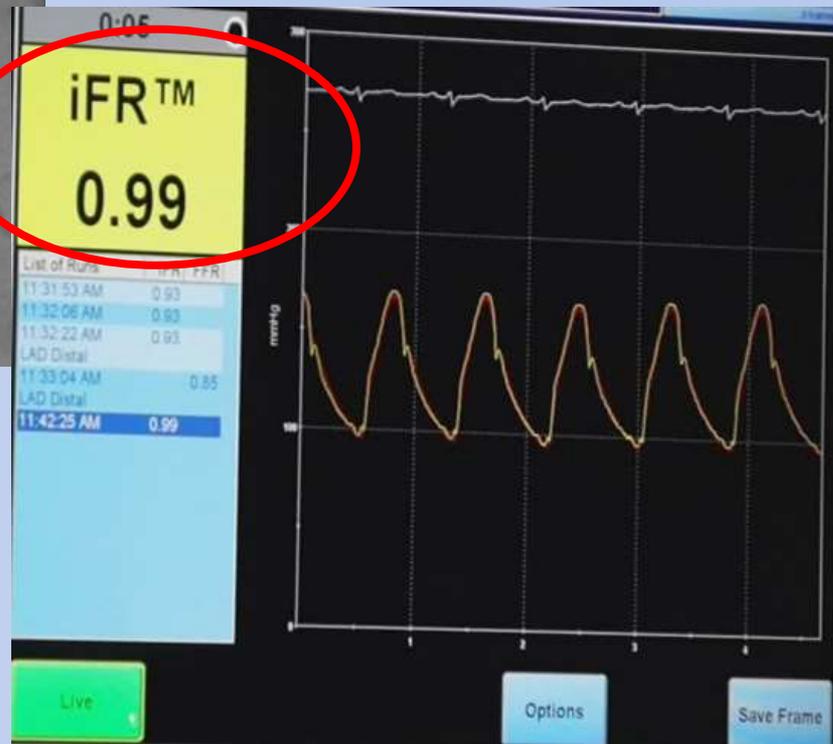
How can we access the lesion?

1. QCA
2. Physiology
3. Imaging (IVUS or OCT)
 - Lumen Diameter and Lumen Area
 - Vessel Diameter and Vessel Area
 - Lesion length



i-FR (Physiology)

Min CSA 3.5 mm sq

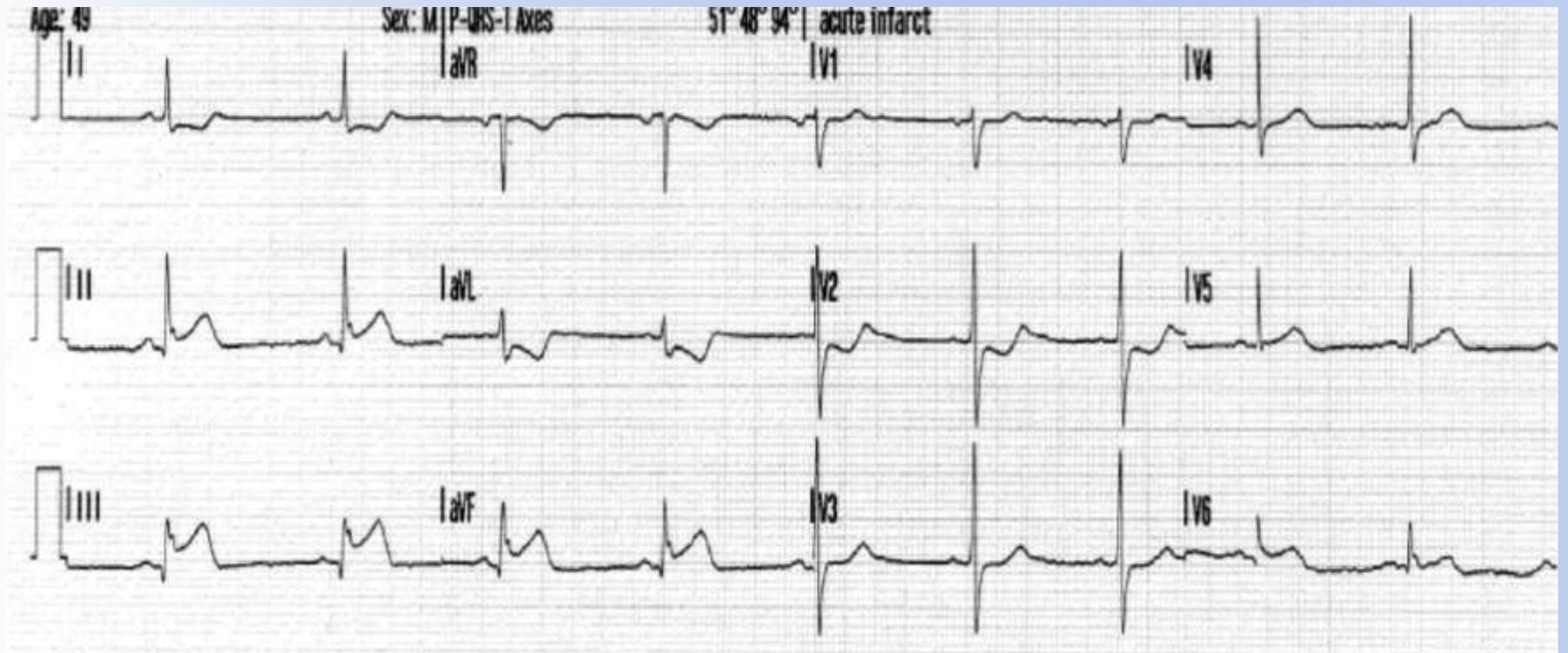


**NO MAXIMAL
HYPEREMIA**

ACS-ST Elevation Myocardial Infarction

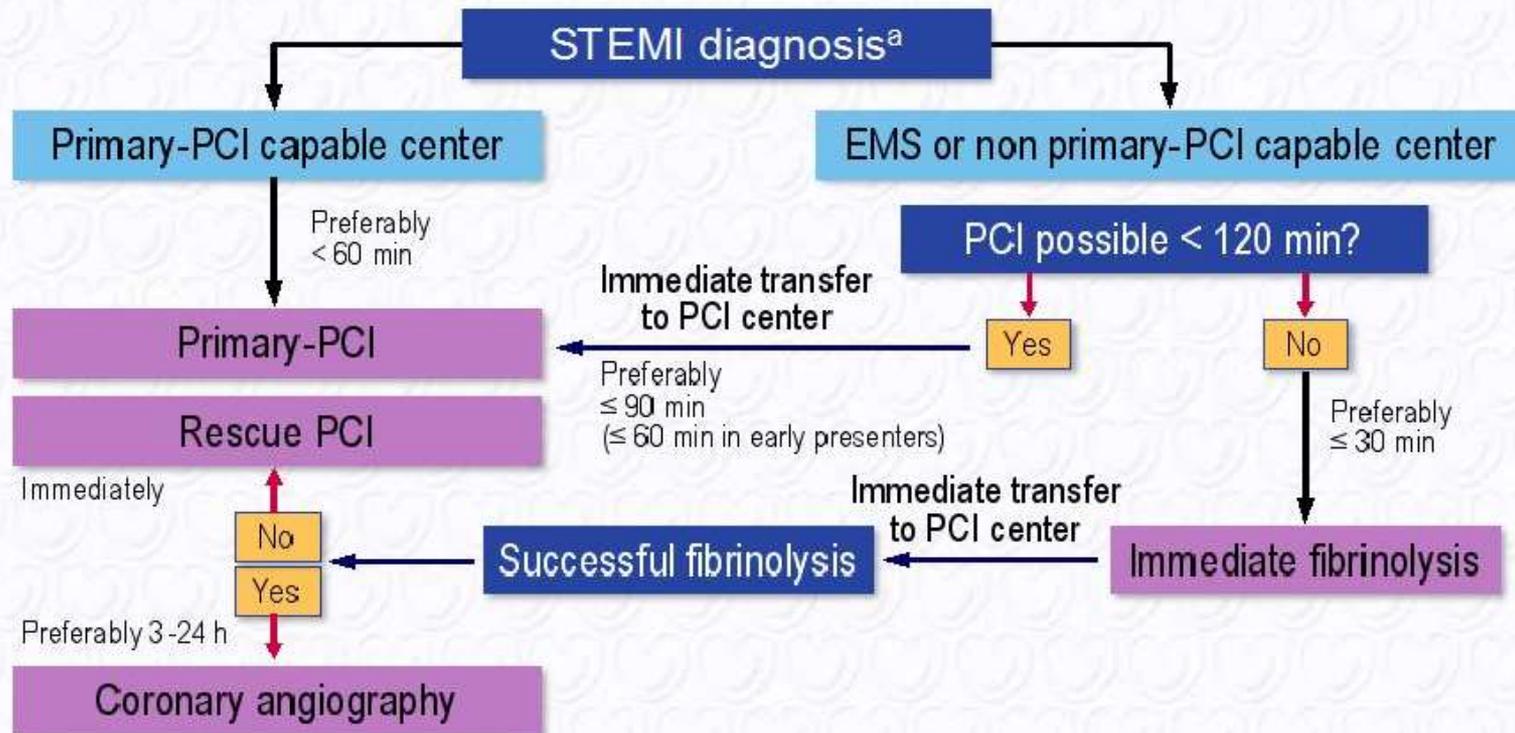
- Percutaneous Coronary Intervention
- Fibrinolysis

STEMI ECG



When is thrombolysis then
an option?

Prehospital and in-hospital management and reperfusion strategies within 24 h of FMC



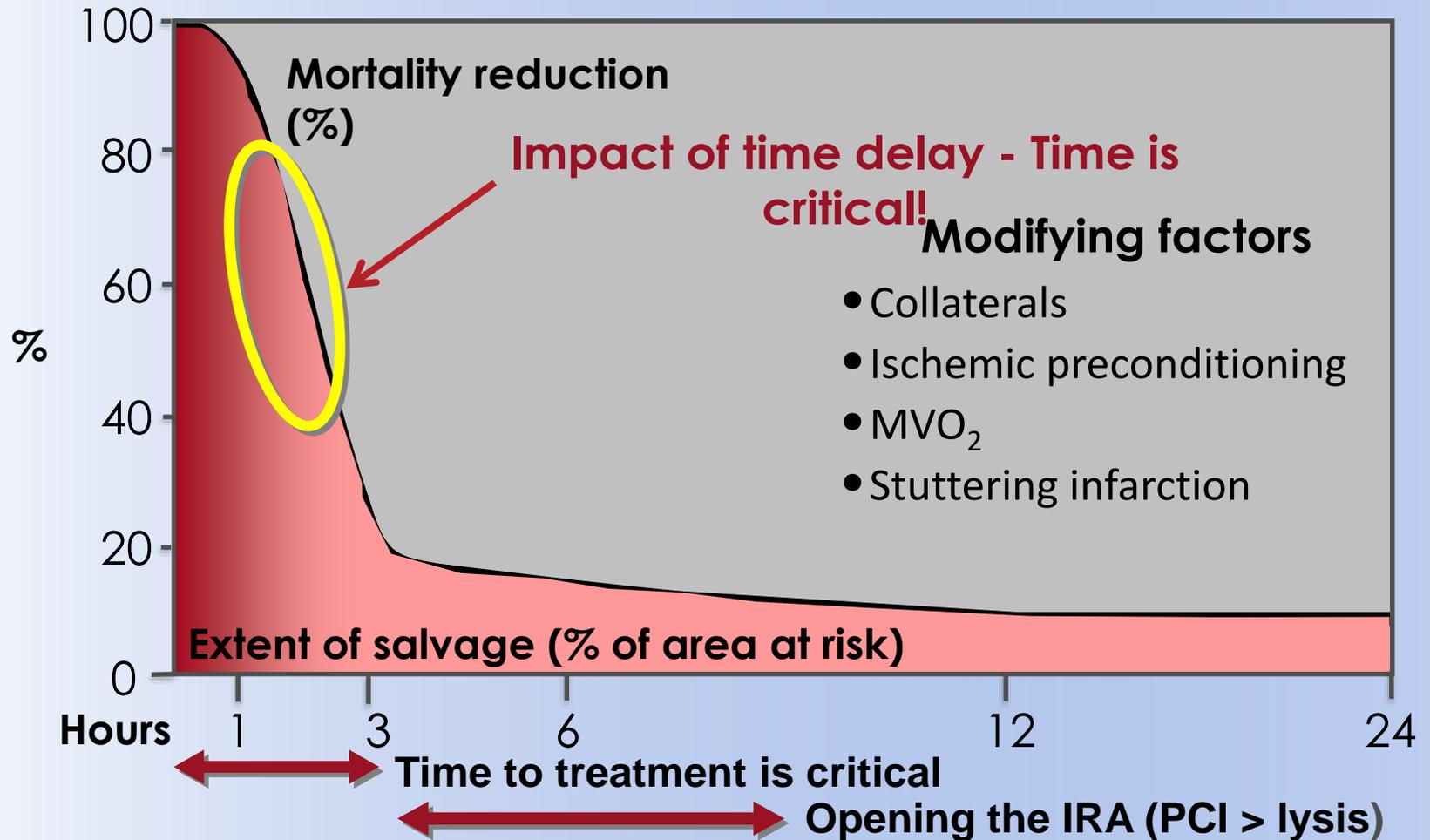
^a The time point the diagnosis is confirmed with patient history and ECG ideally within 10 min from the first medical contact (FMC). All delays are related to FMC (first medical contact).

Cath = catheterization laboratory; EMS = emergency medical system; FMC = first medical contact; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

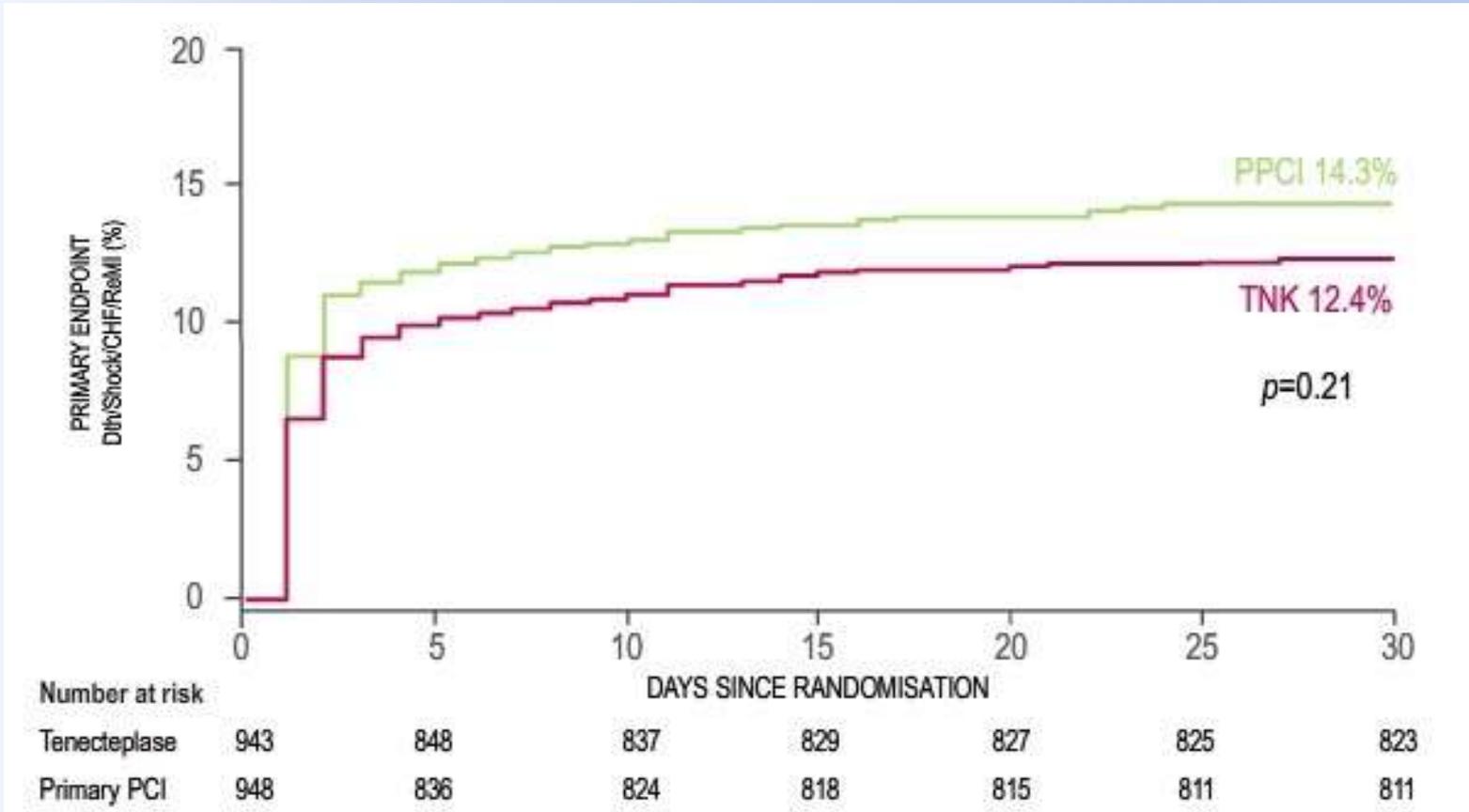
Choice of fibrinolytic agent

	Initial treatment	Specific contraindications
Streptokinase (SK)	1.5 million units over 30–60 min i.v.	Prior SK or anistreplase
Alteplase (tPA)	15 mg i.v. bolus 0.75 mg/kg over 30 min (up to 50 mg) then 0.5 mg/kg over 60 min i.v. (up to 35 mg)	
Retepase (r-PA)	10 units + 10 units i.v. bolus given 30 min apart	
Tenecteplase (TNK-tPA)	Single i.v. bolus: 30 mg if <60 kg 35 mg if 60 to <70 kg 40 mg if 70 to <80 kg 45 mg if 80 to <90 kg 50 mg if ≥90 kg	

Relationship Between Mortality Reduction and Extent of Salvage



Primary end point at 30 days



Mortality and morbidity the same between the two groups

Conclusions

- STEMI patients who presented within 3 hours of symptom onset:
 - No difference in outcomes irrespective of whether they received early fibrinolysis (and subsequent PCI) or primary PCI

A patient with STEMI with failed Fibrinolysis

- 50 years old man – 80kg
- Teacher with active lifestyle
- NIDDM 5yrs – Non smoker – Dyslipidemia
- No Past History of CAD
- Presented with persistent 2hours of chest pain – ECG – Anterior ST elevation

In Hospital Management

- Oral Aspirin
- Clopidogrel 600mg (Ticagrelor, Prasugrel)
- Esomeprazole 40mg IV
- Heparin IV
- Morphine (analgesic)
- Blood Tests sent
- Tenecteplase 40mg IV bolus in 10secs administered in I.C.U (patient initially refused P.C.I)
- Metoprolol and Atorvastatin

Immediate Follow up

- Persistent of chest pain and ST elevation
- No sign of heart failure – Bp 130/80 - P 80bpm
- Troponin mildly elevated
- Renal function normal
- TT Echocardiography – Antero septal Hypokinesia - LVEF 0.50
- SPAP 25mmhg
- Urgent Coronary Angiography

Coronary Angiography

- 4 hours after onset of pain
- Baseline Coronary Angio by Right Radial approach – LAD II 100%
- Insufficient support to cross the lesion - switch to femoral approach
- Lesion wired, pre-dilated, DES 3x28, high pressure inflation
- Flow TIMI3

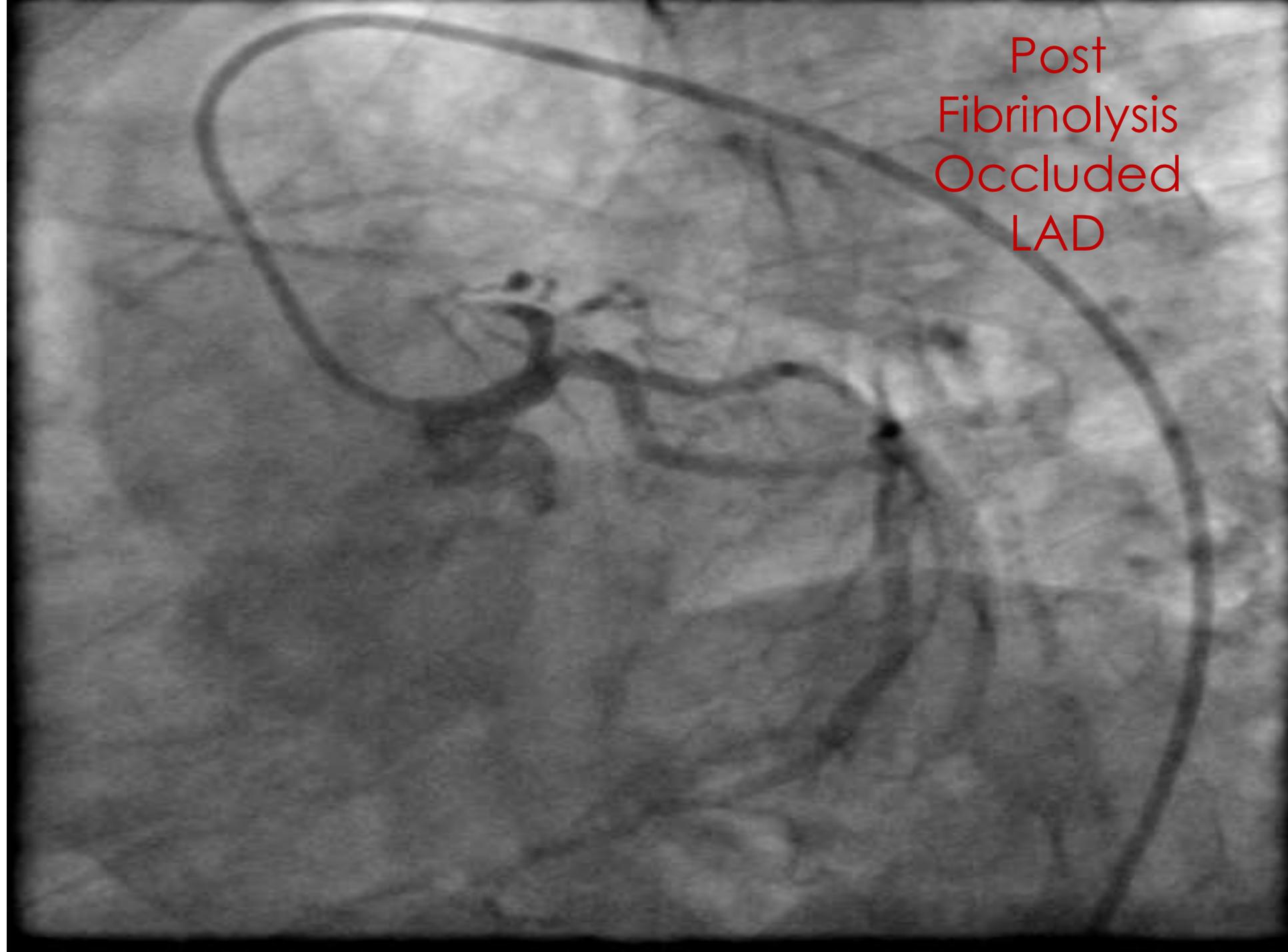
Post Procedure

- Relief of chest pain and ST elevation
- TT Echo – Anterior wall Hypokinesia – LVEF 0.45 – 0.50, SPAP 35mmhg. No pericardial effusion
- Heparin 48hours – Clopidogrel – ASA – Metoprolol – Ramipril - Atorvastatin
- Discharge Day 6 and Reviewed regularly – At twelve months was Asymptomatic – LVEF 0.5
- TMT 9 mins Stage 3 Bruce Protocol – Asymptomatic – Non significant ST Depression Anteriorly – 90% THR

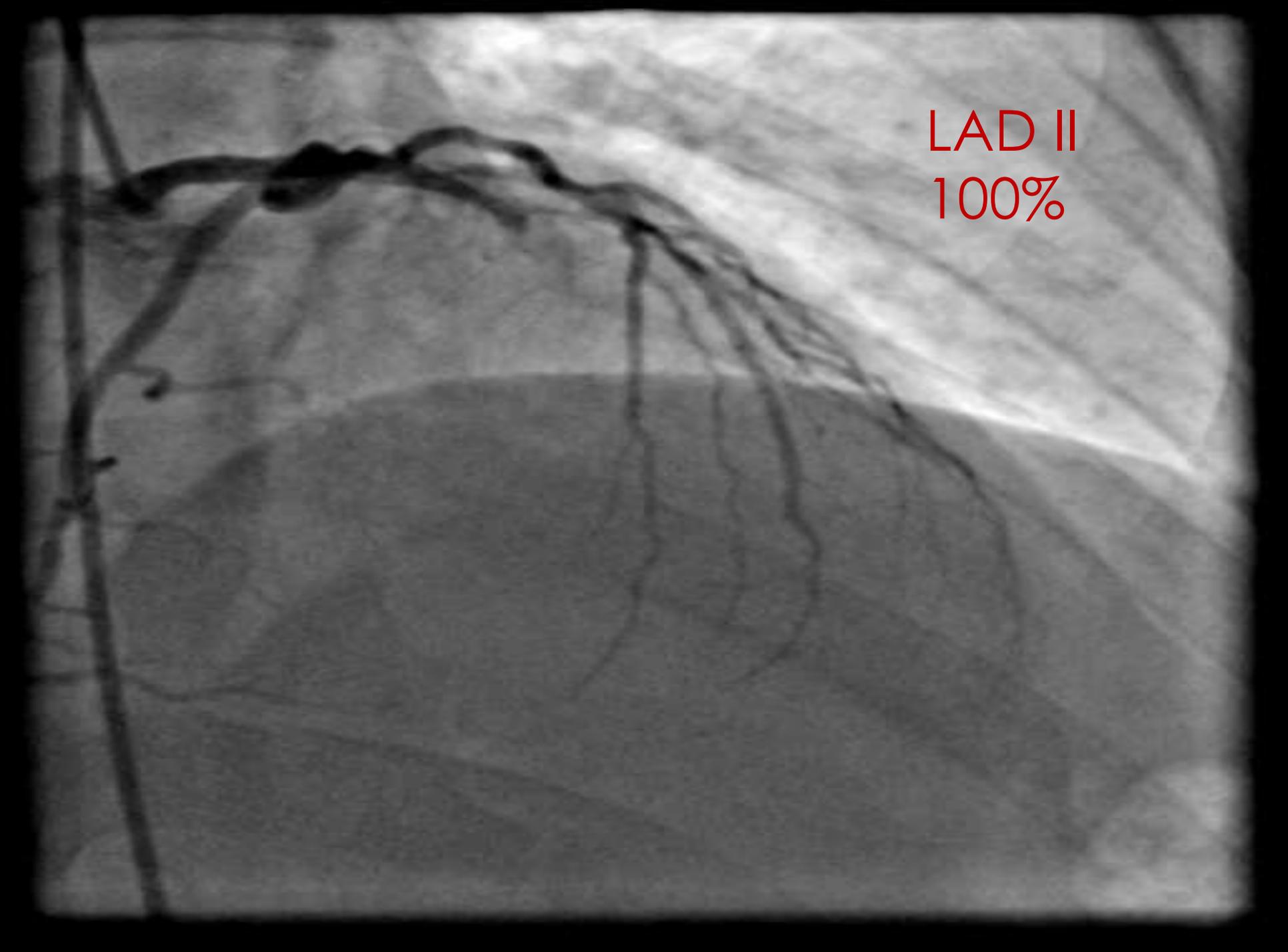
RCA



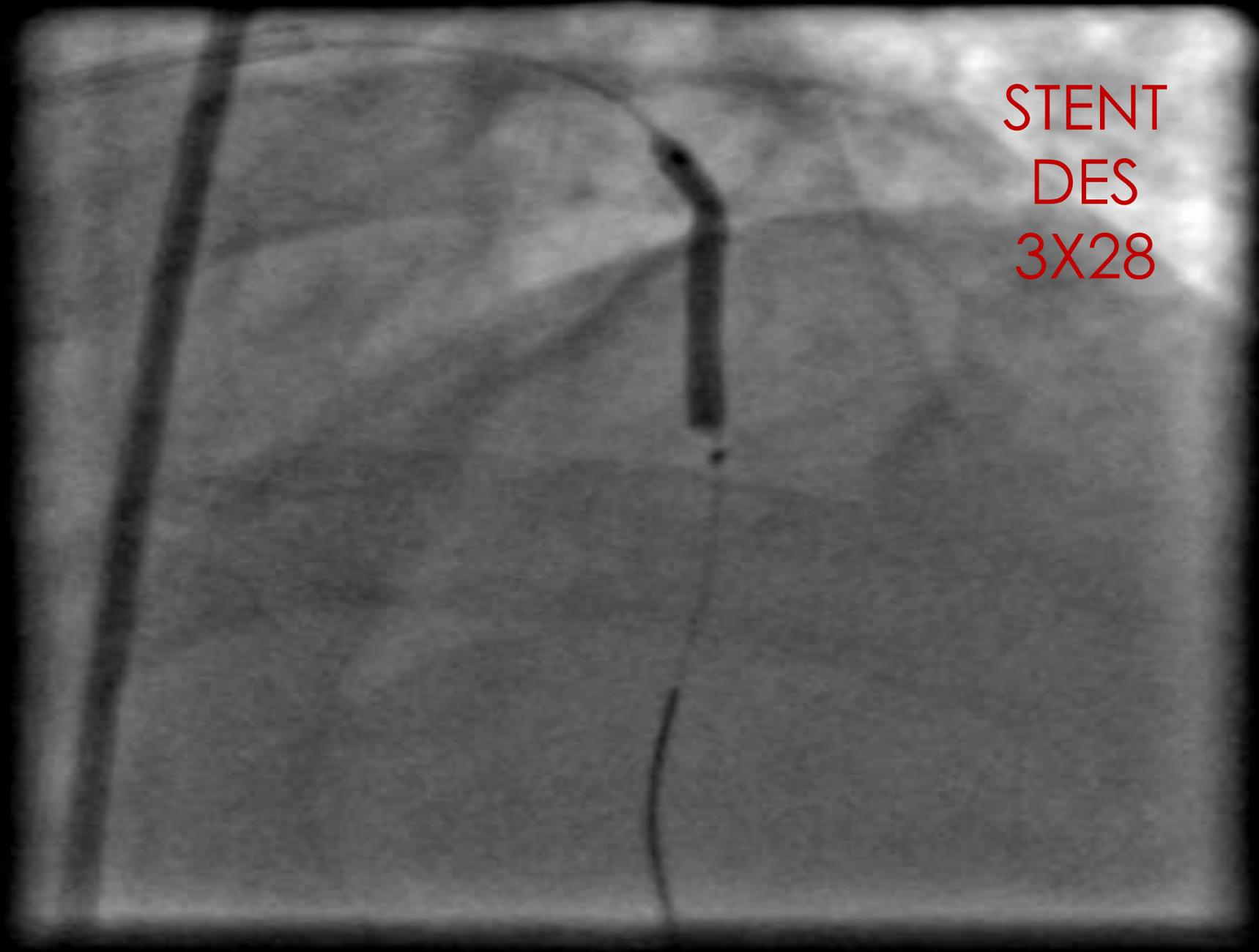
Post
Fibrinolysis
Occluded
LAD



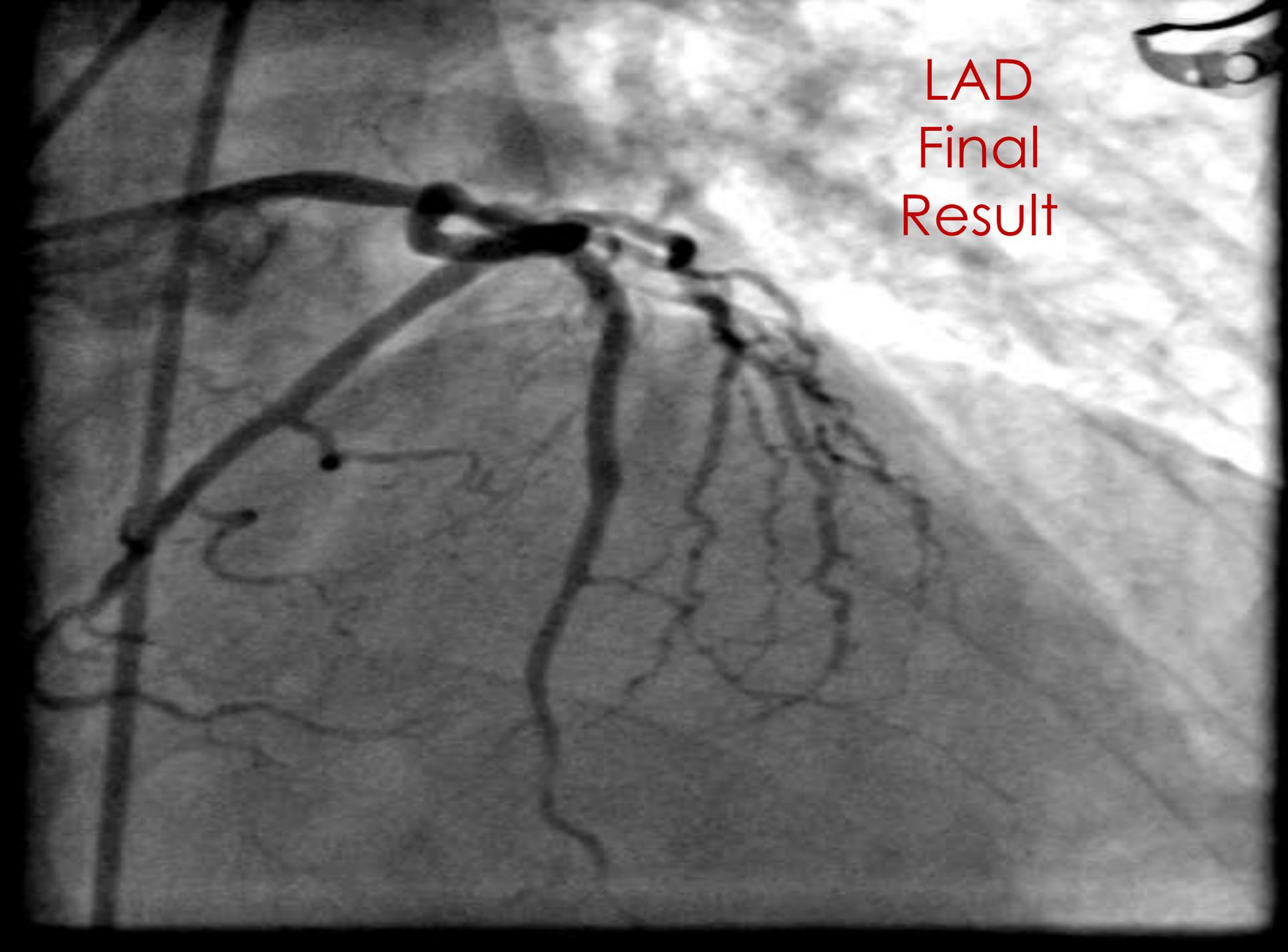
LAD II
100%



STENT
DES
3X28



LAD
Final
Result



Management of In Stent Re Stenosis

- POBA (Plain Balloon)
- Stent in Stent
- Drug coated Balloons

BALLOON NATURE:
SEMI COMPLIANT



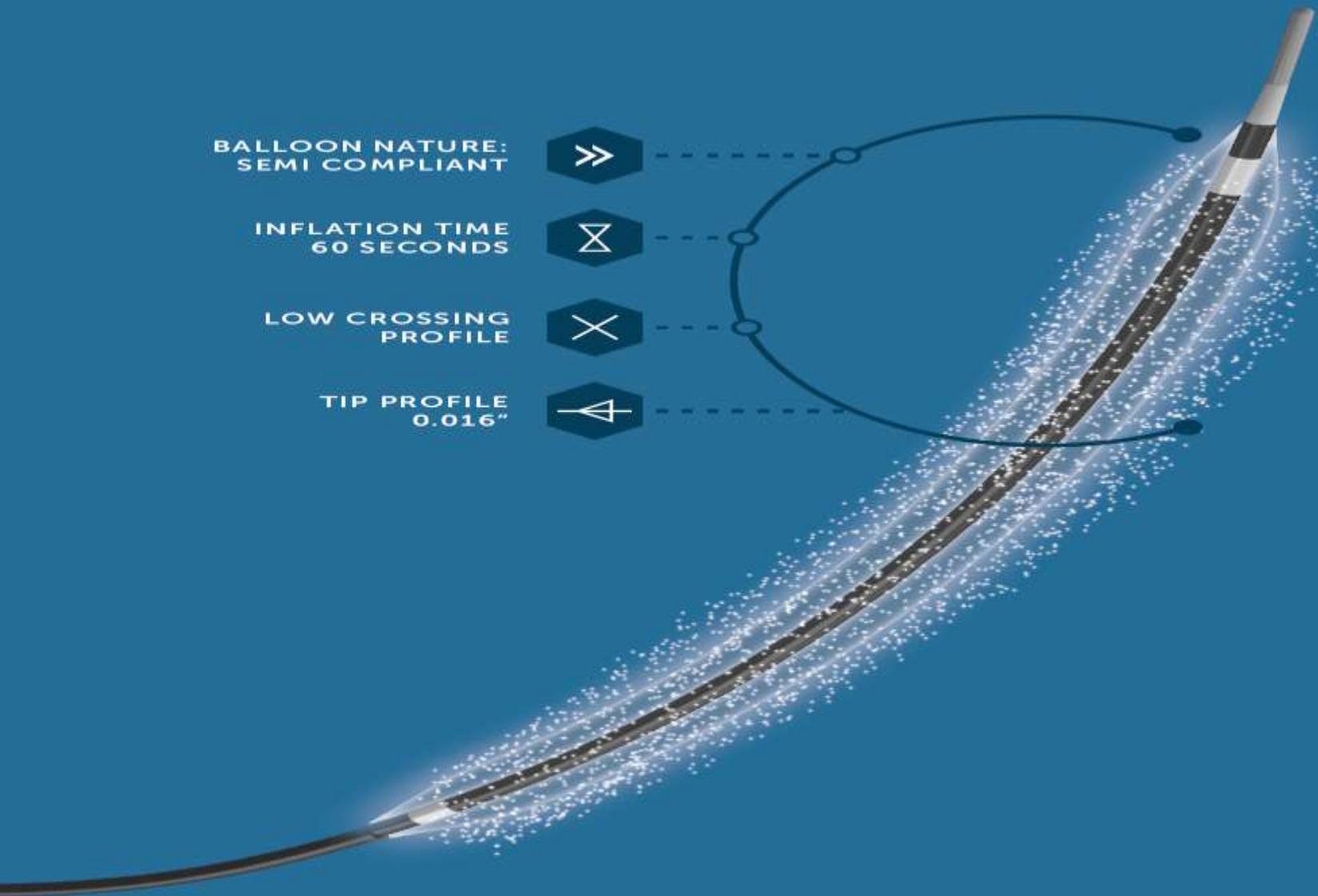
INFLATION TIME
60 SECONDS



LOW CROSSING
PROFILE



TIP PROFILE
0.016"



Drug Coated Balloon Catheter for Percutaneous Transluminal Coronary application.

Intended for applications like In-stent restenosis, small vessels, bifurcation lesions and De-Novo lesions.

Based on the concept of Nanolute technology, it provides more benefits than the existing balloons available in the market and has broad horizon in terms of treatment.

FOLDED BALLOON



HANDLE WITH CARE

Do not rub the coated balloon

Do not use any protective/insertion sheath to advance the SCB through the introducer sheath and/or haemostatic valve

LESION



PRE-DILATATION



SCB



PRE-DILATATION RECOMMENDED IN ALL CASES

For pre-dilatation in all cases use a standard balloon
(approx. 0.5mm smaller than RVD)

Choose a SCB with nominal size equal to
reference diameter.

Duration time 30-60 sec



DRUG RELEASE WITHIN 60 SECONDS

Longer inflation times are possible at discretion of operator - to pursue optimal mechanical dilatation - but do not lead to further drug release.

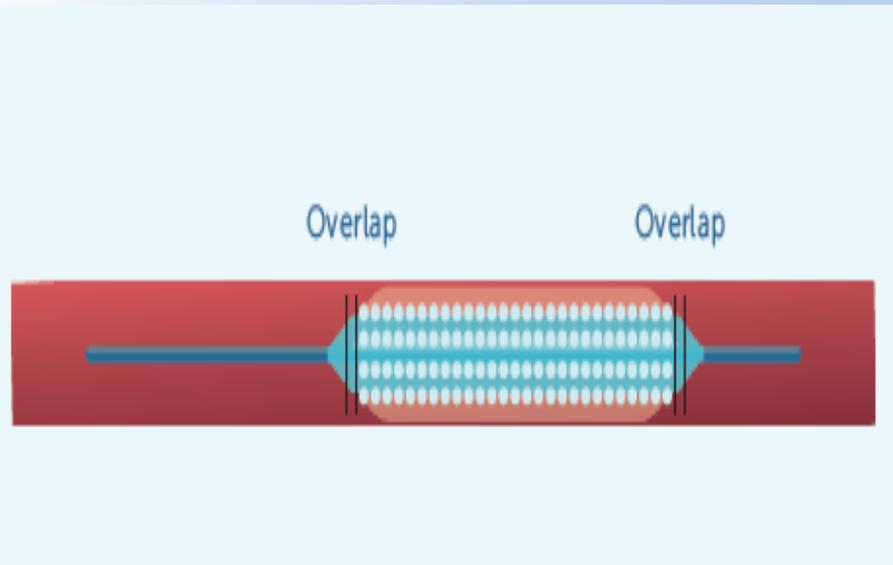
LESION 1, SCB 1



LESION 2, SCB 2

ONE DRUG-ELUTING BALLOON FOR EACH LESION

Each lesion should be addressed with a separate balloon.
In longer lesions SCB overlapping is indicated

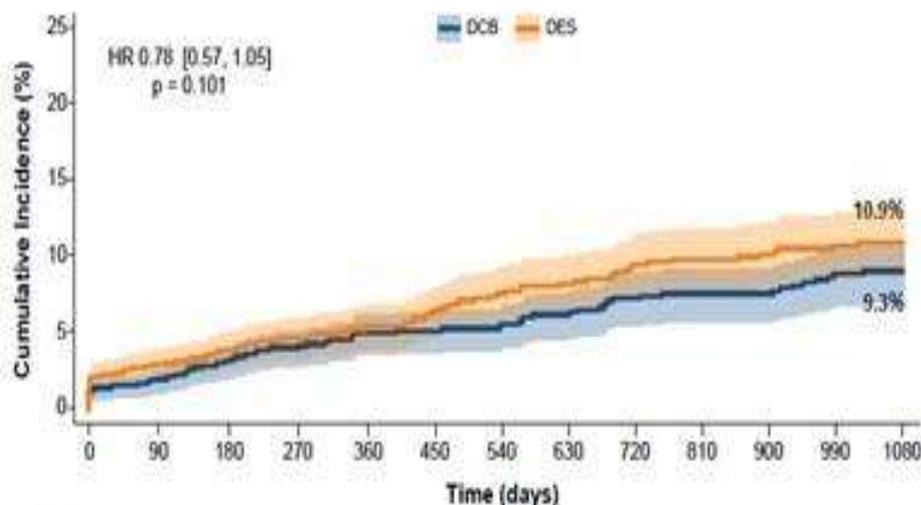


IF A STENT IS PRESENT, SIZE OF BALLOON SHOULD BE GREATER THAN STENT BY 0.5MM ON EACH SIDE.

1976 patients treated for coronary ISR with DCB (N=1033) versus DES (N=943)
 Safety (all death, MI, ST) at 3 years favors DCB

"One-Stage" Analysis

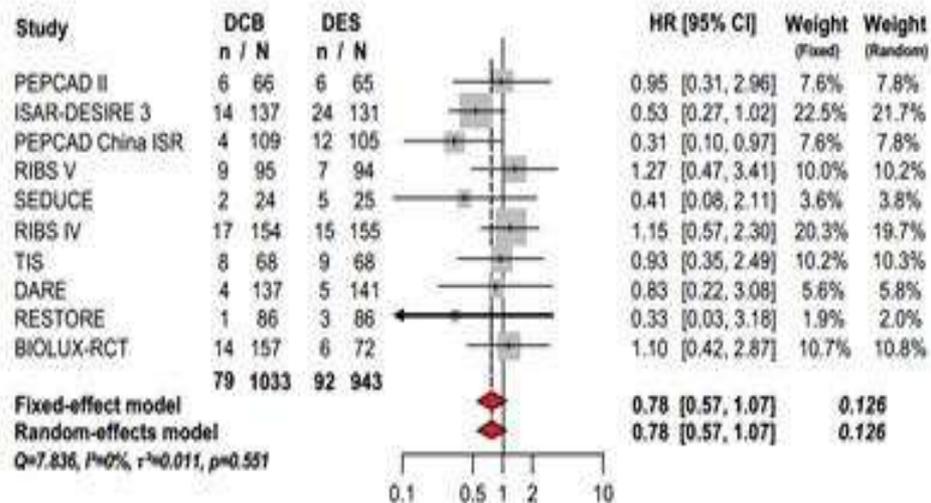
Cox Mixed-Effects Model*



1033	1000	987	969	940	752	691	612	606	575	541	504	464
943	908	894	882	852	666	622	584	578	552	522	480	443

"Two-Stage" Analysis

Fixed-Effect and Random-Effects Model*



All-Cause Death, Myocardial Infarction, Target Lesion Thrombosis

No mortality signal in coronary trials of DCB

In patients with recurrent ISR, implantation of new DES would result in a vessel with multiple metal layers ("onion skin" phenomena). These "frequent flyer" patients seem to be at high risk for additional recurrences. SCB might emerge as the treatment strategy in this setting.

Nanolute ISR subgroup results showed good clinical outcomes with no death reported at 12 months.