AZ AKUT KORONÁRIA SZINDRÓMÁK ELLÁTÁSA

Horváth Iván

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Intervenciós Kardiológia

PTE, KK, Szívgyógyászati Klinika

PARADIGMA VÁLTÁS:

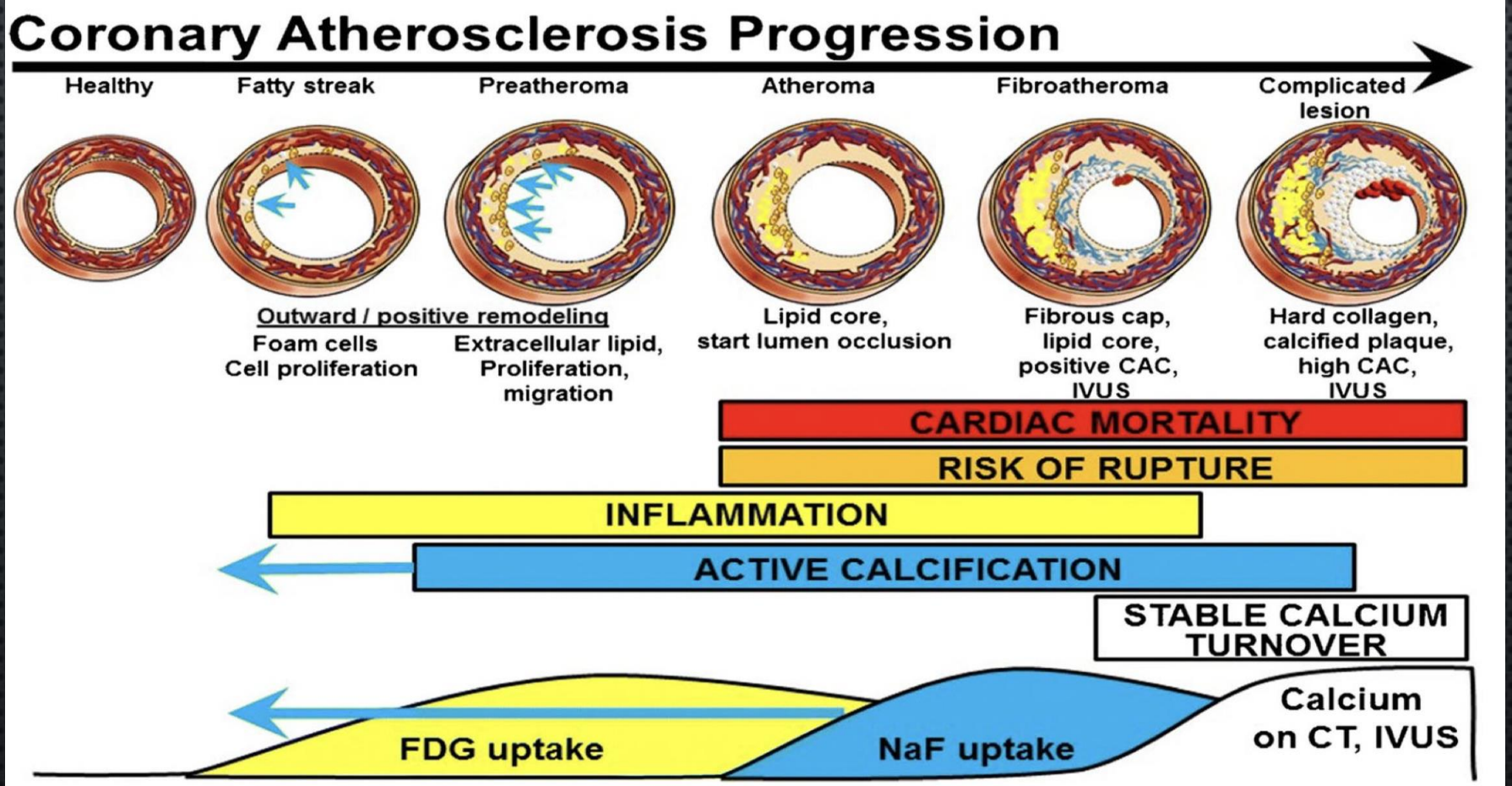
XIX. Század vége New York:

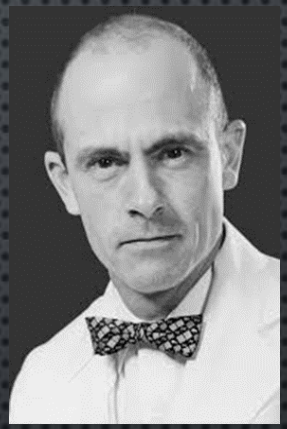
170 000 ló élt a városban (átlag 2-4 év)

1 206 299 ember élt ugyanitt

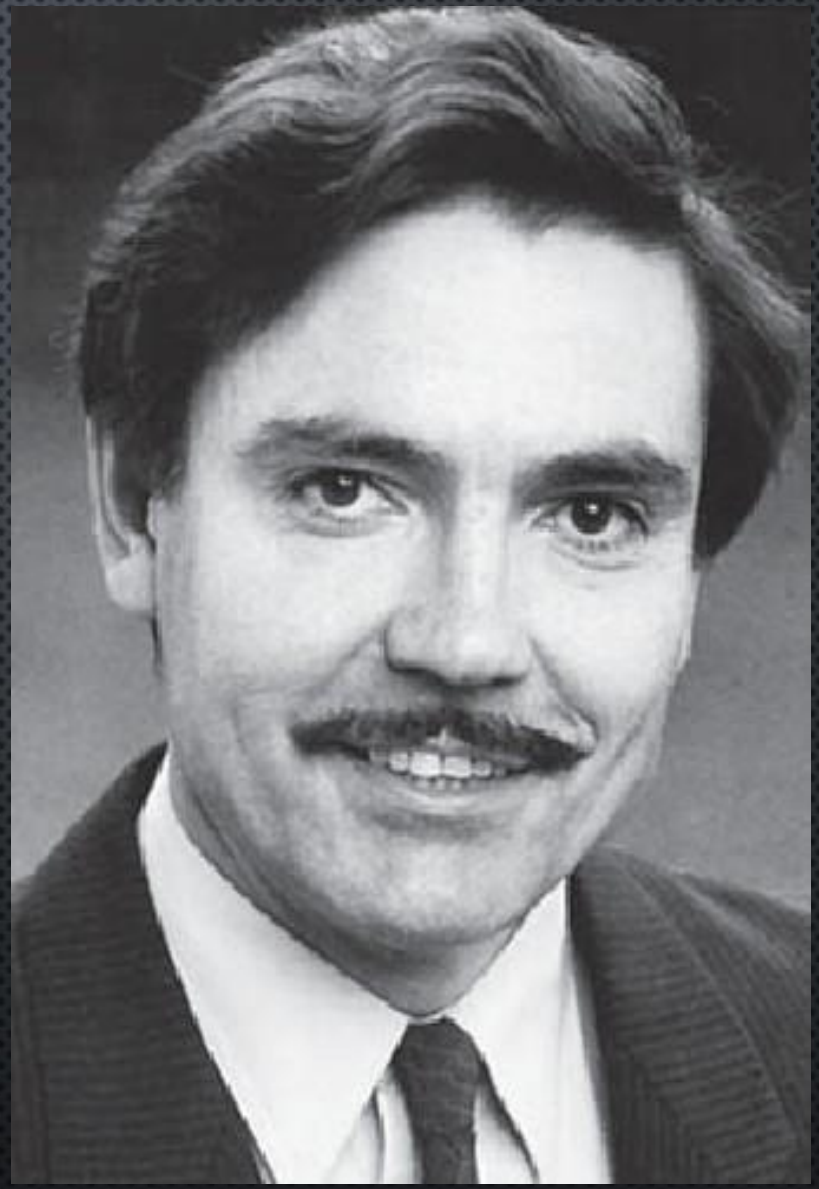
Napi 1812 t szilárd ürülék – 1930 toronyházak felső szintjét érte volna el!

Coronary ^{18}F-Sodium Fluoride Uptake



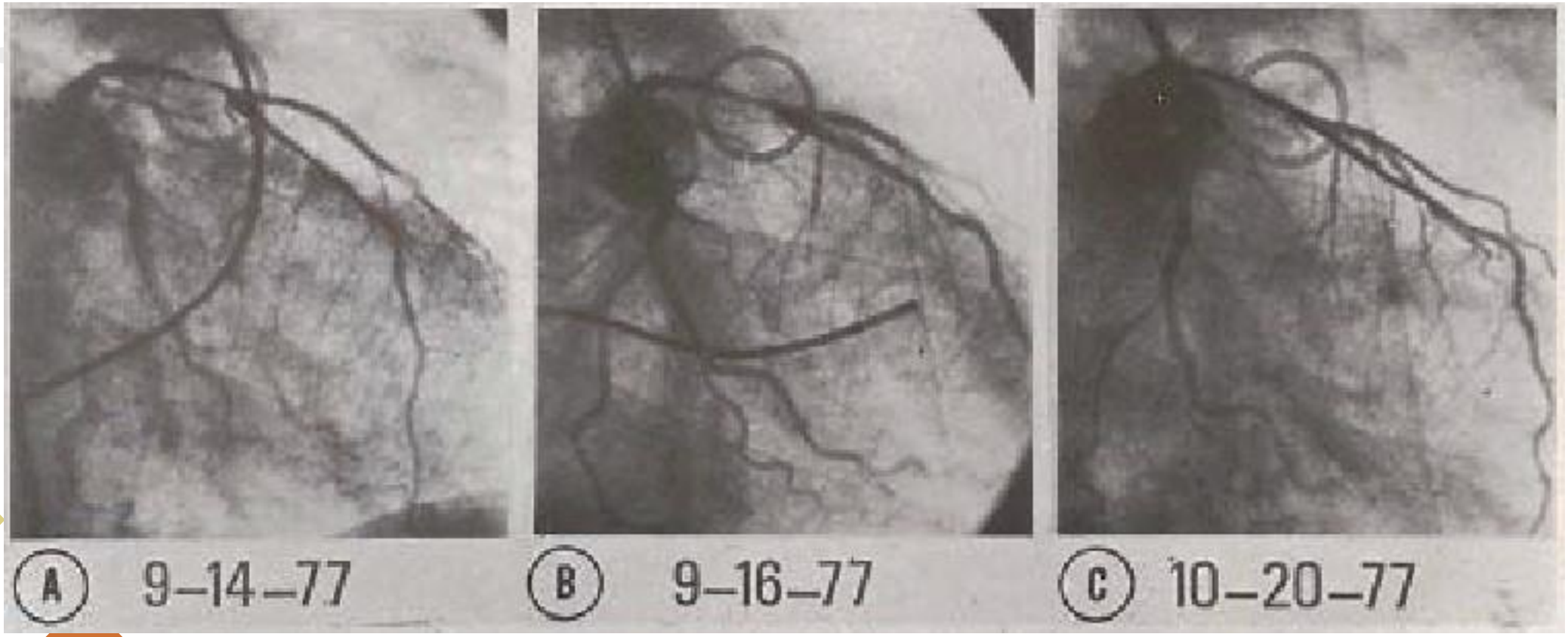


Charles Theodore Dotter 1964 (1920- 1985)

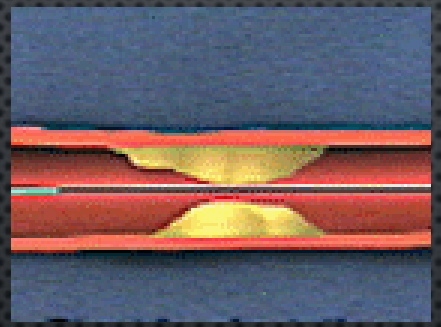
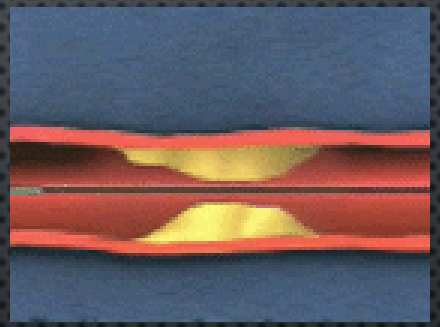


Andreas Gruentzig 1976
(25 June 1939 – 27 October 1985)

AZ ELSŐ HUMÁN 1977

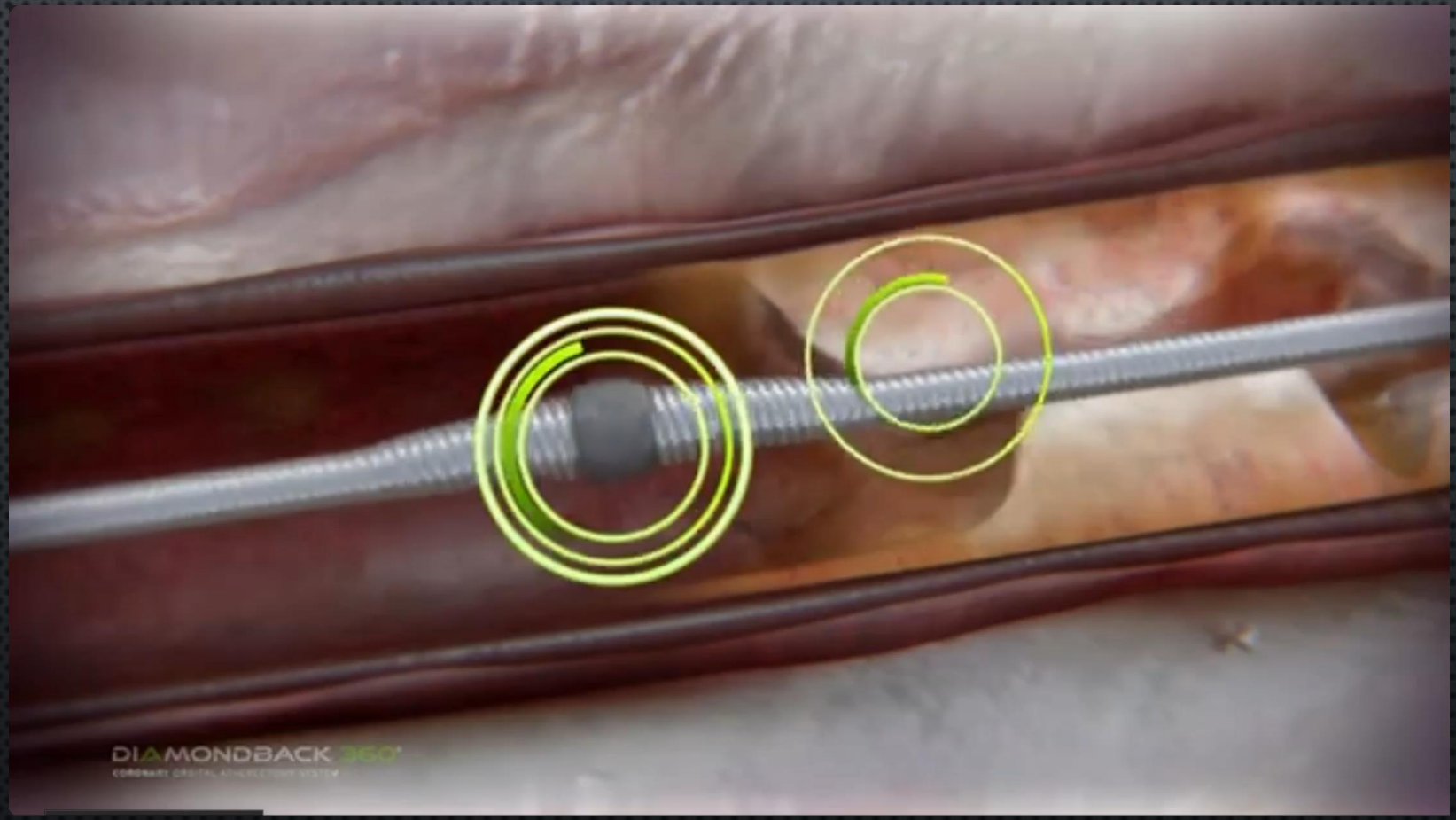


STENT *VS. BALLON*

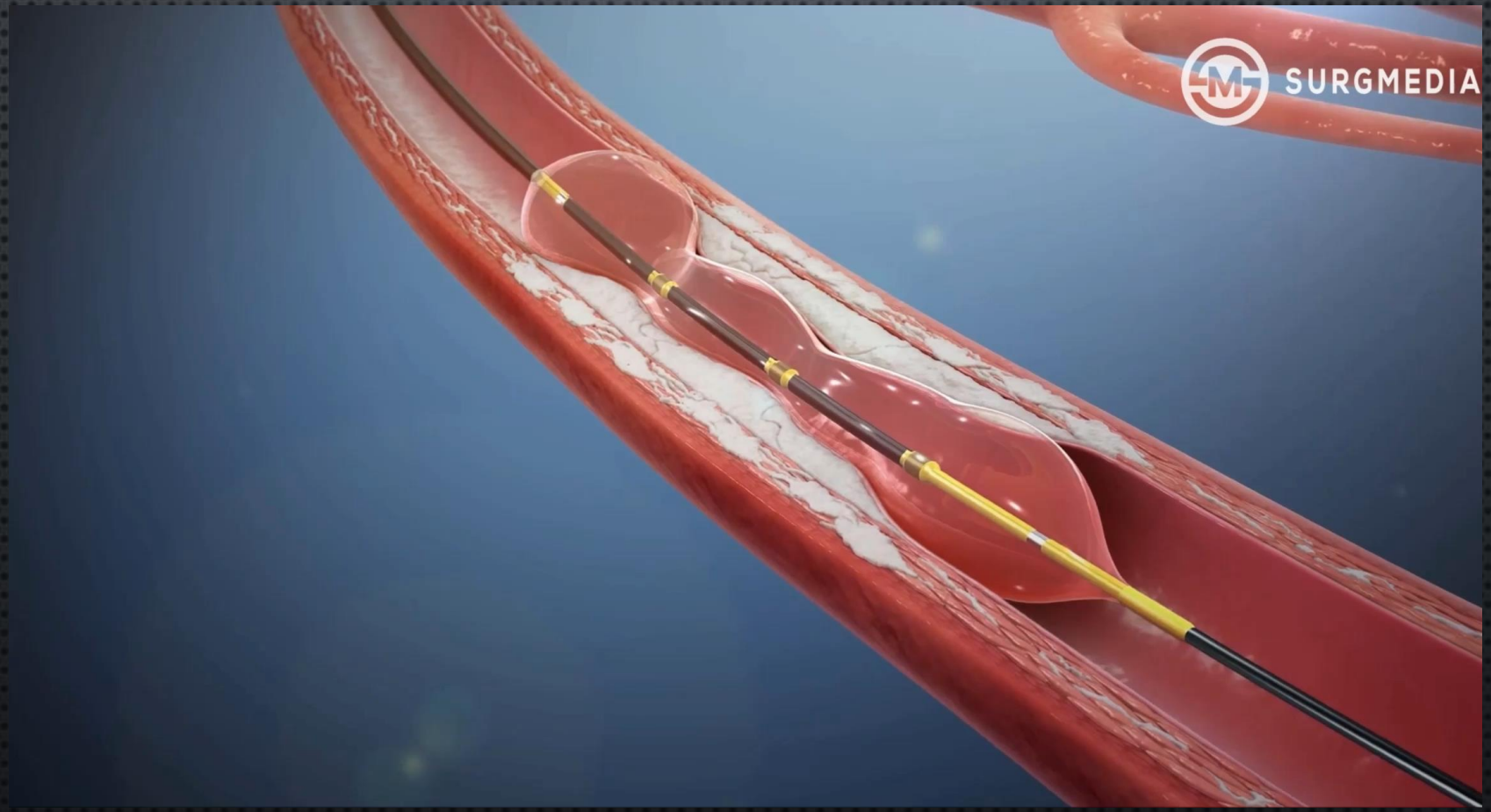





ROTÁCIÓS KEZELÉS

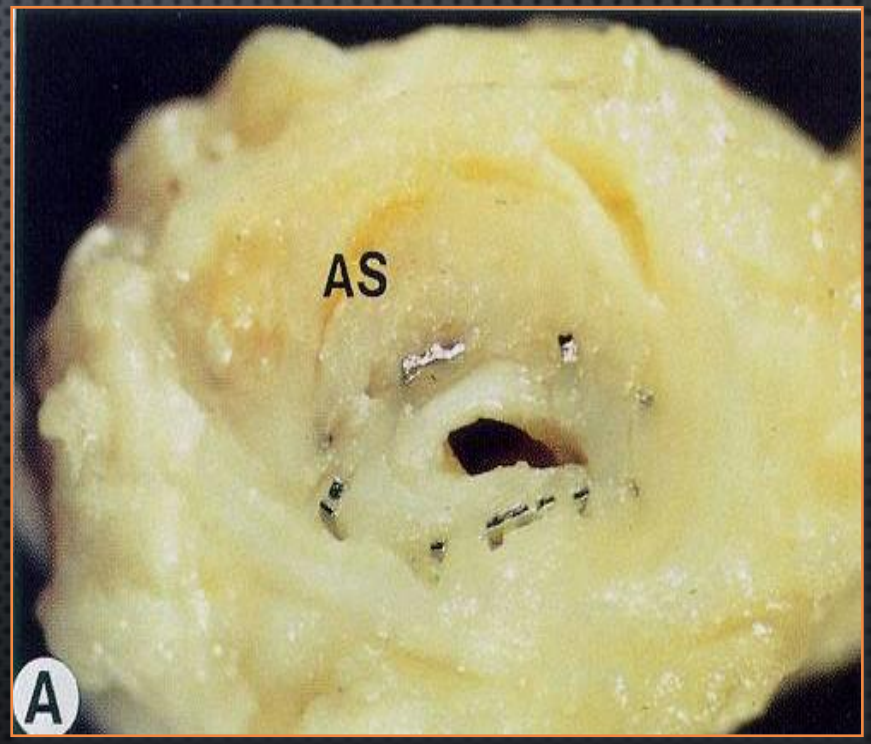
ORBITA



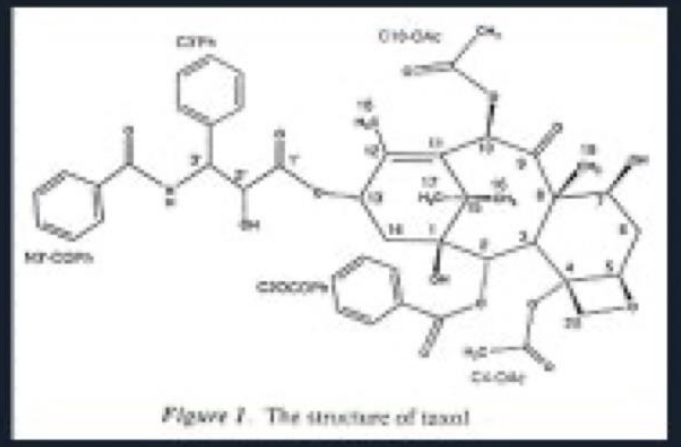
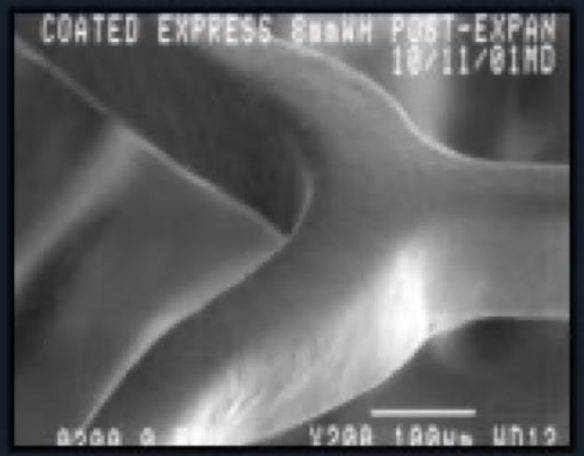
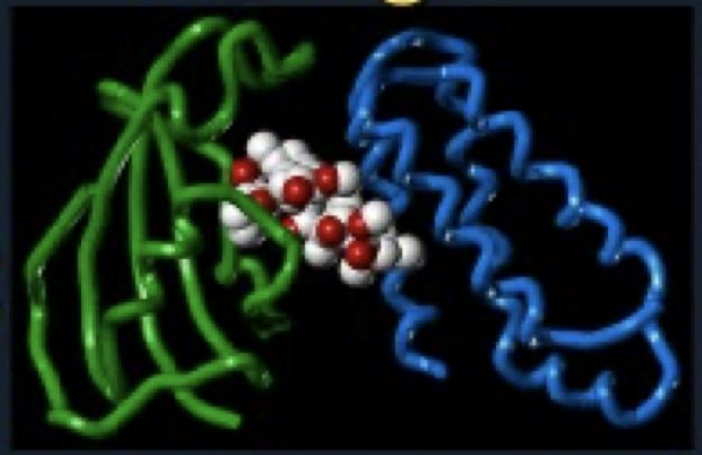
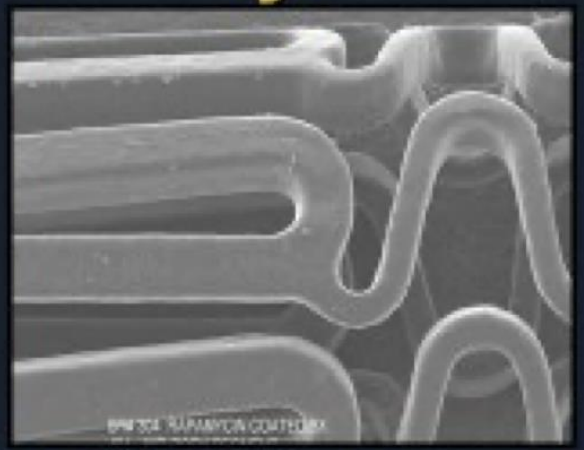
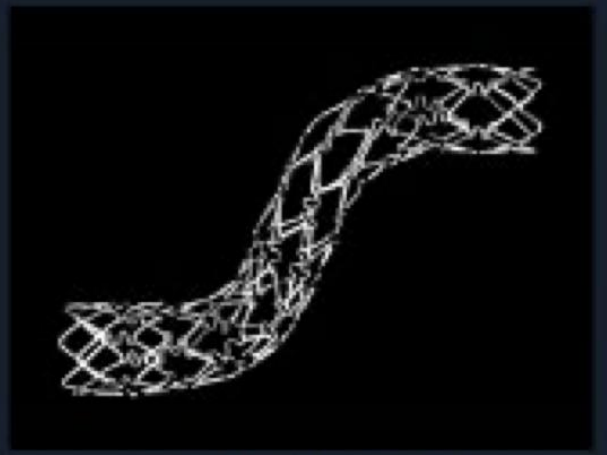
KŐZÚZÁS

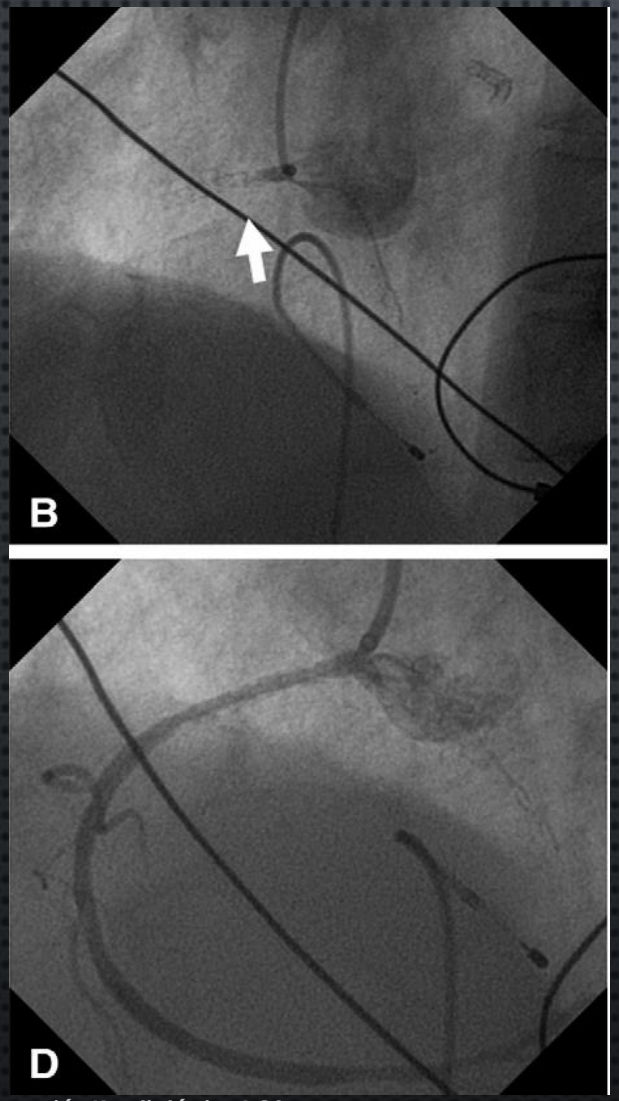


RESTENOSIS



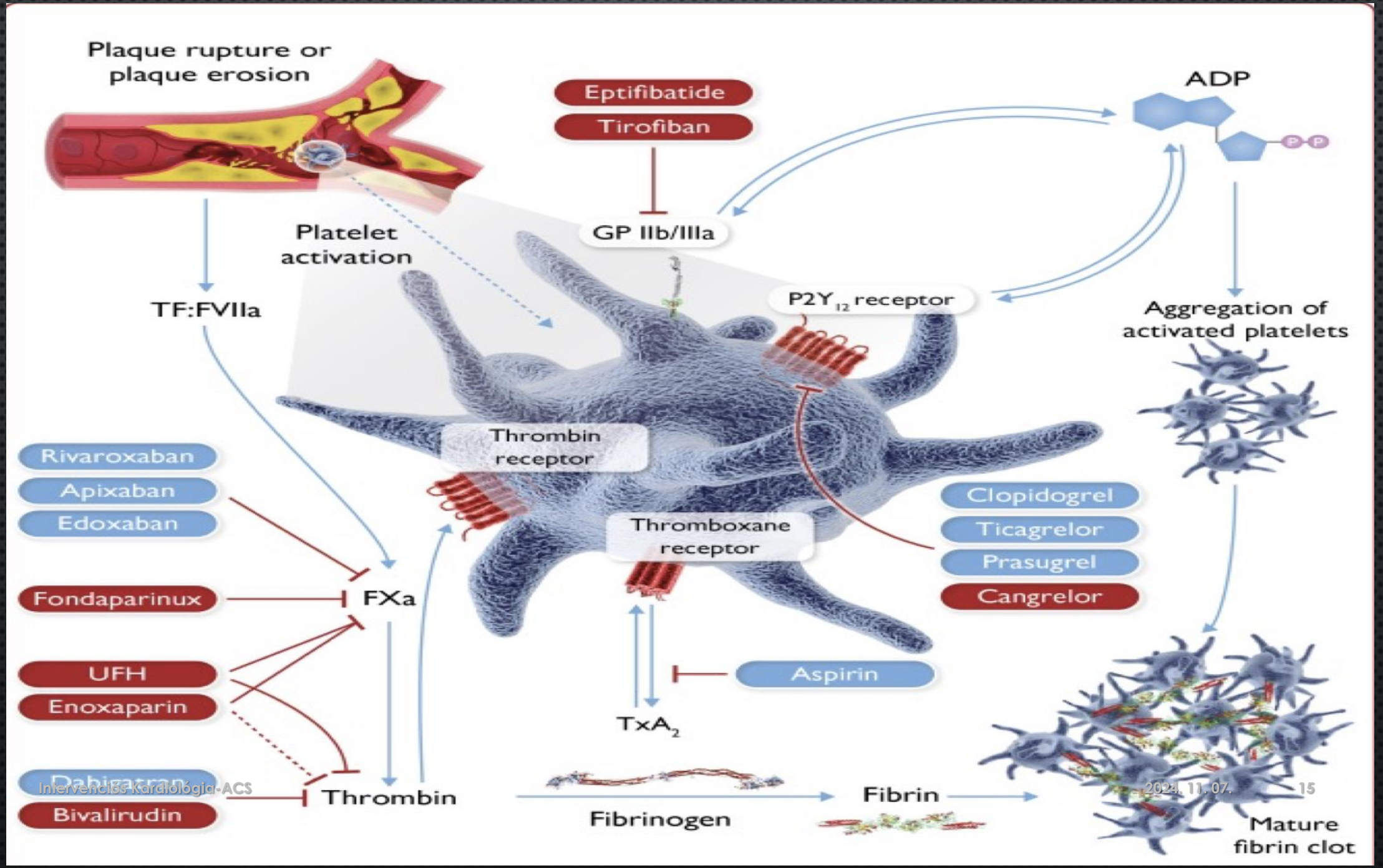
First Generation DES

	Drug	Polymer	Stent
TAXUS	 Paclitaxel	 Polyolefin derivative	 Express^2
Cypher	 Sirolimus	 PEVA + PBMA blend	 BX Velocity



STENT THROMBOSIS

- ARTEFICIAL :
- 100% AMI
- 50% DEATH


Plaque rupture or plaque erosion
Eptifibatide
Tirofiban
ADP
GP IIb/IIIa
Platelet activation
TF:FVIIa
P2Y12 receptor
Aggregation of activated platelets
Thrombin receptor
Rivaroxaban
Apixaban
Edoxaban
Clopidogrel
Ticagrelor
Prasugrel
Cangrelor
Thromboxane receptor
Fondaparinux
FXa
UFH
Enoxaparin
Aspirin
TxA2
Dabigatran
Bivalirudin
Thrombin
Fibrinogen
Fibrin
Mature fibrin clot

Evidence basis (1974-2021)

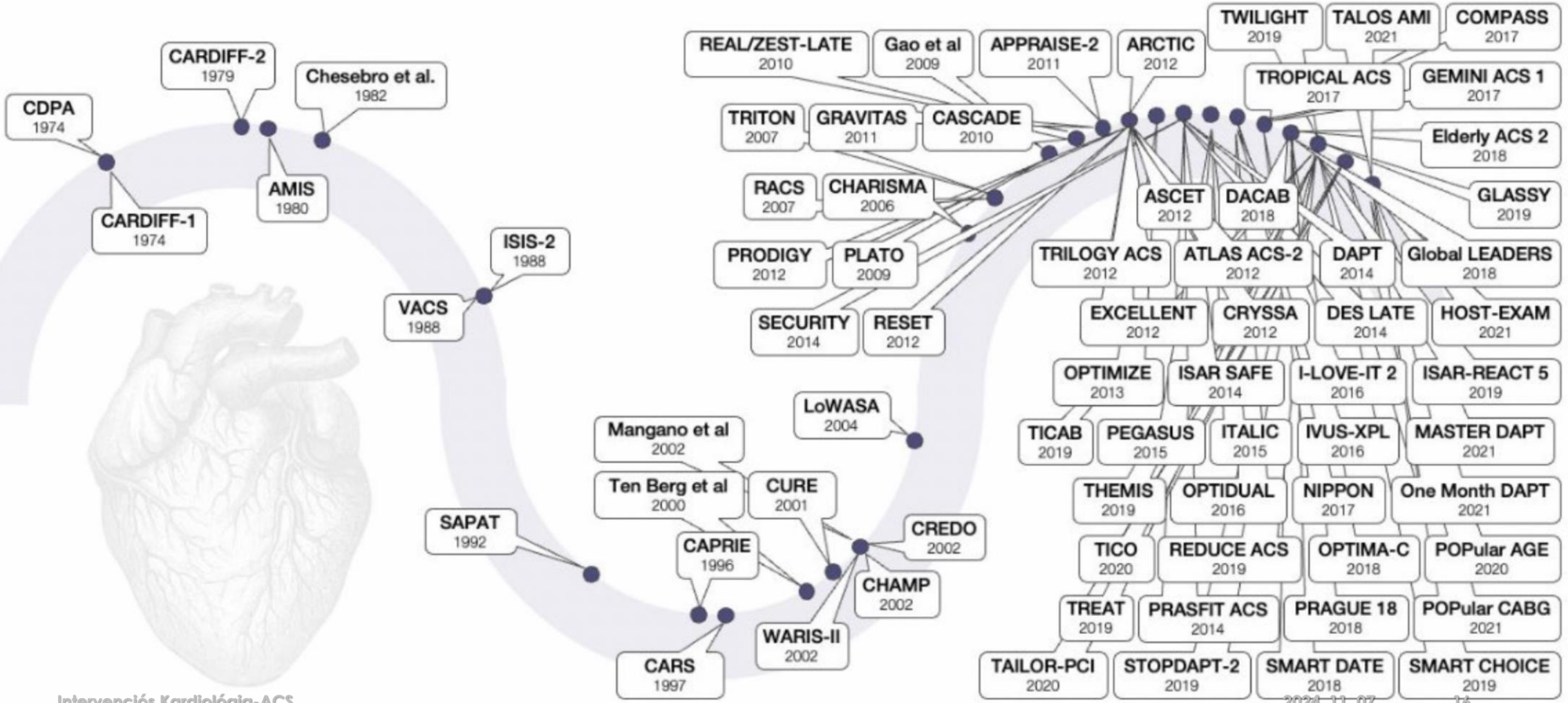
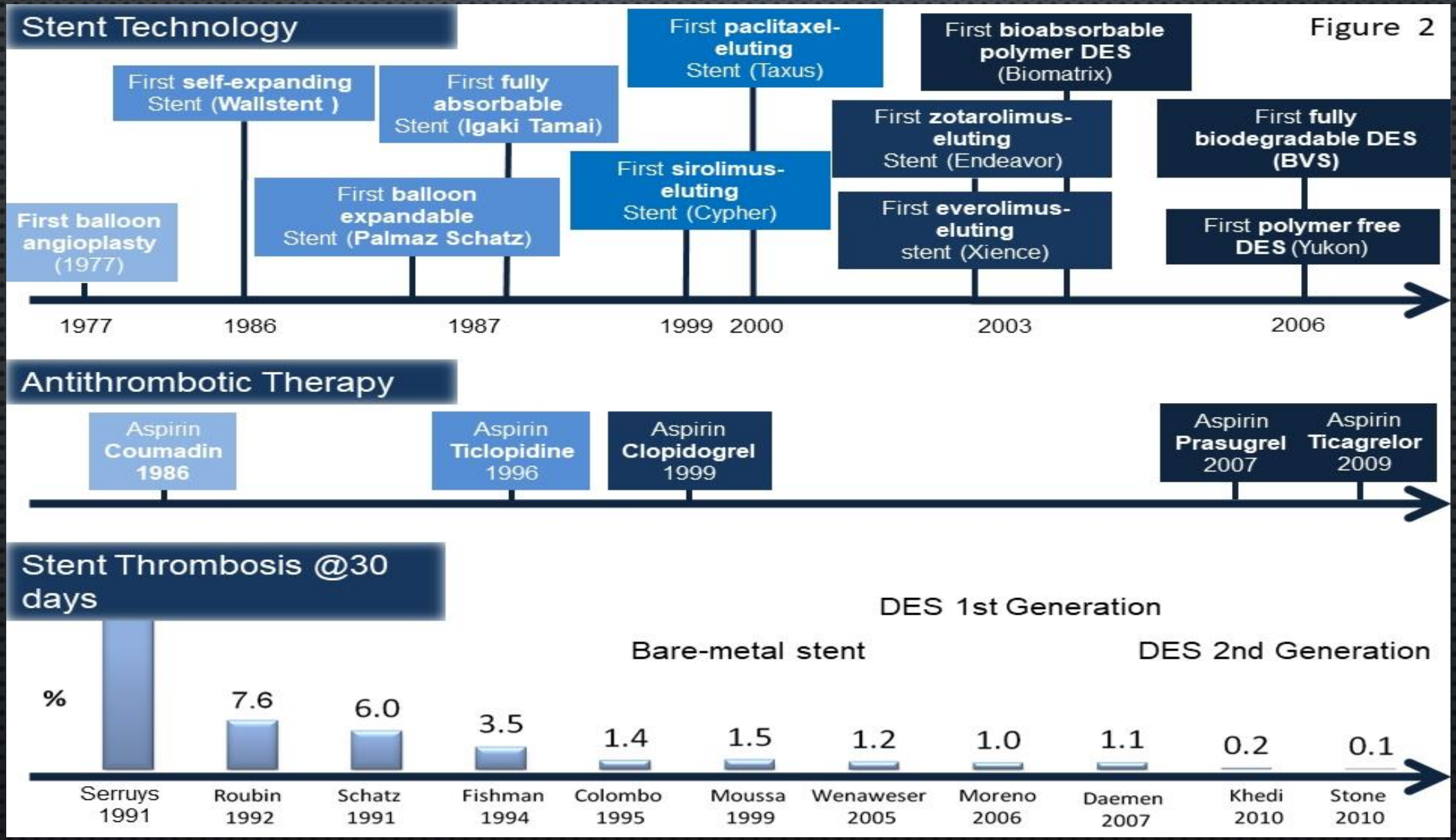


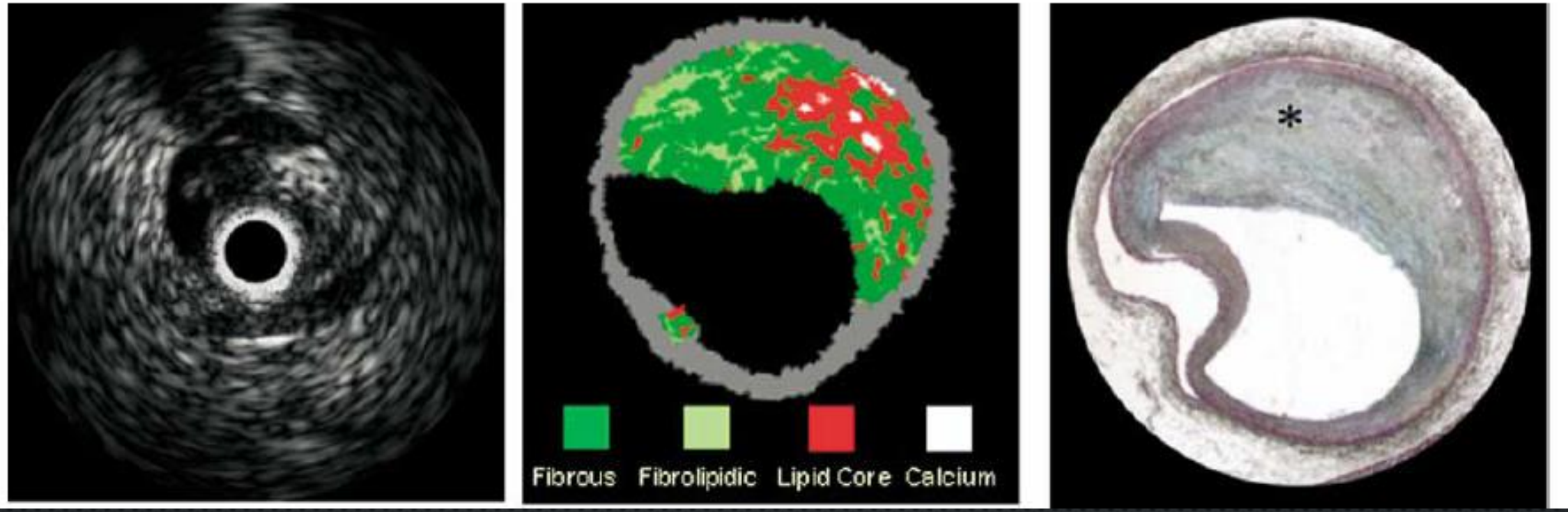
Figure 2


Stent Technology
First balloon angioplasty (1977)
First self-expanding Stent (Wallstent)
First balloon expandable Stent (Palmaz Schatz)
First fully absorbable Stent (Igaki Tamai)
First paclitaxel-eluting Stent (Taxus)
First sirolimus-eluting Stent (Cypher)
First bioabsorbable polymer DES (Biomatrix)
First zotarolimus-eluting Stent (Endeavor)
First everolimus-eluting stent (Xience)
First fully biodegradable DES (BVS)
First polymer free DES (Yukon)
1977
1986
1987
1999
2000
2003
2006
Antithrombotic Therapy
Aspirin Coumadin 1986
Aspirin Ticlopidine 1996
Aspirin Clopidogrel 1999
Aspirin Prasugrel 2007
Aspirin Ticagrelor 2009
Stent Thrombosis @30 days
DES 1st Generation
Bare-metal stent
DES 2nd Generation
%
7.6
6.0
3.5
1.4
1.5
1.2
1.0
1.1
0.2
0.1
Serruys 1991
Roubin 1992
Schatz 1991
Fishman 1994
Colombo 1995
Moussa 1999
Wenaweser 2005
Moreno 2006
Daemen 2007
Khedi 2010
Stone 2010

Proximal
Fiduciary
Site
Distal
Fiduciary
Site

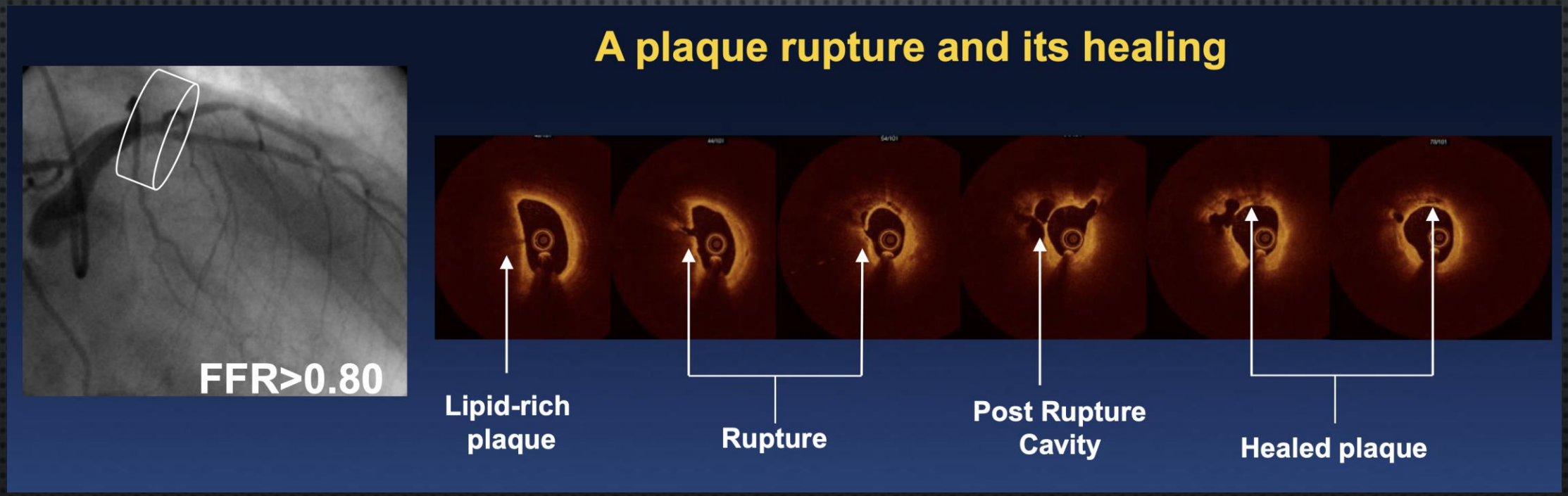
Proximal Fiduciary
Site

Distal Fiduciary
Site

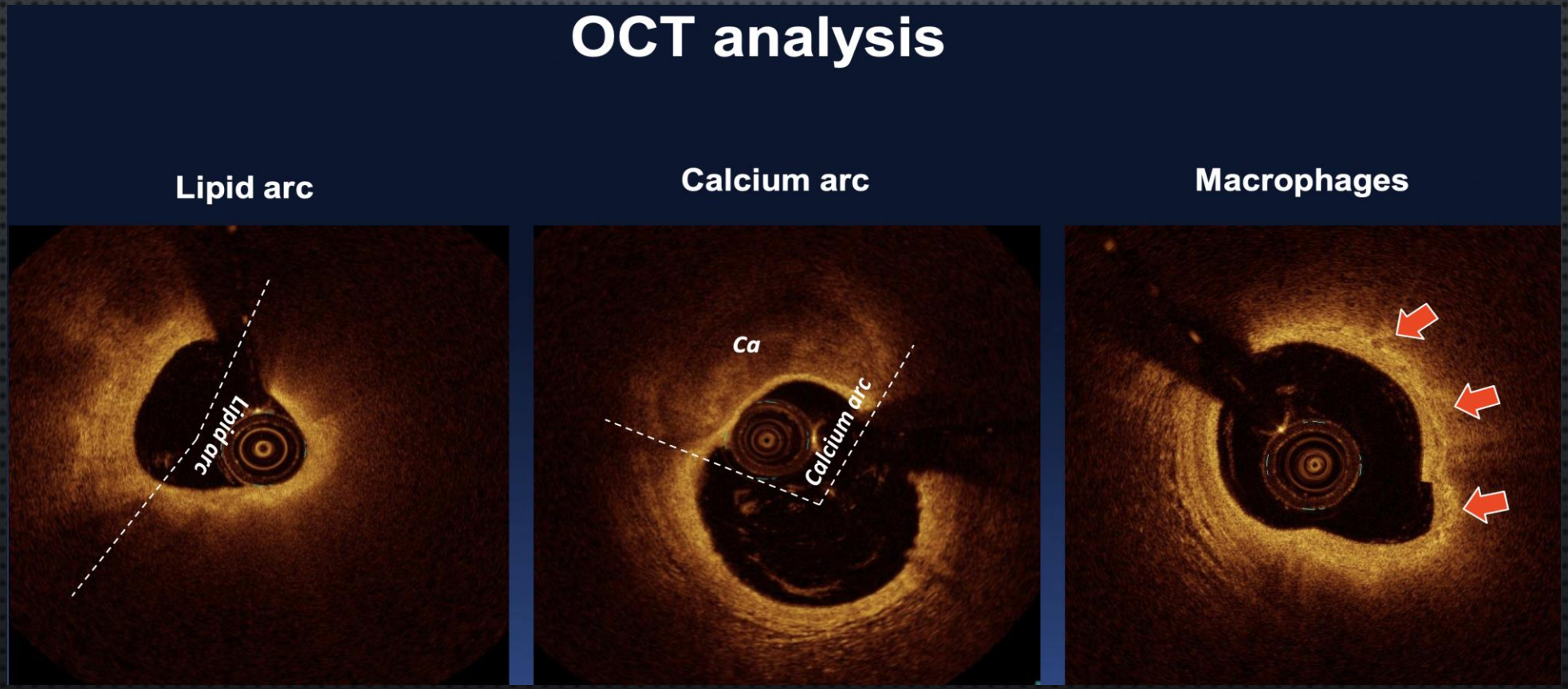


GS-IVUS, VH-IVUS, HISTOLOGY

ANGIO VS. OCT

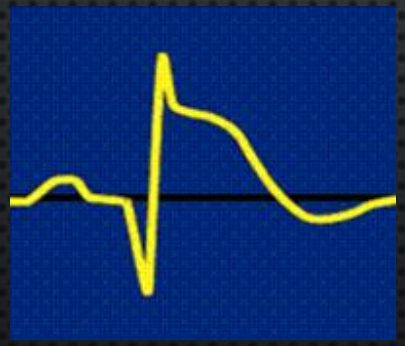

A plaque rupture and its healing
FFR>0.80
Lipid-rich plaque
Rupture
Post Rupture Cavity
Healed plaque

LDL-C OPTIMALIZÁLÁS



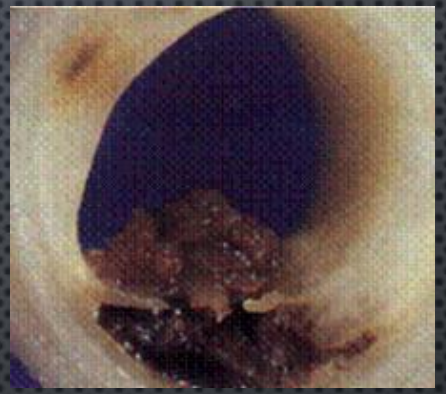
ACS – STEMI

Adapted from Michael Davies

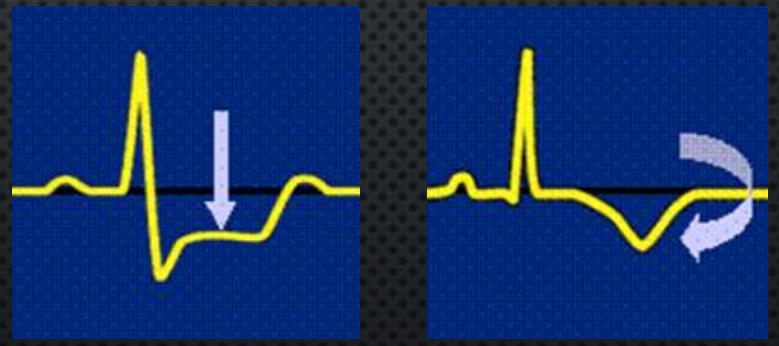


Troponin elevated

ACS – NSTEMI

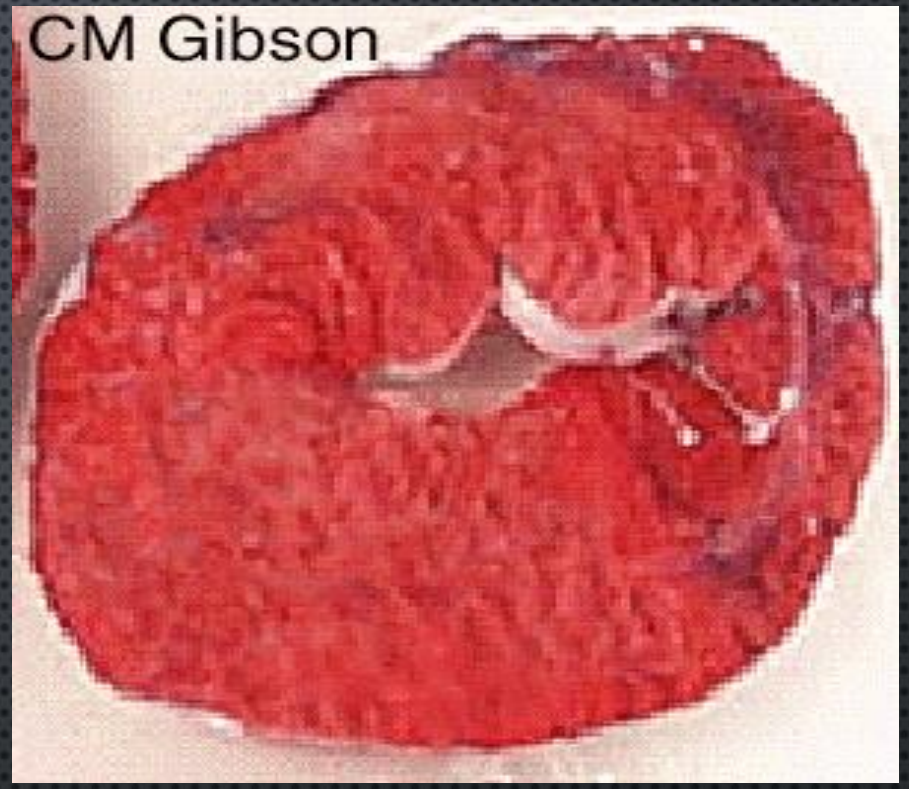


Adapted from Michael Davies

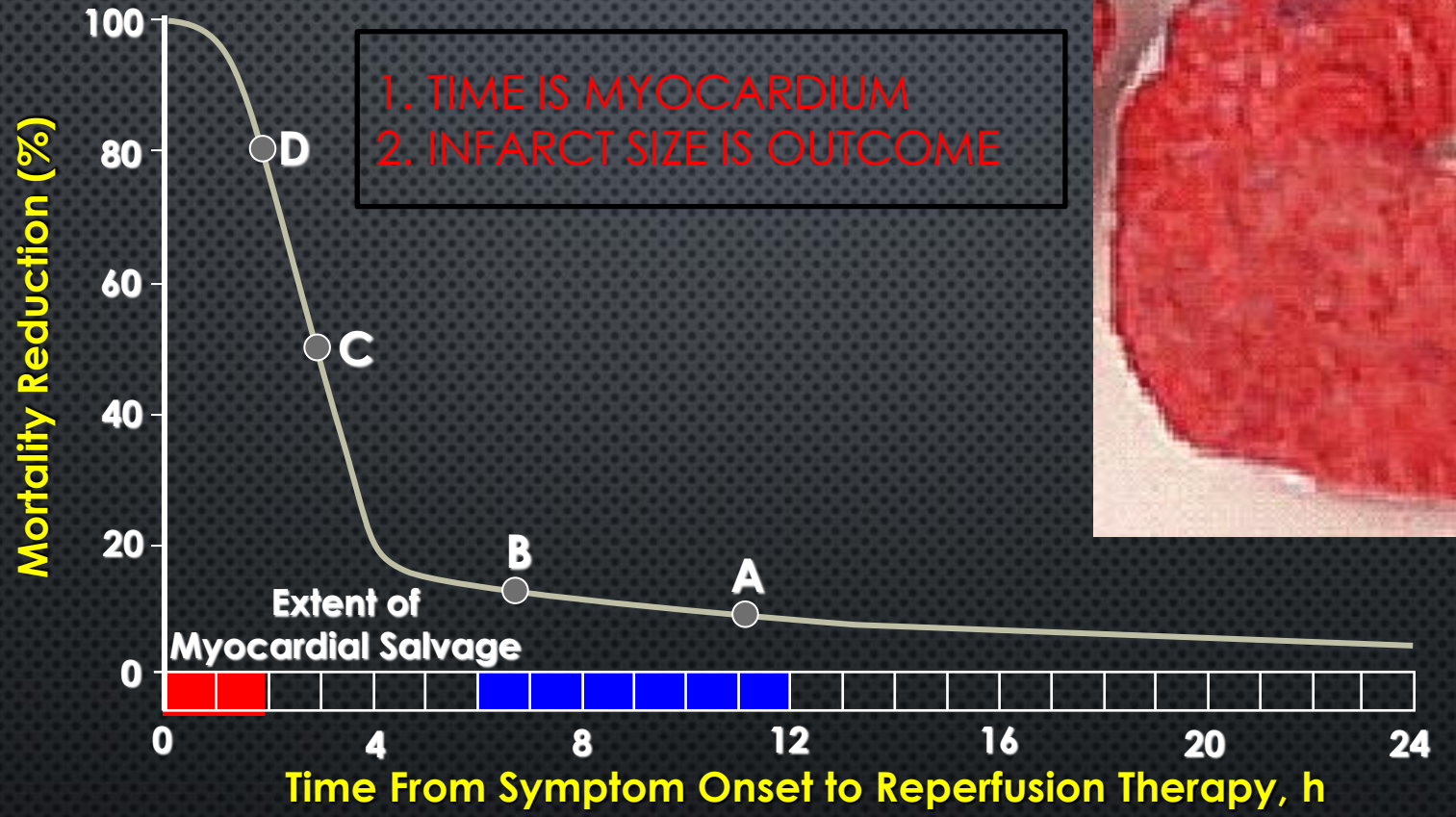


Troponin elevated or normal

Time !

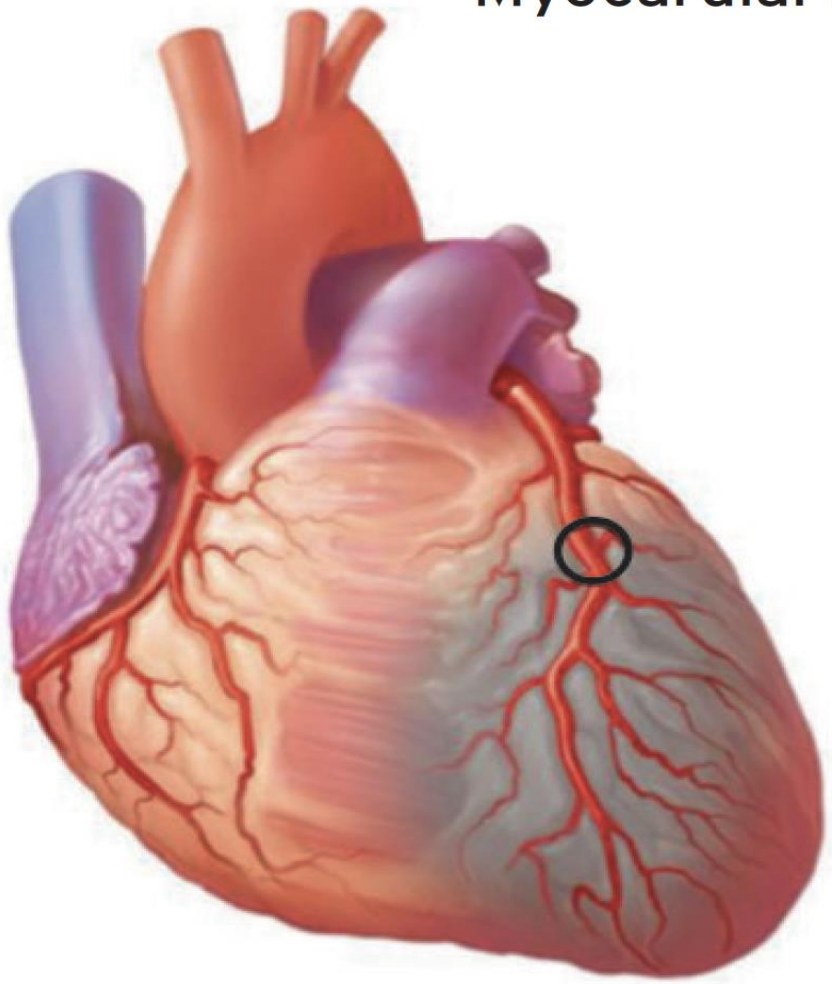


1. TIME IS MYOCARDIUM
2. INFARCT SIZE IS OUTCOME



Gersh BJ, et al. JAMA. 2005;293:979.

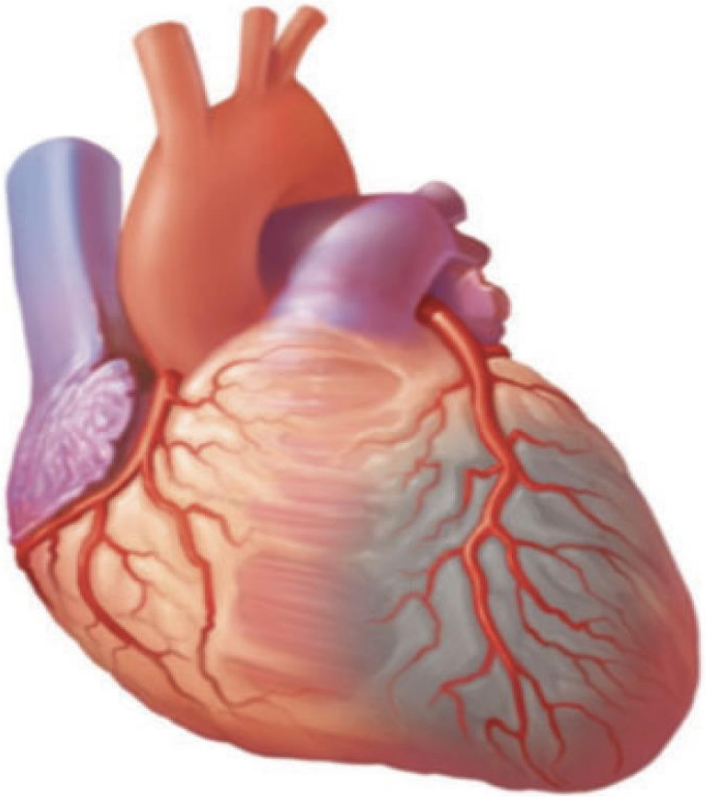
Myocardial Infarction Type 1

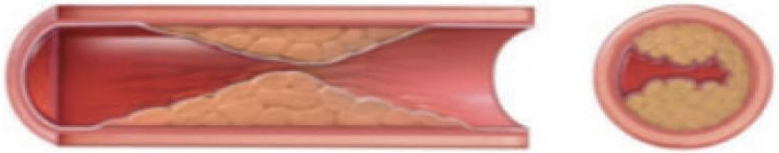


Plaque rupture/erosion with occlusive thrombus

Plaque rupture/erosion with non-occlusive thrombus

Myocardial Infarction Type 2



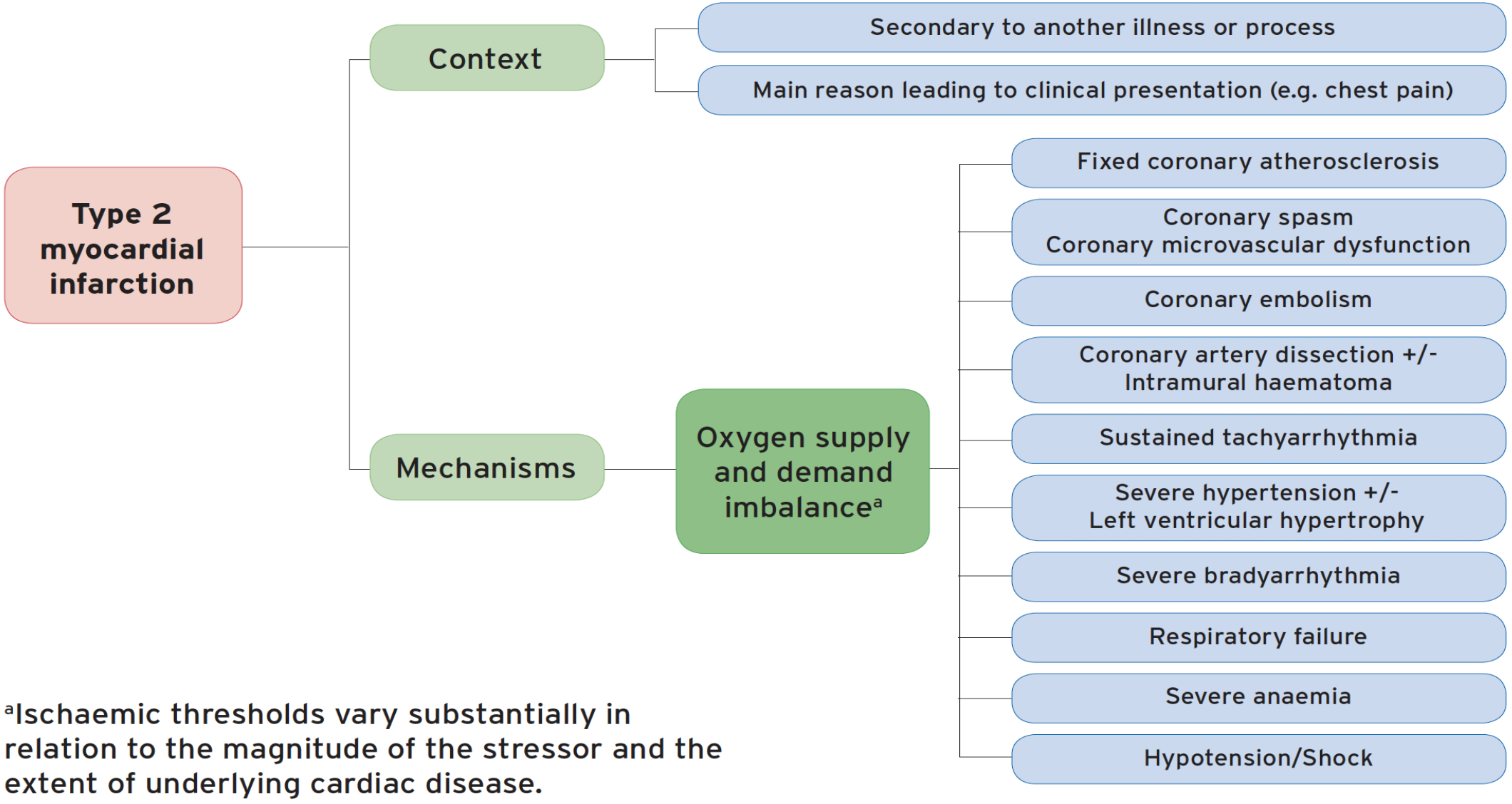


Atherosclerosis and oxygen supply/demand imbalance

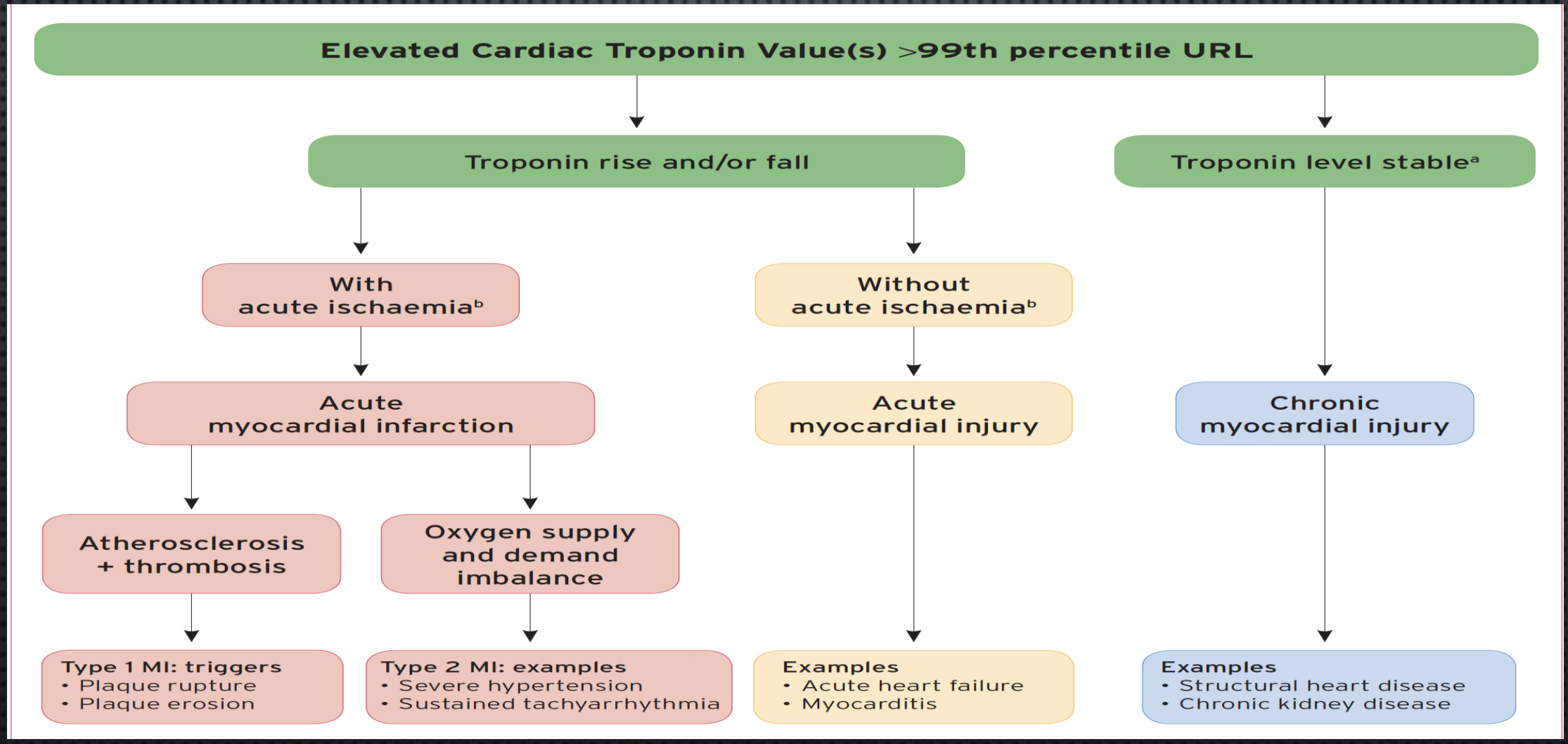
Vasospasm or coronary microvascular dysfunction

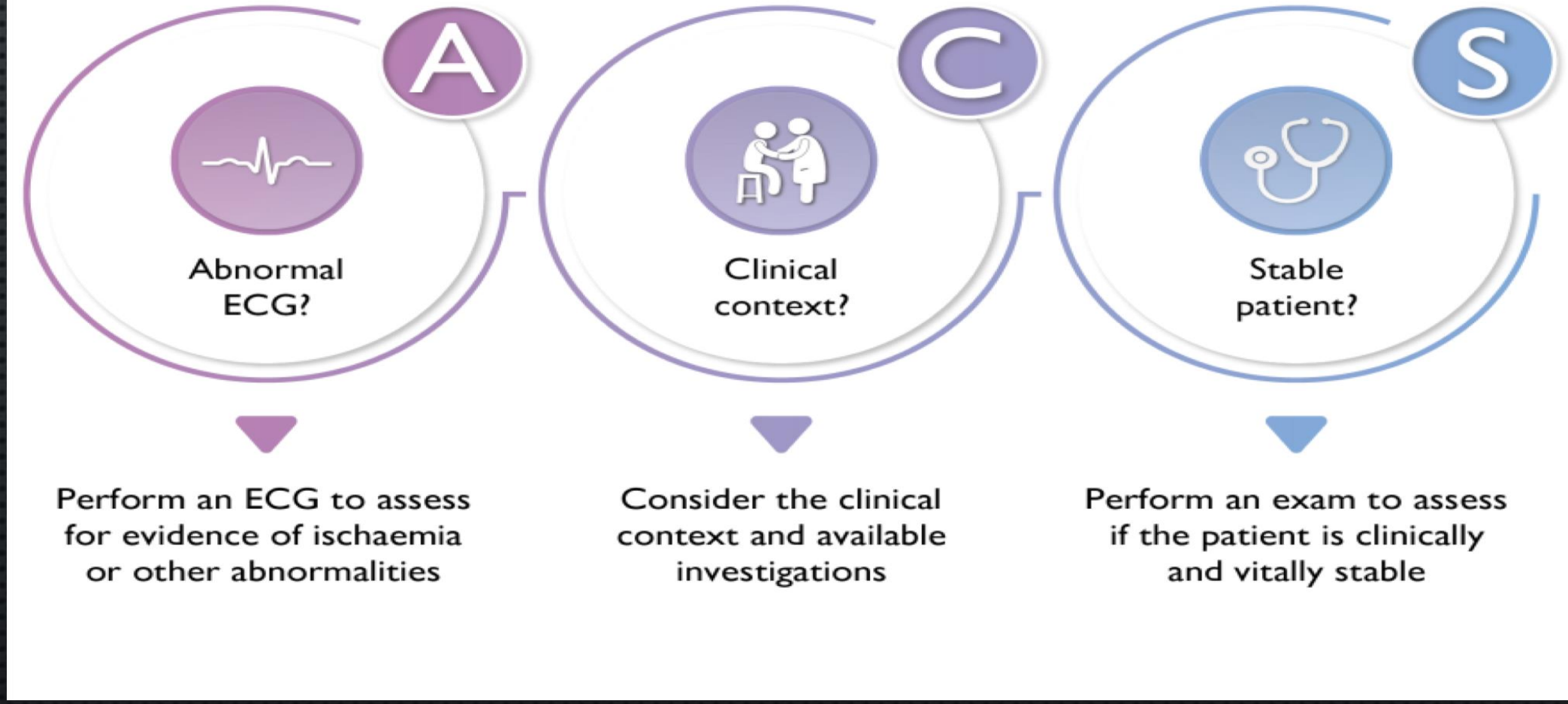
Non-atherosclerotic coronary dissection

Oxygen supply/demand imbalance alone



[a]Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.


Elevated Cardiac Troponin Value(s) >99th percentile URL
Troponin rise and/or fall
Troponin level stable[a]
With acute ischaemia[b]
Without acute ischaemia[b]
Acute myocardial infarction
Acute myocardial injury
Chronic myocardial injury
Atherosclerosis + thrombosis
Oxygen supply and demand imbalance
Type 1 MI: triggers
• Plaque rupture
• Plaque erosion
Type 2 MI: examples
• Severe hypertension
• Sustained tachyarrhythmia
Examples
• Acute heart failure
• Myocarditis
Examples
• Structural heart disease
• Chronic kidney disease


A
Abnormal ECG?
Perform an ECG to assess for evidence of ischaemia or other abnormalities
C
Clinical context?
Consider the clinical context and available investigations
S
Stable patient?
Perform an exam to assess if the patient is clinically and vitally stable

Clinical presentation

ECG

If a patient has signs/symptoms suggestive of ACS, perform an ECG within 10 min of FMC

Working diagnosis[a]

STEMI

NSTE-ACS

Further investigations

hs-cTn levels

± Angiography

± Imaging

Final diagnosis[b]

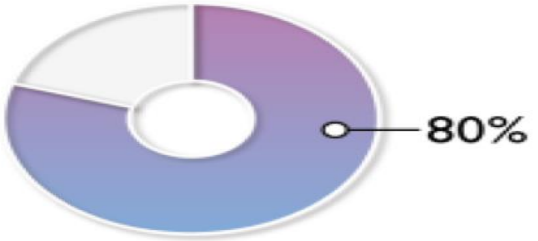
STEMI

NSTEMI

Unstable angina

Non-ACS diagnosis

Chest pain
or pressure



of women and men with ACS
present with chest pain or pressure

Diaphoresis

Epigastric pain/
Indigestion

Shoulder/
Arm pain

Other symptoms, like diaphoresis, indigestion/epigastric pain and shoulder/arm pain occur commonly in both women and men with ACS

Dizziness

Nausea/
Vomiting

Jaw/Neck
pain

Shortness
of breath

Some symptoms may be more common in women with ACS, including:

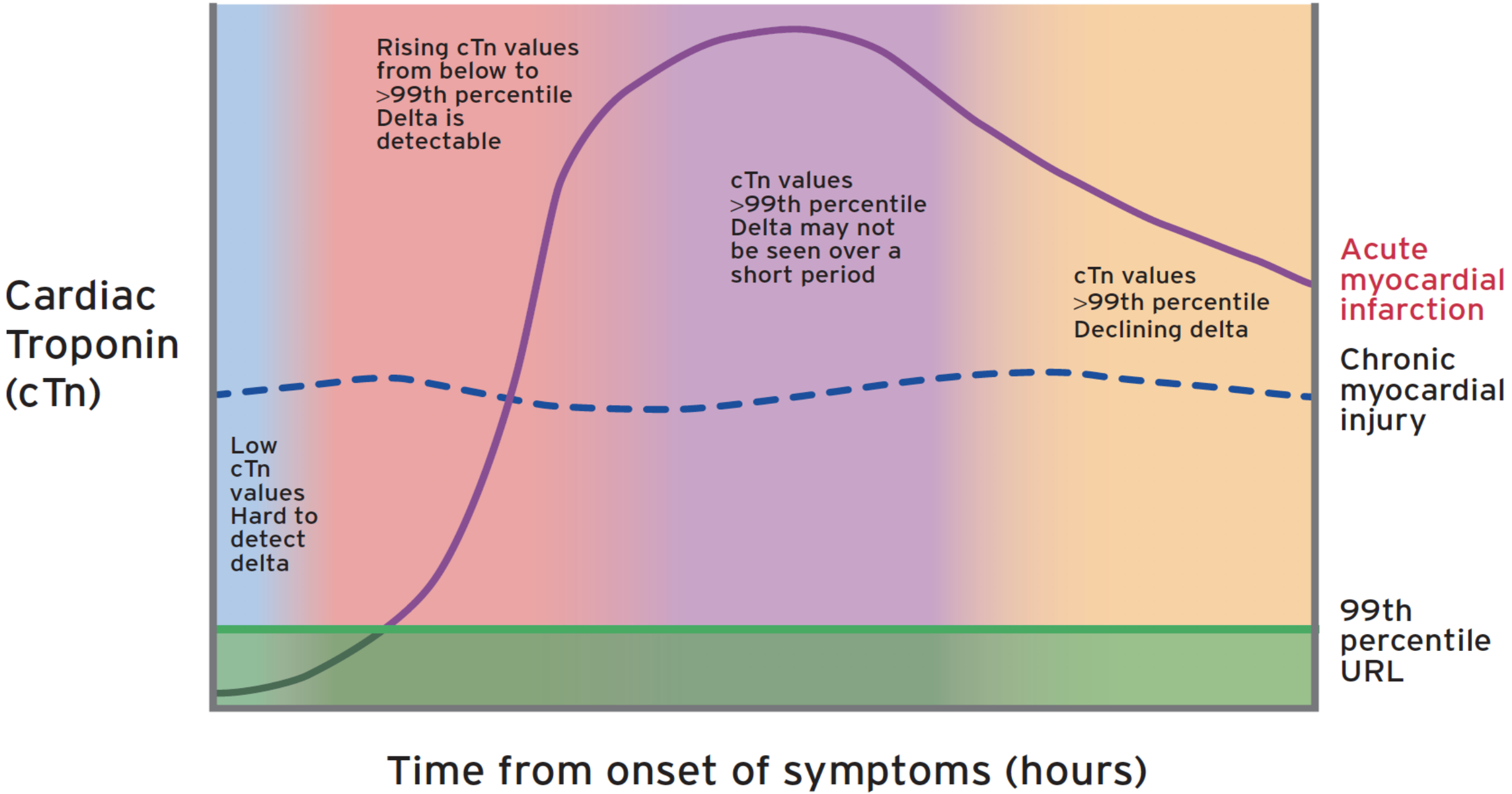
- Dizziness/Syncope
- Nausea/Vomiting
- Jaw/Neck pain
- Shortness of breath
- Pain between the shoulder blades
- Palpitations
- Fatigue

The ACS spectrum

Clinical presentation	Oligo/ asymptomatic 	Increasing chest pain/symptoms 	Persistent chest pain/symptoms 	Cardiogenic shock/ acute heart failure 	Cardiac arrest 
ECG findings	Normal 	ST segment depression 	ST segment elevation 	Malignant arrhythmia 	
Working diagnosis	NSTE-ACS			STEMI	
hs-cTn levels	Non-elevated 		Rise and fall 		
Final diagnosis	Unstable angina		NSTEMI	STEMI	

	Prognostic impact	Diagnostic impact	Therapeutic impact
Markers of necrosis			
Creatine phosphokinase MB	+++	+++	++
Myoglobin	++	++	++
Troponin	++++	++++	++++
Markers of myocardial dysfunction or stress			
Atrial natriuretic peptides	+++	+++*	?
Brain natriuretic peptides	++++	++++*	+++*
Copeptin	++	+	?
Proadrenomedullin	++	+	?
Markers of inflammation			
Adiponectin	++	?	?
C-reactive protein	++++	?	++
Growth differentiation factor 15	+++	?	+
Interleukin 6	+++	?	?
Soluble ST2	+	?	?
Tumor necrosis factor α	++	?	?
Myeloid-related protein 8/14	+	?	?
Markers of ischemia			
Choline	++	?	?
Heart-type fatty acid-binding protein	++	++	?
Ischemia modified albumin	+	+	?
Markers of plaque destabilization/rupture			
Lipoprotein-associated phospholipase A2	+++	?	?
Matrix metalloproteinase-9	++	?	?
Myeloperoxidase	+++	++	?
Placental growth factor	++	?	?
Pregnancy-associated plasma protein A	+++	+	?
Secretory phospholipase A2	+	?	?
Soluble fms-like tyrosine kinase 1	+	+	?
Soluble intercellular adhesion molecule 1	+++	?	?
Markers of platelet activation			
Soluble CD40 ligand	++/?	?	?
Soluble P-selectin	++	?	?

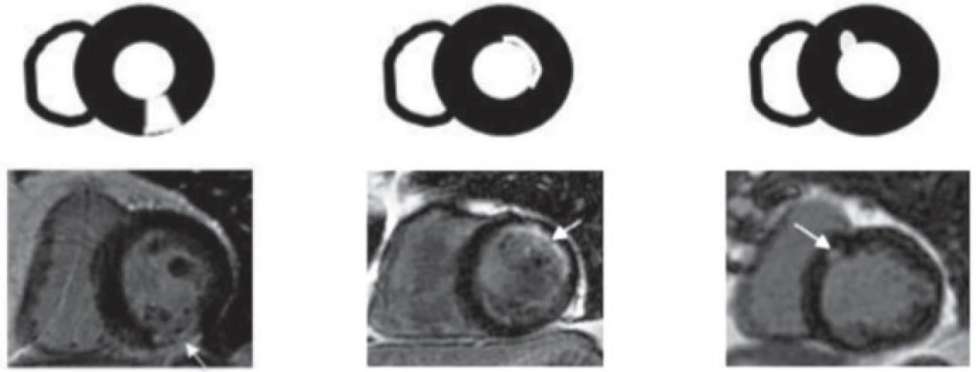
+, Some evidence by small studies; ++, intermediate evidence from several studies or one large study or trial; +++, good evidence from several large studies or trials; ++++, evidence; ?, conflicting results or no results available or not applicable.
This table only gives an overview of the evidence published for the various markers. It does not indicate the clinical utility of different markers (eg, a marker might be very use stratification, but not feasible for the clinical setting due to limitations in detection or because it is also elevated at important differential diagnoses).
*For stratification of patients with heart failure.


Cardiac Troponin (cTn)
Rising cTn values from below to >99th percentile Delta is detectable
cTn values >99th percentile Delta may not be seen over a short period
cTn values >99th percentile Declining delta
Low cTn values Hard to detect delta
Acute myocardial infarction
Chronic myocardial injury
99th percentile URL
Time from onset of symptoms (hours)

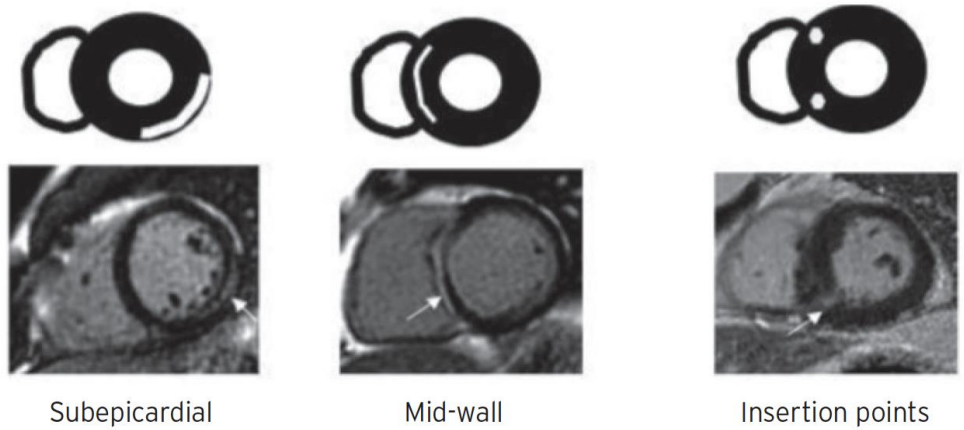
0 h/1 h algorithm	Very low	Low	No 1hΔ	High	1hΔ
hs-cTn T (Elecsys; Roche)	<5	<12	<3	≥52	≥5

0 h/2 h algorithm	Very low	Low	No 2hΔ	High	2hΔ
hs-cTn T (Elecsys; Roche)	<5	<14	<4	≥52	≥10

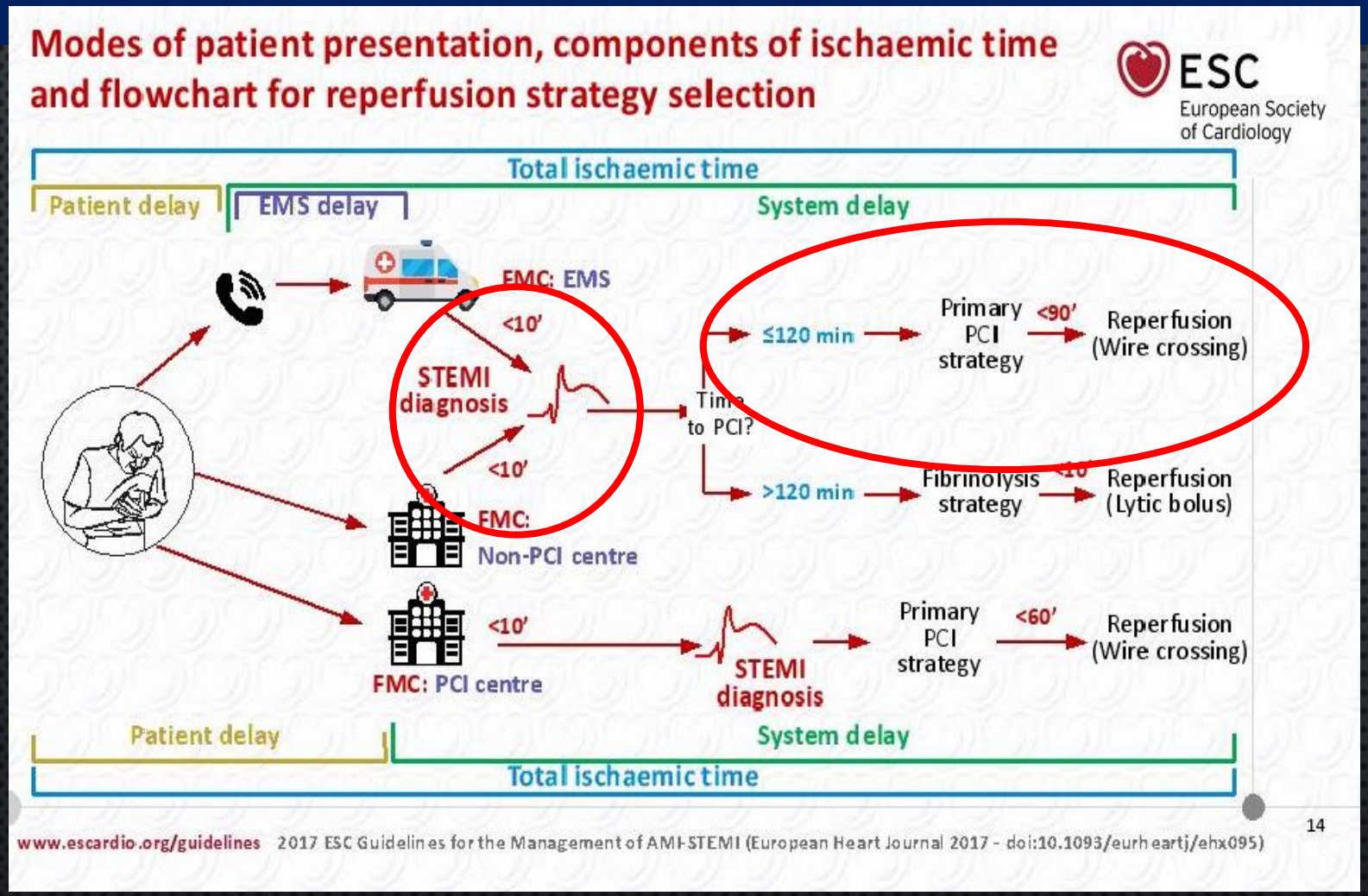
ISCHAEMIC

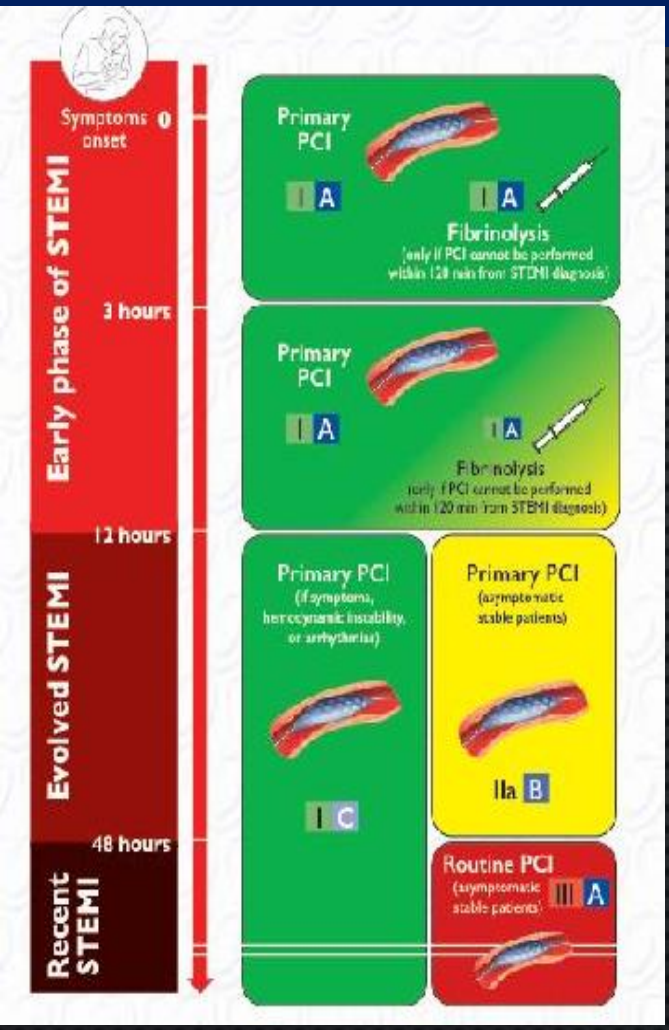


NON-ISCHAEMIC



STEMI


Modes of patient presentation, components of ischaemic time and flowchart for reperfusion strategy selection
ESC
European Society of Cardiology
Total ischaemic time
Patient delay
EMS delay
System delay
FMC: EMS
<10'
STEMI diagnosis
<10'
Time to PCI?
≤120 min
Primary PCI strategy
<90'
Reperfusion (Wire crossing)
>120 min
Fibrinolysis strategy
<10'
Reperfusion (Lytic bolus)
FMC: Non-PCI centre
FMC: PCI centre
<10'
STEMI diagnosis
Primary PCI strategy
<60'
Reperfusion (Wire crossing)
Patient delay
System delay
Total ischaemic time
www.escardio.org/guidelines 2017 ESC Guidelines for the Management of AMI-STEMI (European Heart Journal 2017 - doi:10.1093/eurheartj/ehx095)
14


Symptoms onset
0
Early phase of STEMI
3 hours
12 hours
Evolved STEMI
48 hours
Recent STEMI
Primary PCI
I A
I A
Fibrinolysis
Primary PCI
I A
I A
Fibrinolysis
Primary PCI
(if symptoms, hemodynamic instability, or arrhythmias)
I C
Primary PCI
(asymptomatic stable patients)
IIa B
Routine PCI
(asymptomatic stable patients)
III A

NSTE-ACS

Betegek akut mellkasi fájdalommal, tartós ST-szakasz eleváció nélkül (NSTE-ACS betegek)

Az EKG:

- átmeneti ST-eleváció
- tartós vagy átmeneti ST depresszió
- T-hullám inverzió
- lapos T-hullámok
- T-hullámok pszeudonormalizációja
- EKG akár eltérés nélküli is lehet

Byrne RA et al. 2023 ESC Guidelines for the management of acute coronary syndromes.
Eur Heart J. 2023 Oct 12;44(38):3720-3826.

Acut coronaria syndroma – ACS ST eleváció nélkül

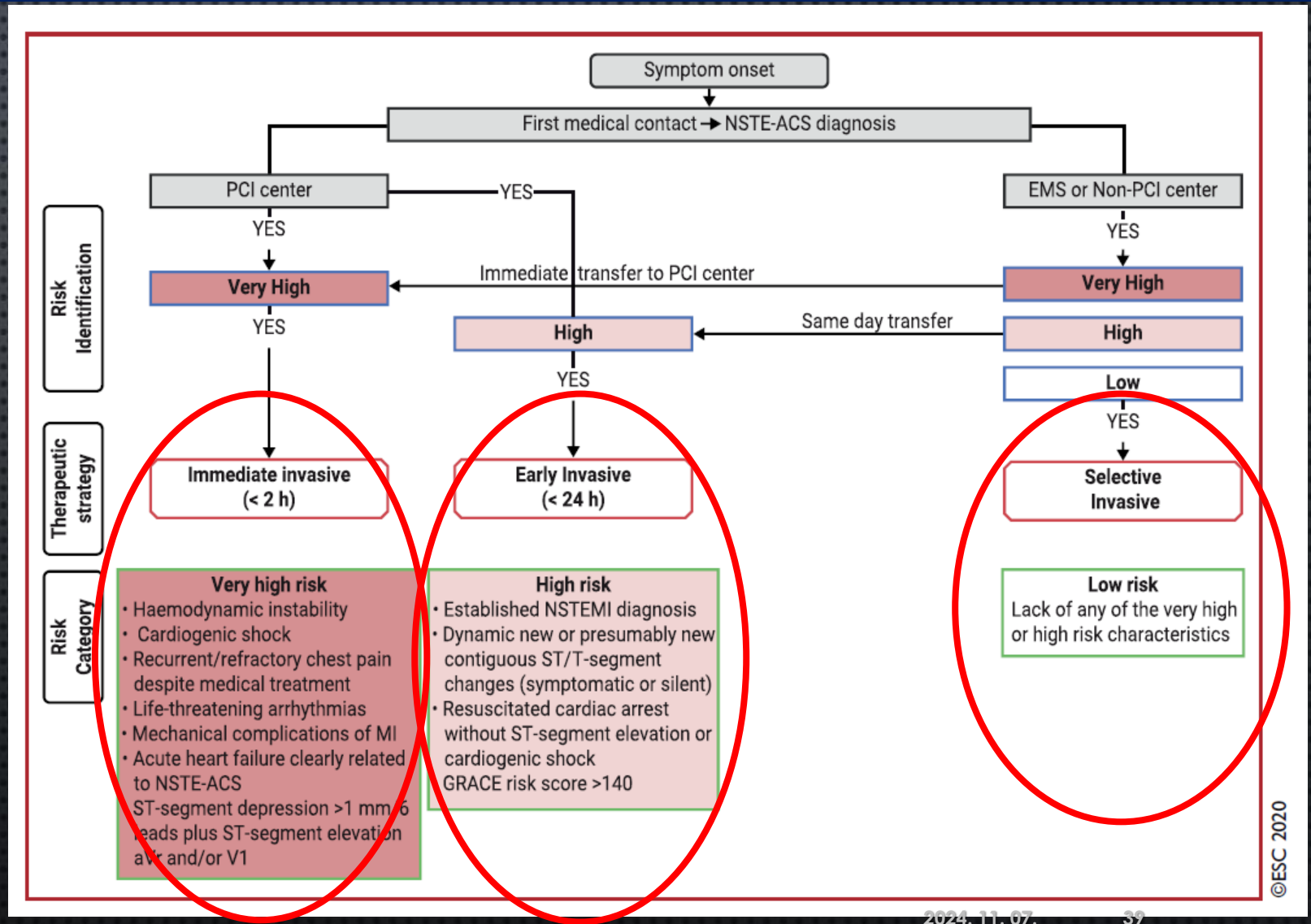
Rizikó stratifikáció az alábbiak alapján:

1. **Klinikai állapot**
   - Folyamatos mellkasi fájdalom
   - Hemodynamikai státusz (szívelégtelenség/kardiogén sokk/újraélesztés)
   - Szövődmény – arrhythmia, mechanikai szövődmény
2. **Komorbiditás – nagy valószínűséggel utal ISZB-re**
   - Diabetes mellitus
   - Csökkent bal kamra funkció
   - Korábbi myocardialis infarctus
   - Korábbi coronaria revascularizatio – PCI vagy CABG
3. **GRACE score**

A szerző saját ábrája

NSTE-ACS RIZIKÓSTRATIFIKÁCIÓ

- KLINIKUM
- EKG
- TROPONIN, VESEFUNKCIÓ
- ECHOCARDIOGRAPHIA
- GRACE



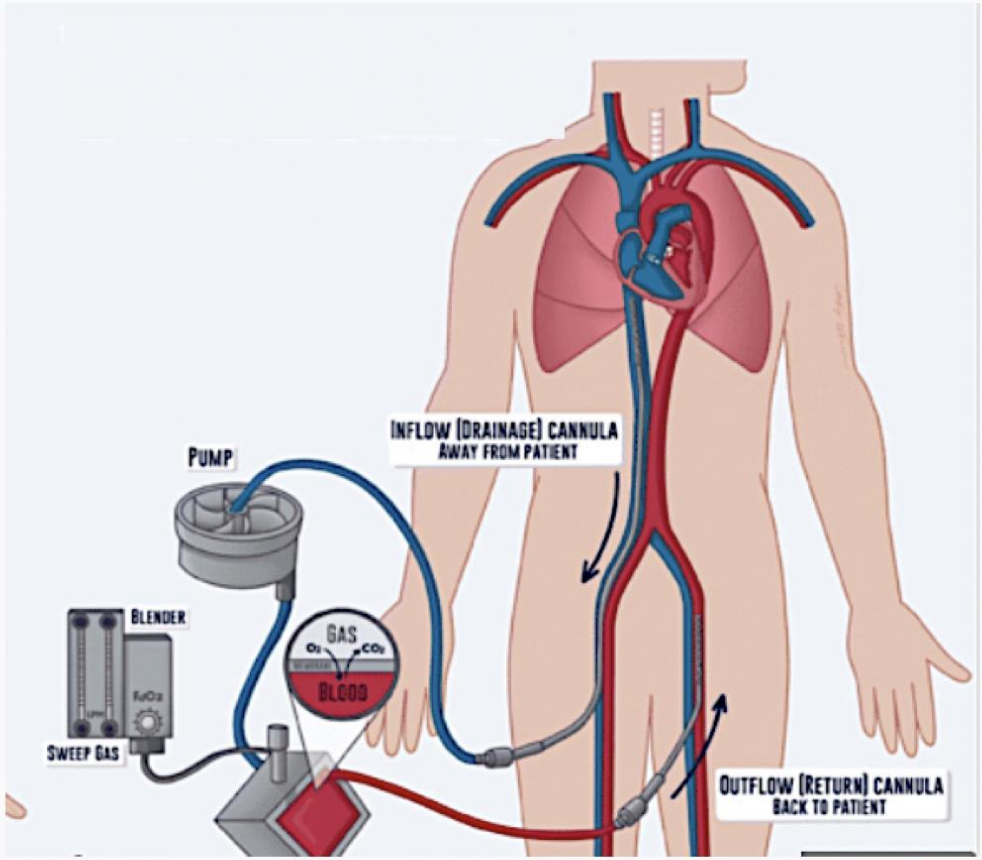
Collet JP, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021 Apr 7;42(14):1289-1367. doi: 10.1093/eurheartj/ehaa575.

Veinoarterial ECMO

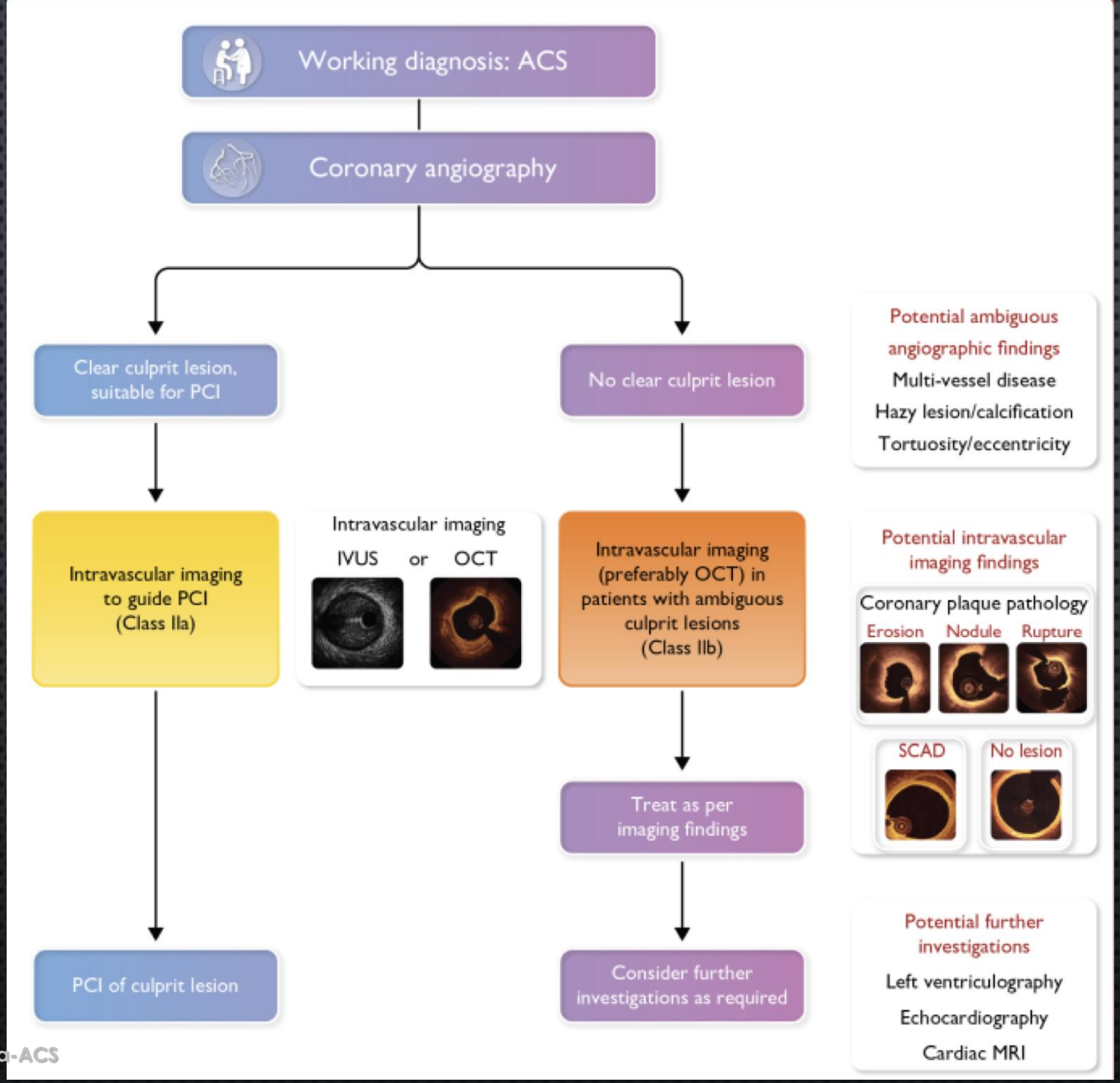
Perform 15 min after call

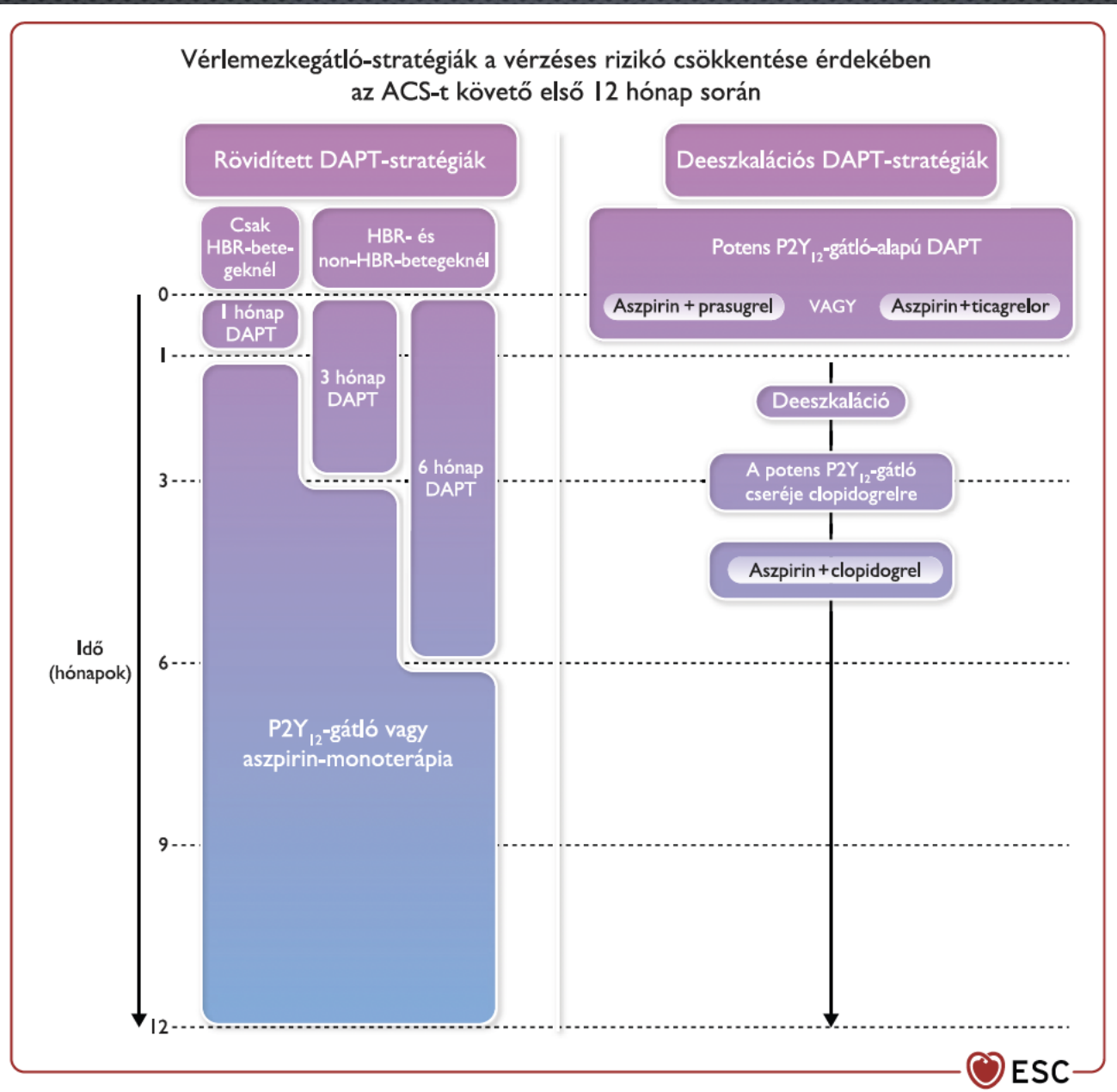
The femoral venous cannula is inserted into the right atrium.

The femoral arterial cannula is left with its tip in the external iliac artery and provides retrograde flow in the aorta. Routine use of an antegrade distal limb perfusion cannula is recommended

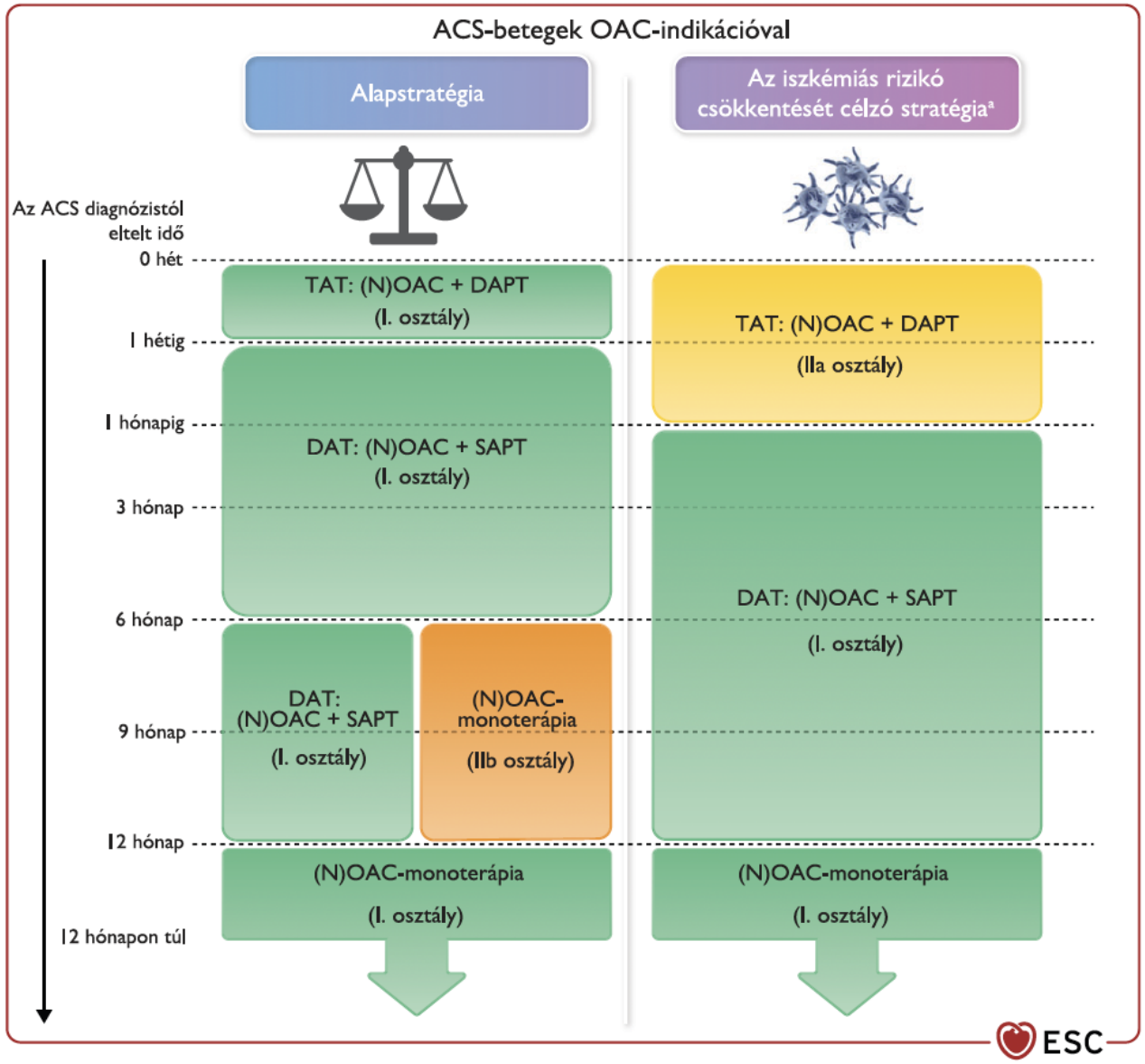


Keringés támogató eszköz használata - ECMO


Working diagnosis: ACS
Coronary angiography
Clear culprit lesion, suitable for PCI
No clear culprit lesion
Potential ambiguous angiographic findings
Multi-vessel disease
Hazy lesion/calcification
Tortuosity/eccentricity
Intravascular imaging to guide PCI (Class IIa)
Intravascular imaging
IVUS or OCT
Intravascular imaging (preferably OCT) in patients with ambiguous culprit lesions (Class IIb)
Potential intravascular imaging findings
Coronary plaque pathology
Erosion
Nodule
Rupture
SCAD
No lesion
Treat as per imaging findings
PCI of culprit lesion
Consider further investigations as required
Potential further investigations
Left ventriculography
Echocardiography
Cardiac MRI

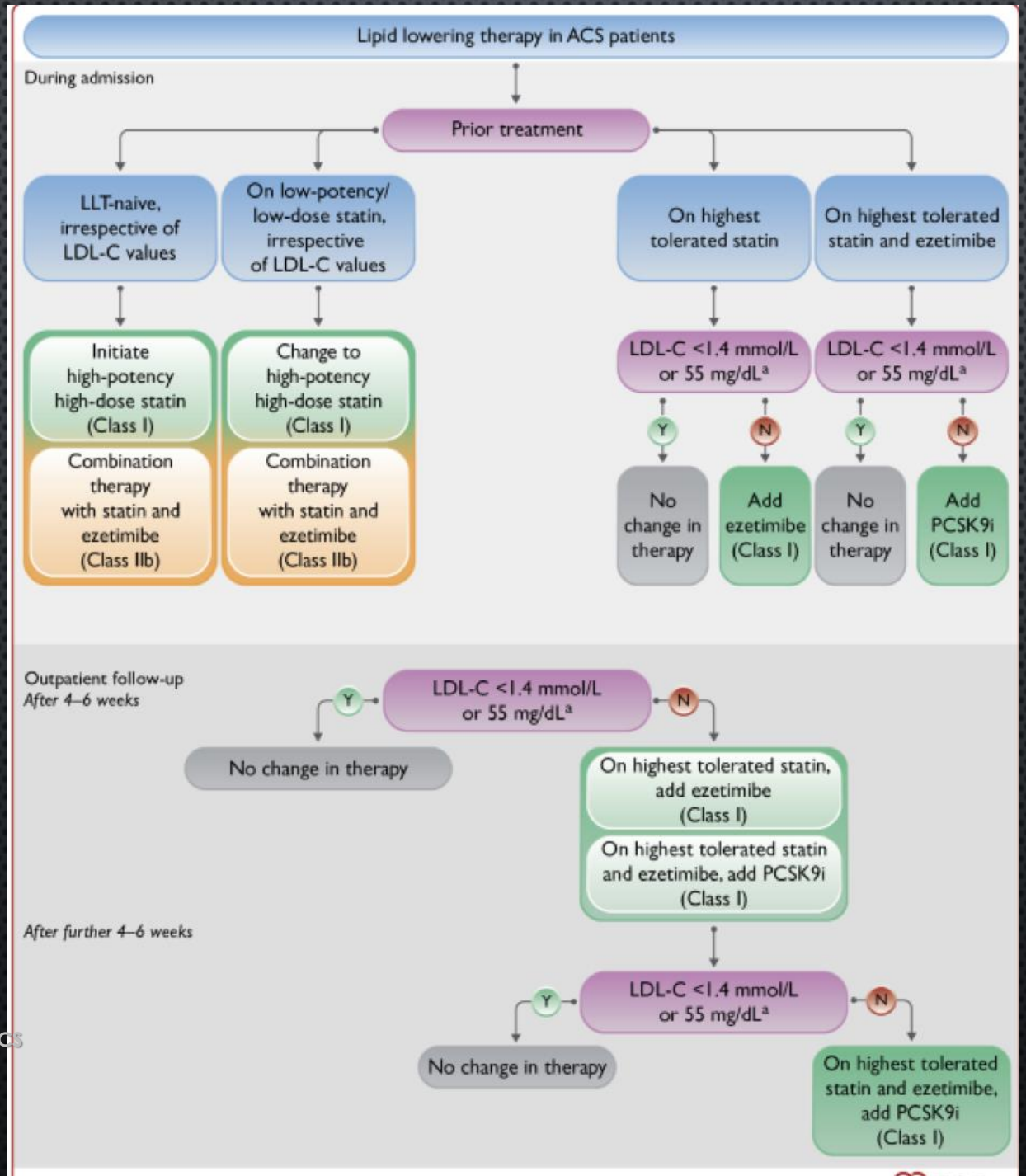


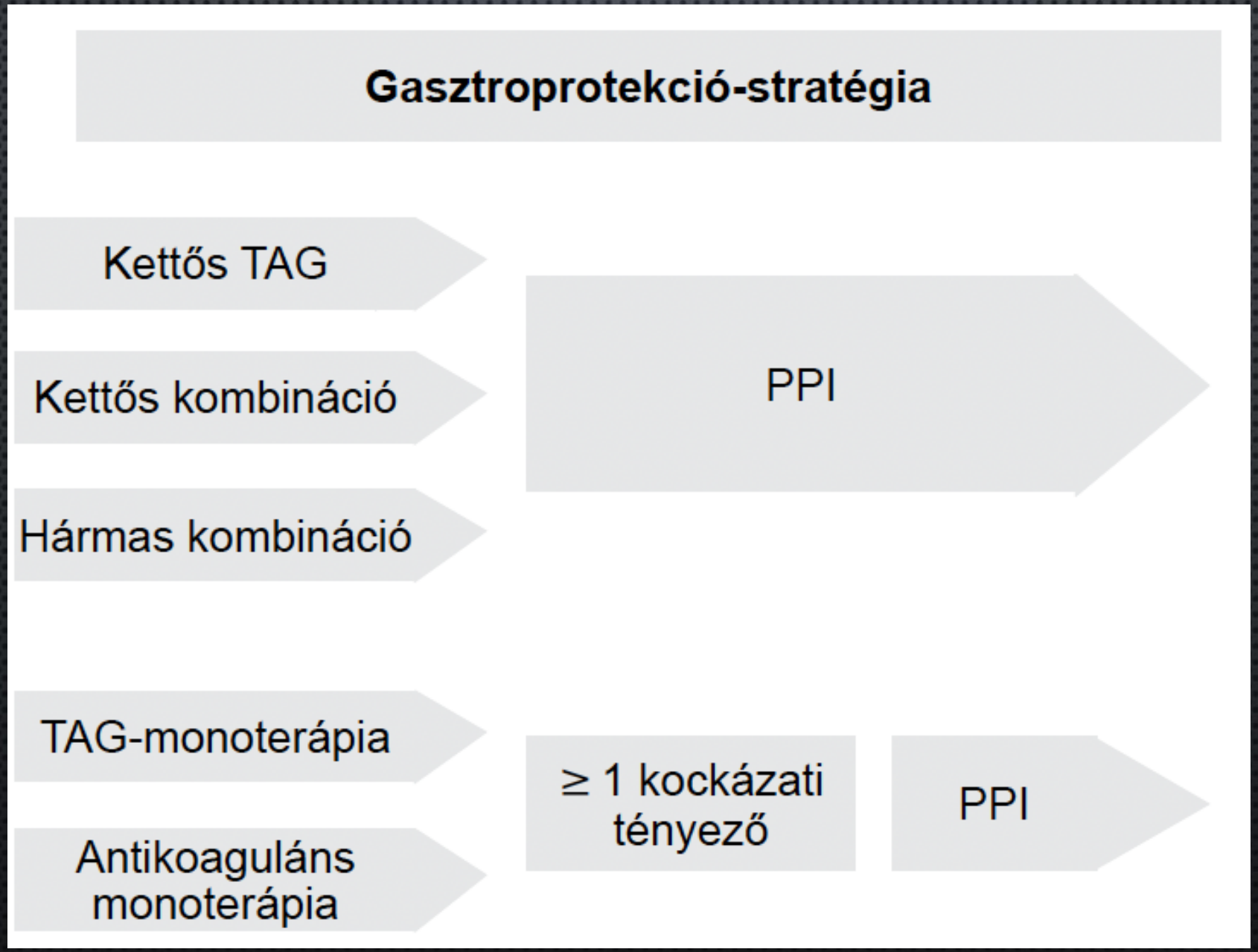
ACS: akut koronáriaszindróma; DAPT: kettős vérlemezkegátló-terápia; HBR: magas vérzéses rizikó; PFT: vérlemezke-funkció

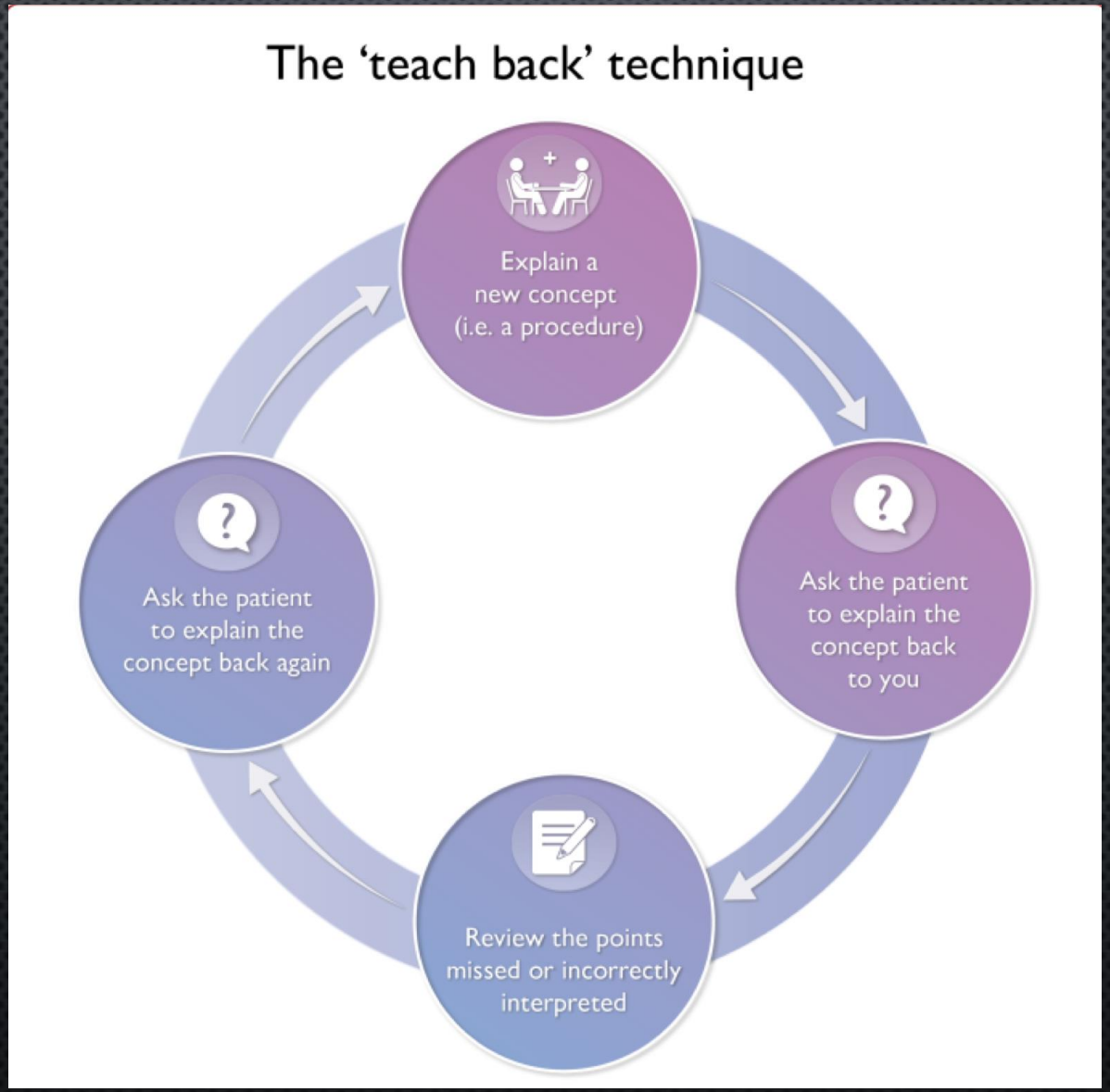


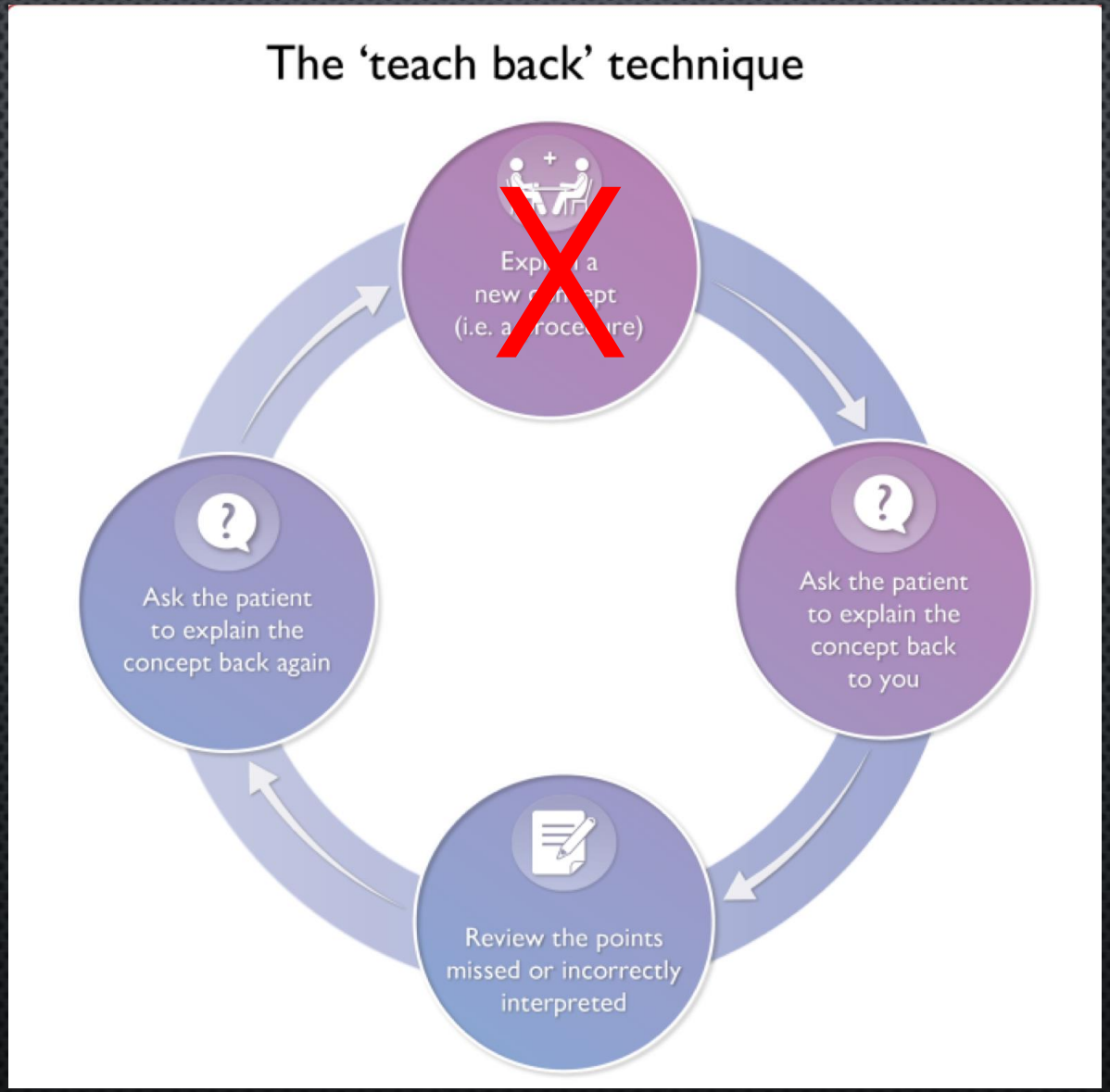
ACS: akut koronáriaszindróma; ARC-HBR: Academic Research Consortium – High Bleeding Risk kritérium; DAPT: kettős vérlemezkegátló-terápia; NOAC: nem K-vitamin-antagonista típusú antikoaguláns; OAC: orális antikoaguláns; SAPT: egyszeres vérlemezkegátló-terápia; TAT: hármas antitrombotikus terápia; VKA: K-vitamin antagonista
Orális antikoaguláns-terápia esetén a NOAC-terápia az alapstratégia a K-vitamin-antagonista-terápiával szemben, ha nincs ellenjavallat. Mind a TAT-, mind a DAT-sémák esetében a NOAC ajánlott dózisai a következők:

- Apixaban 2×5 mg/nap,
- Dabigatran 2×110 mg/nap vagy 2×150 mg/nap,
- Edoxaban 1×60 mg/nap,
- Rivaroxaban 1×15 mg/nap vagy 1×20 mg/nap.


Lipid lowering therapy in ACS patients
During admission
Prior treatment
LLT-naive, irrespective of LDL-C values
On low-potency/ low-dose statin, irrespective of LDL-C values
On highest tolerated statin
On highest tolerated statin and ezetimibe
Initiate high-potency high-dose statin (Class I)
Change to high-potency high-dose statin (Class I)
Combination therapy with statin and ezetimibe (Class IIb)
Combination therapy with statin and ezetimibe (Class IIb)
LDL-C <1.4 mmol/L or 55 mg/dL[a]
LDL-C <1.4 mmol/L or 55 mg/dL[a]
Y
N
Y
N
No change in therapy
Add ezetimibe (Class I)
No change in therapy
Add PCSK9i (Class I)
Outpatient follow-up
After 4–6 weeks
LDL-C <1.4 mmol/L or 55 mg/dL[a]
Y
N
No change in therapy
On highest tolerated statin, add ezetimibe (Class I)
On highest tolerated statin and ezetimibe, add PCSK9i (Class I)
After further 4–6 weeks
LDL-C <1.4 mmol/L or 55 mg/dL[a]
Y
N
No change in therapy
On highest tolerated statin and ezetimibe, add PCSK9i (Class I)


Gasztroprotekció-stratégia
Kettős TAG
Kettős kombináció
Hármas kombináció
PPI
TAG-monoterápia
Antikoaguláns monoterápia
≥ 1 kockázati tényező
PPI


The ‘teach back’ technique
Explain a new concept (i.e. a procedure)
Ask the patient to explain the concept back to you
Review the points missed or incorrectly interpreted
Ask the patient to explain the concept back again


The ‘teach back’ technique
Ask the patient to explain the concept back to you
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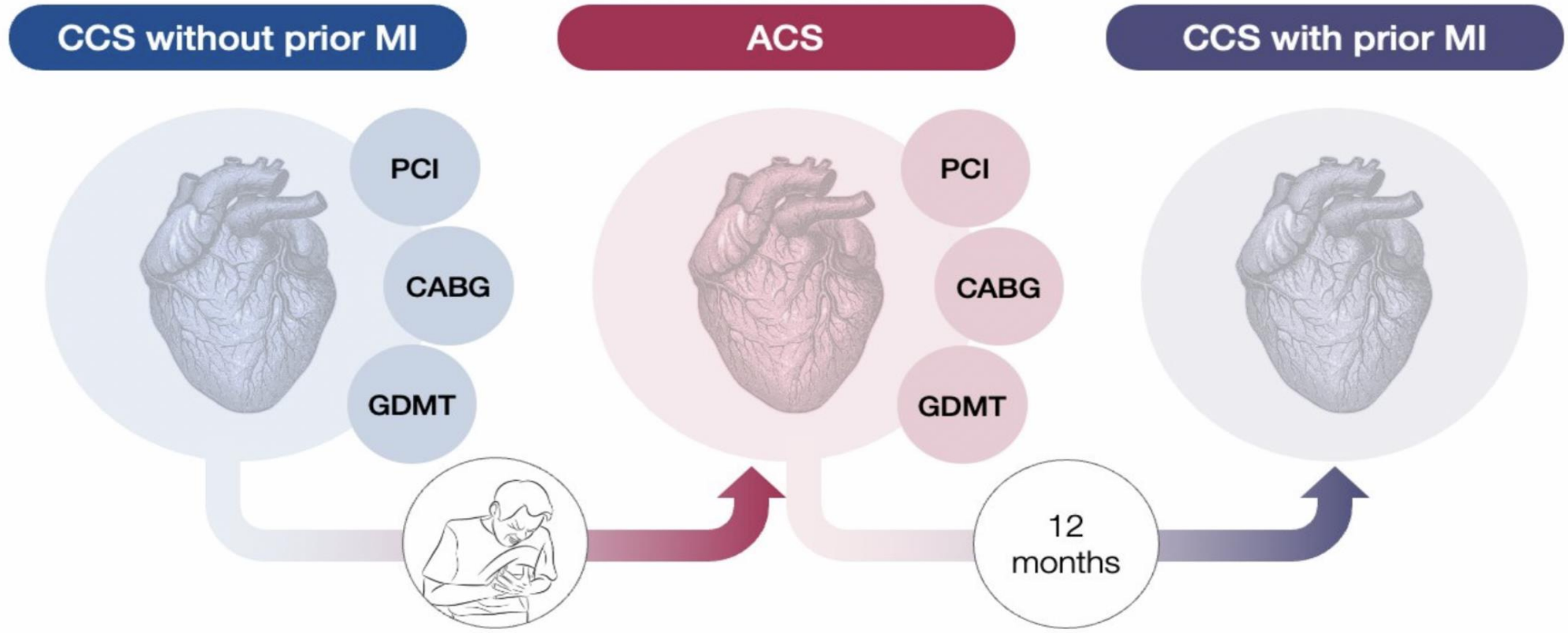
Types of established CAD



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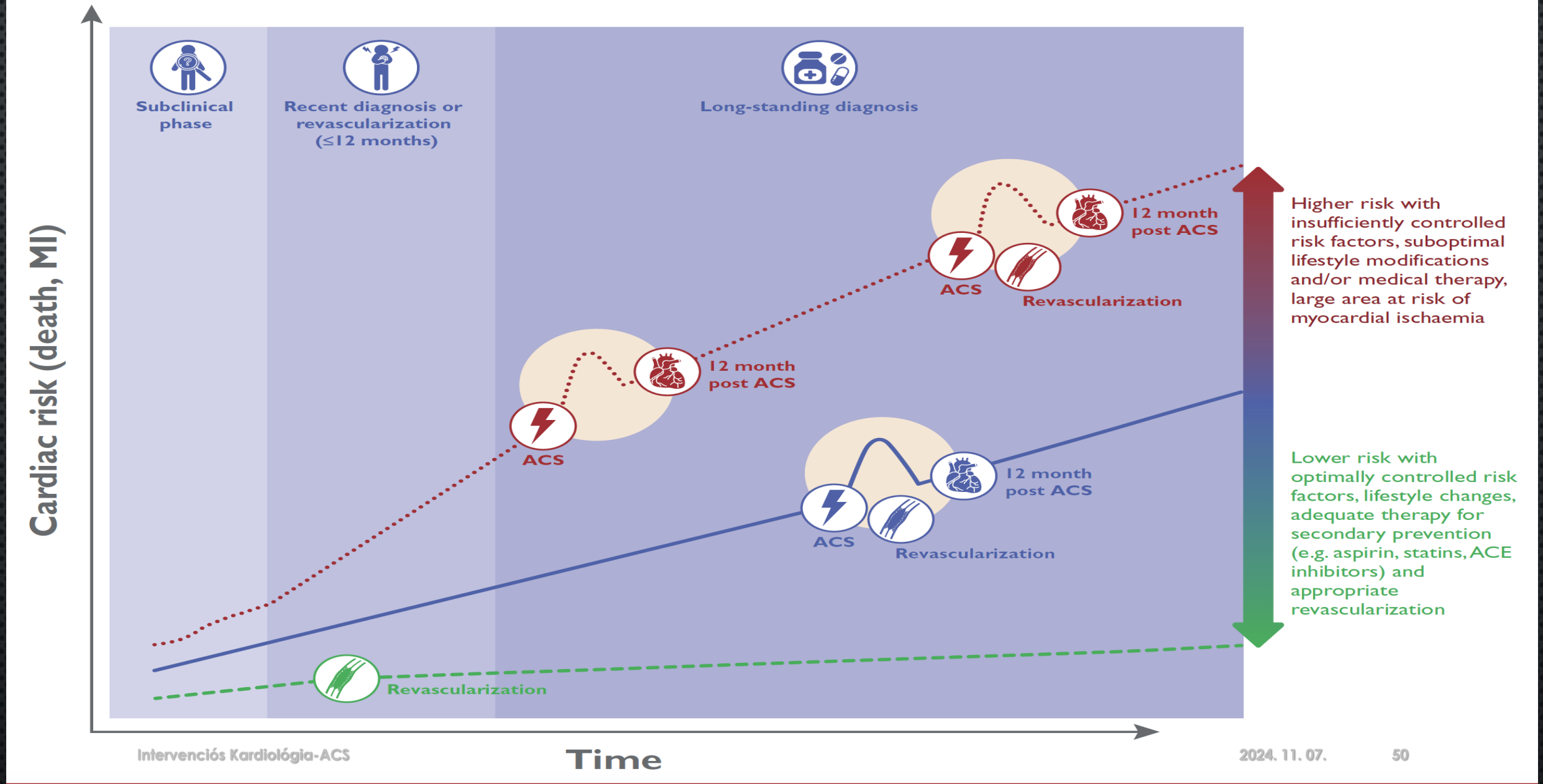
Subclinical phase
Recent diagnosis or revascularization (≤12 months)
Long-standing diagnosis
Cardiac risk (death, MI)
Time
ACS
12 month post ACS
Revascularization
ACS
12 month post ACS
Revascularization
ACS
12 month post ACS
Revascularization
Higher risk with insufficiently controlled risk factors, suboptimal lifestyle modifications and/or medical therapy, large area at risk of myocardial ischaemia
Lower risk with optimally controlled risk factors, lifestyle changes, adequate therapy for secondary prevention (e.g. aspirin, statins, ACE inhibitors) and appropriate revascularization

Metrics

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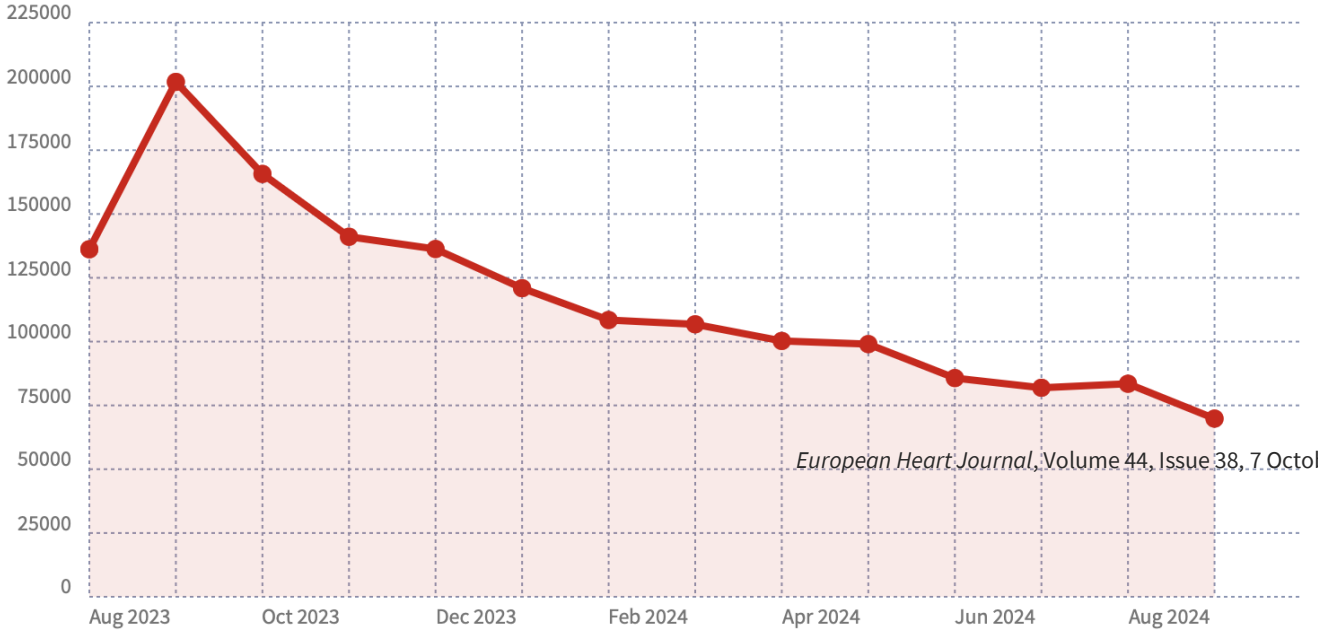

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KÖSZÖNÖM A FIGYELMET!

- "If I have seen further it is by standing on the sholders of giants."