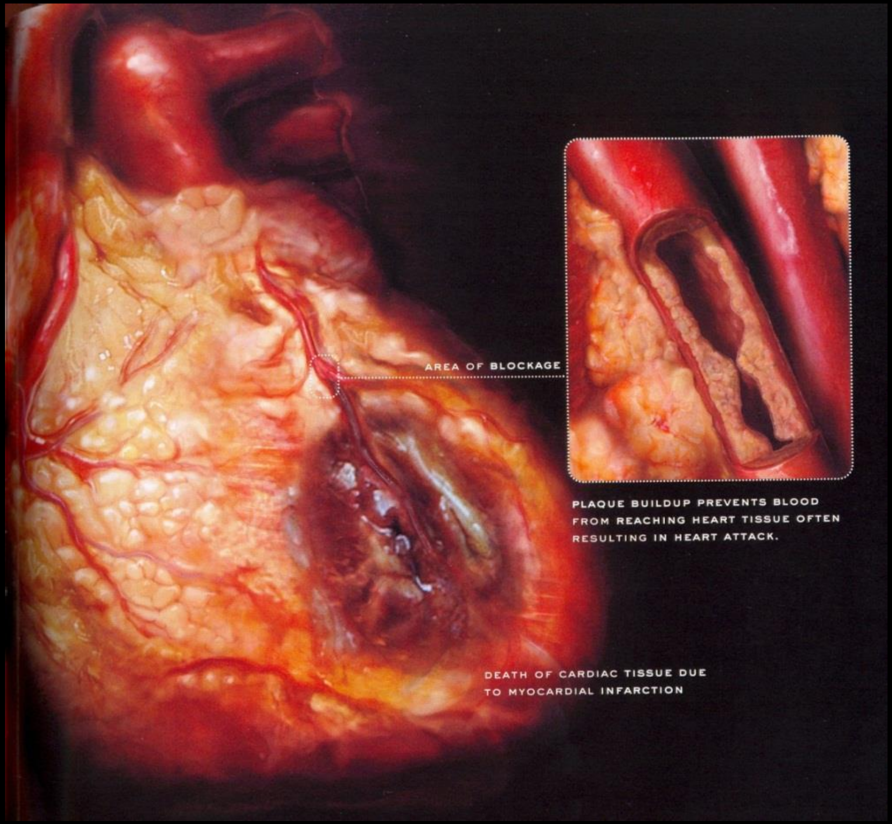
Aktuelles zum Akuten Koronaren Syndrom



PD Dr. med. Gabor Sütsch

ACS

- Grundinformation zum ACS
- Richtlinien: einzelne Aspekte
- Diagnostik
- Fälle

- MINOCA

Main Goal is → reperfusion = restore blood flow

Txt'ing

MI

NSTEMI

MONA * Not in Rt heart infarct

⊕ clopidogrel *

BB * Not in Acute H Failure

* ACEI/ARB

statin

* LMWH – will prevent clot from getting worse

Not in bilat RAS

Not in active bleed

STEMI

MONA

⊕ clopidogrel *

* BB

* ACEI/ARB

statin

* LMWH

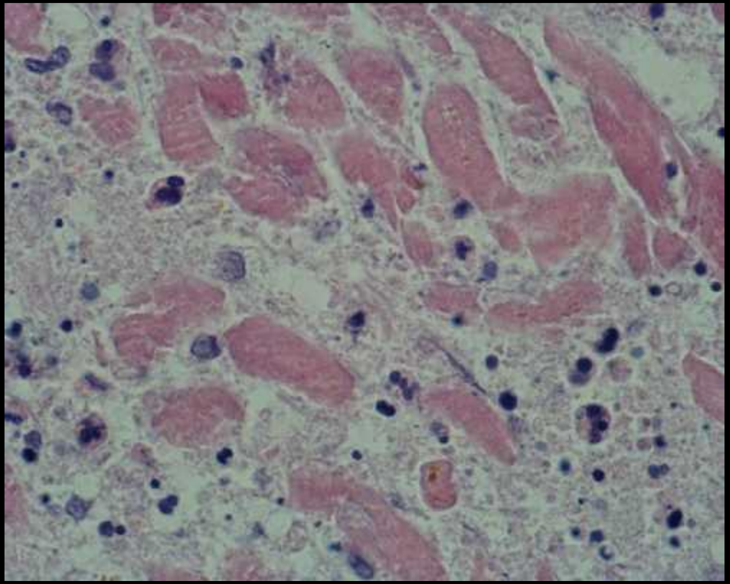
(tPA) thrombolytic vs.

Preferred txt * → PCI ****

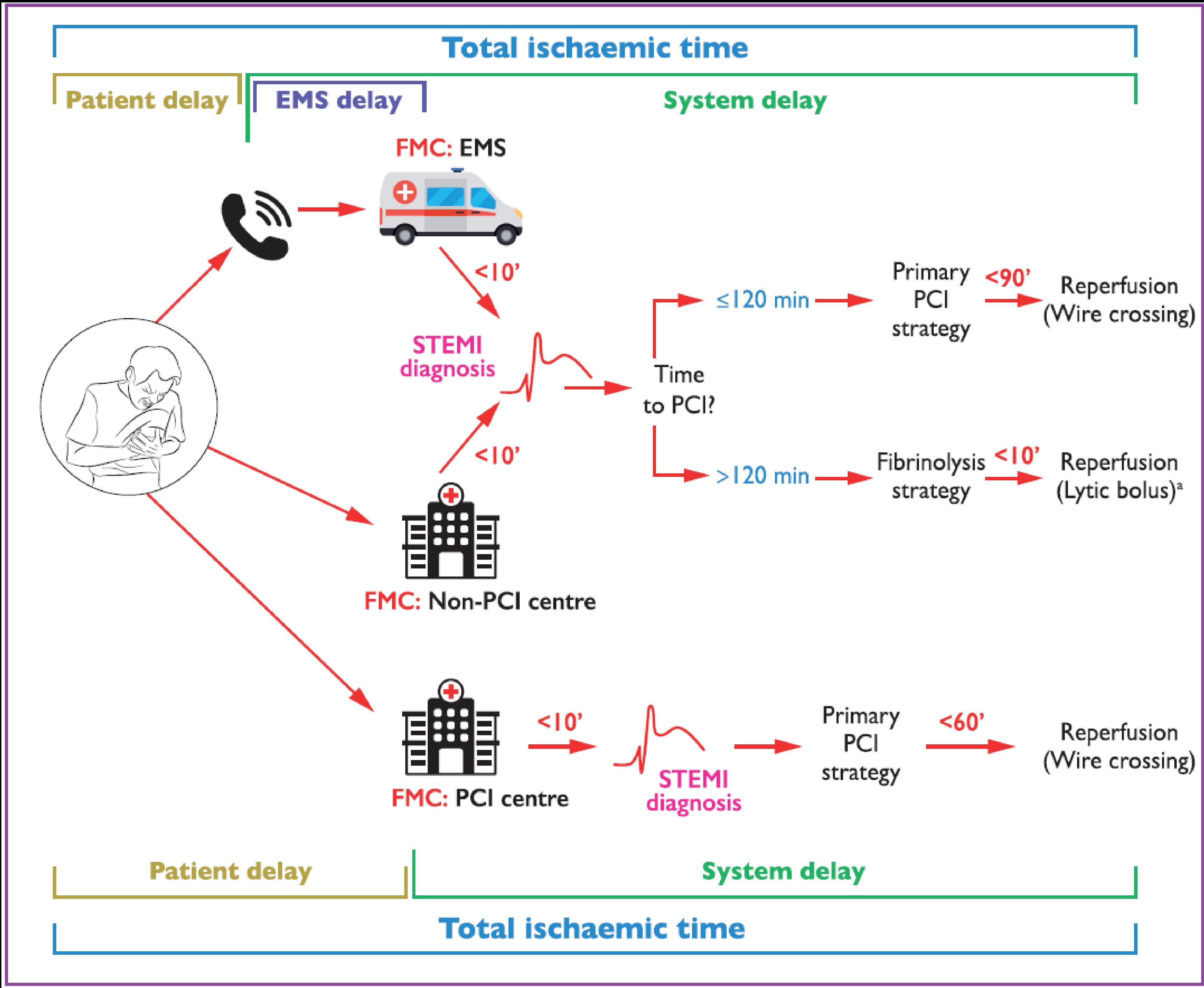
Cath lab? Depends for NSTEMI on TIMI! (risk factor)

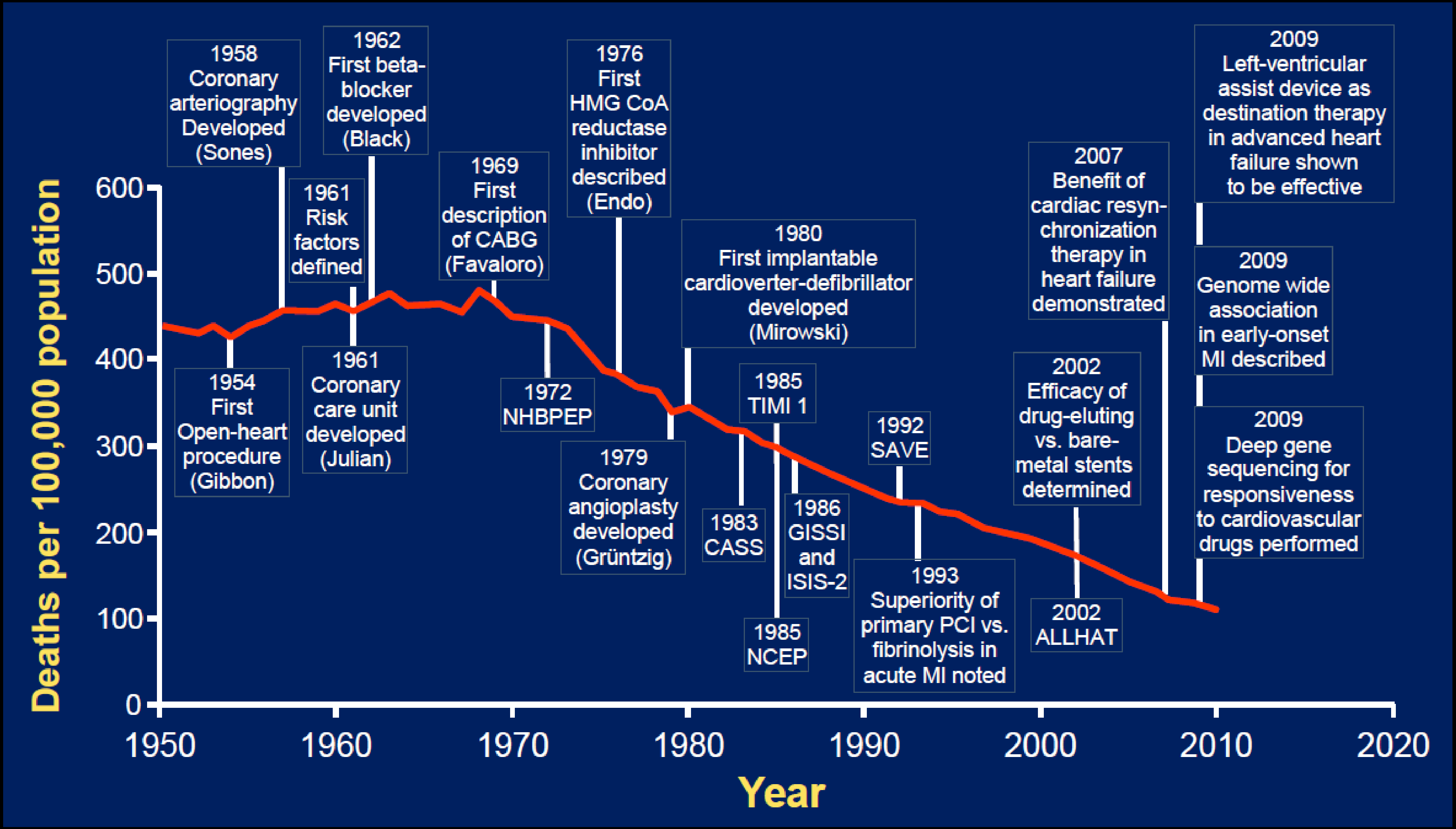
Safet Lekperic

=



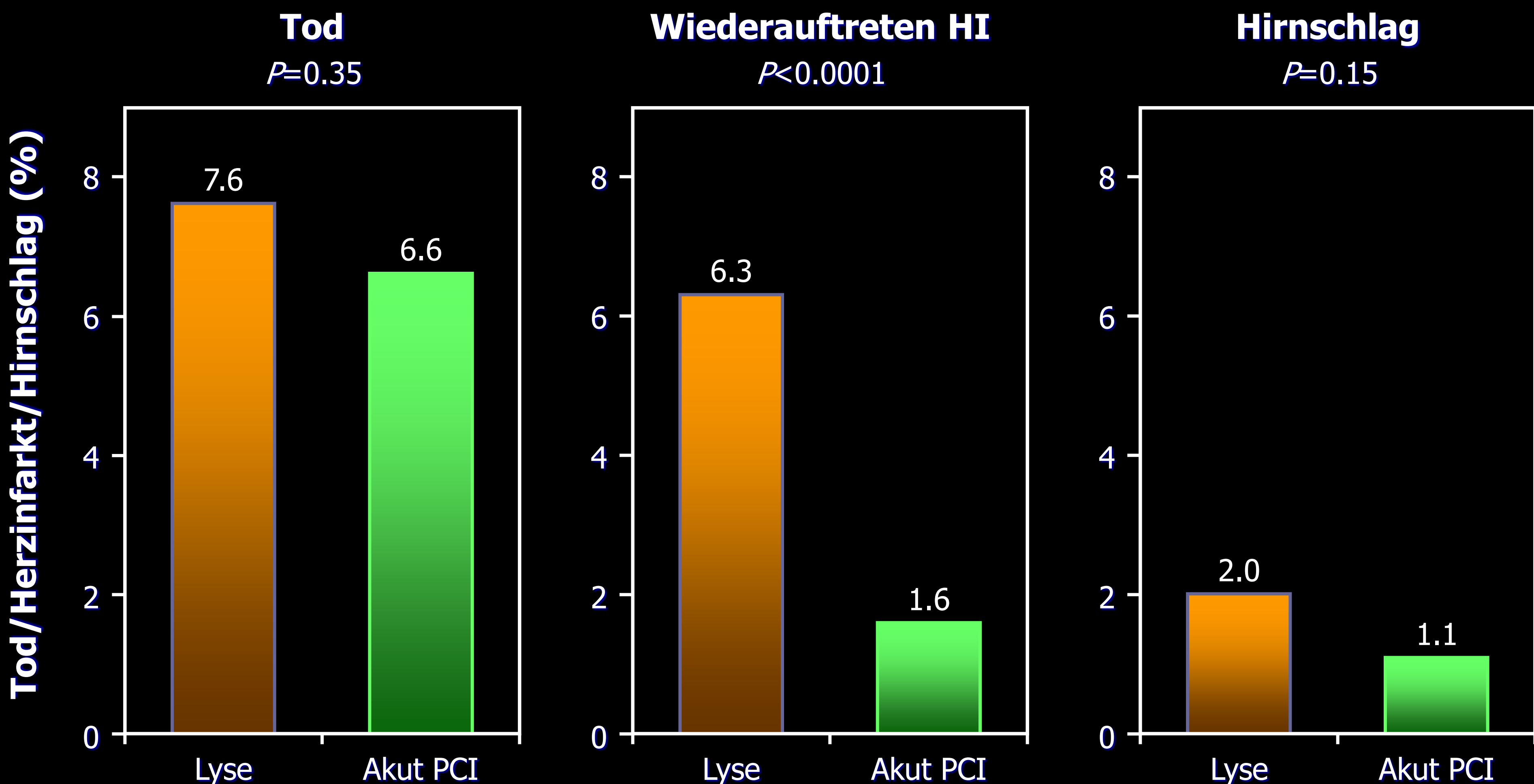
Zeit = Muskel


Total ischaemic time
Patient delay
EMS delay
System delay
FMC: EMS
<10'
STEMI diagnosis
<10'
FMC: Non-PCI centre
Time to PCI?
≤120 min
Primary PCI strategy
<90'
Reperfusion (Wire crossing)
>120 min
Fibrinolysis strategy
<10'
Reperfusion (Lytic bolus)[a]
FMC: PCI centre
<10'
STEMI diagnosis
Primary PCI strategy
<60'
Reperfusion (Wire crossing)
Patient delay
System delay
Total ischaemic time

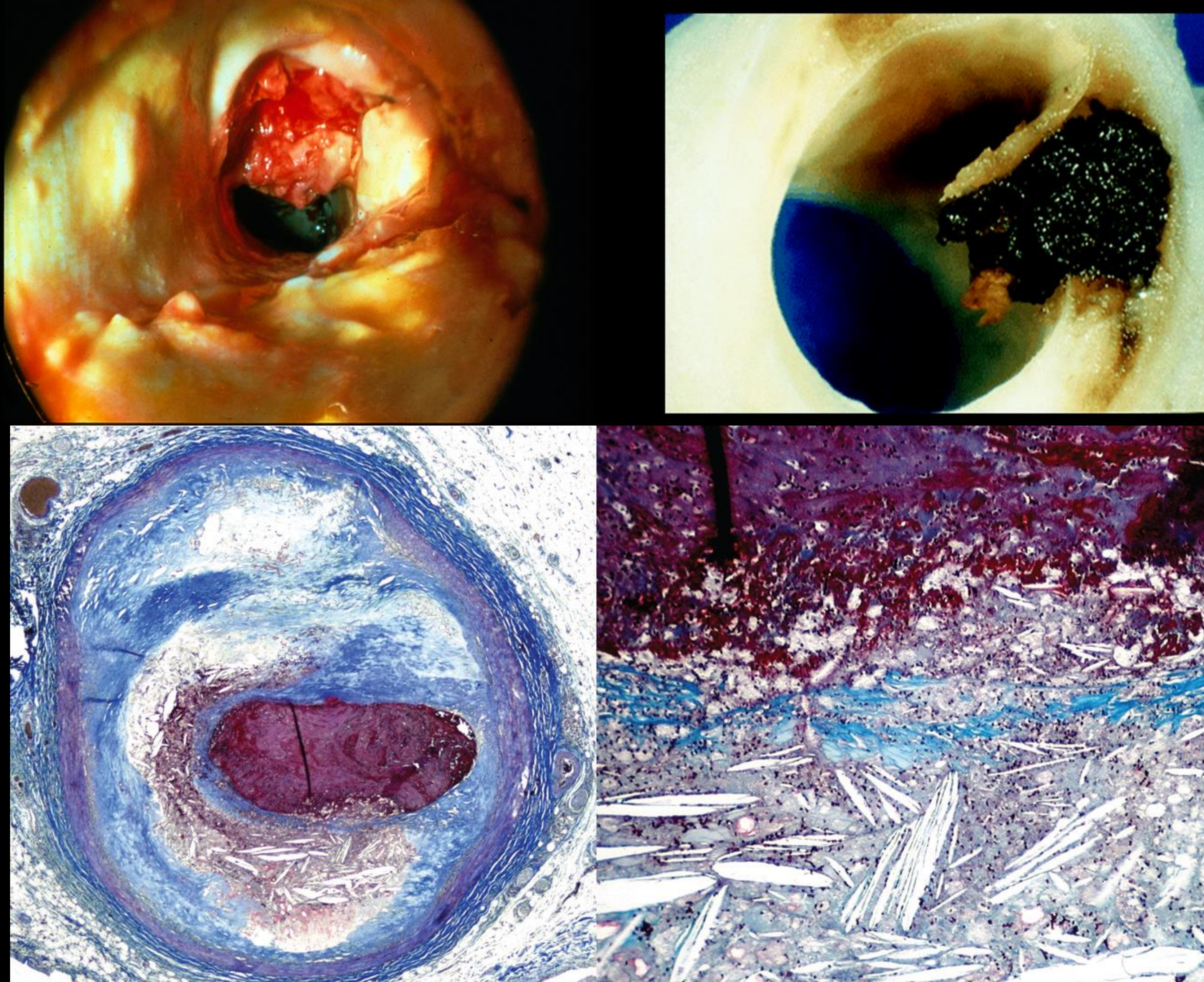


Nabel EG and Braunwald E. 2012;366:54-63

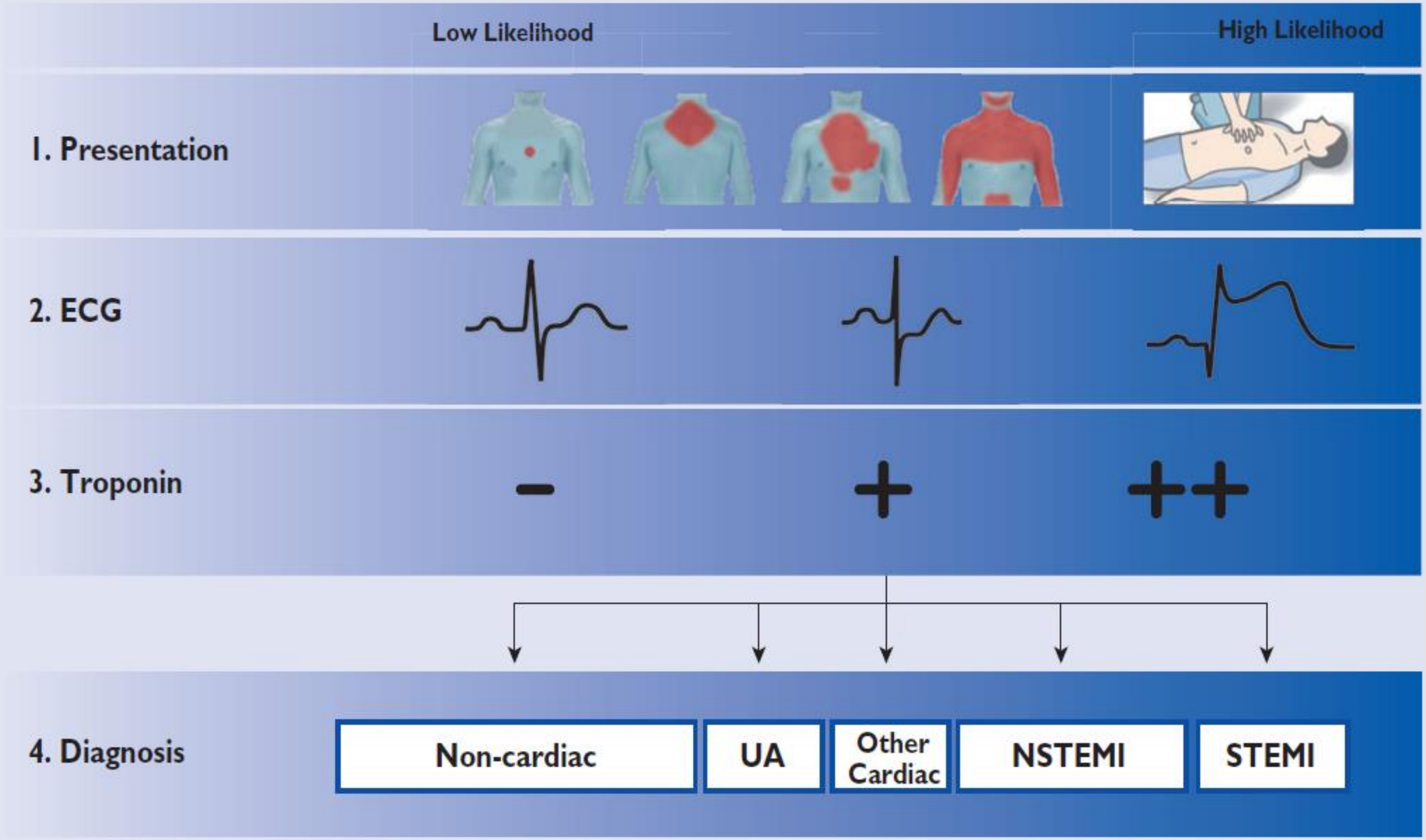
DANAMI-2: Resultate



Anderson HR, et al. *NEJM* 2003;349:733-42

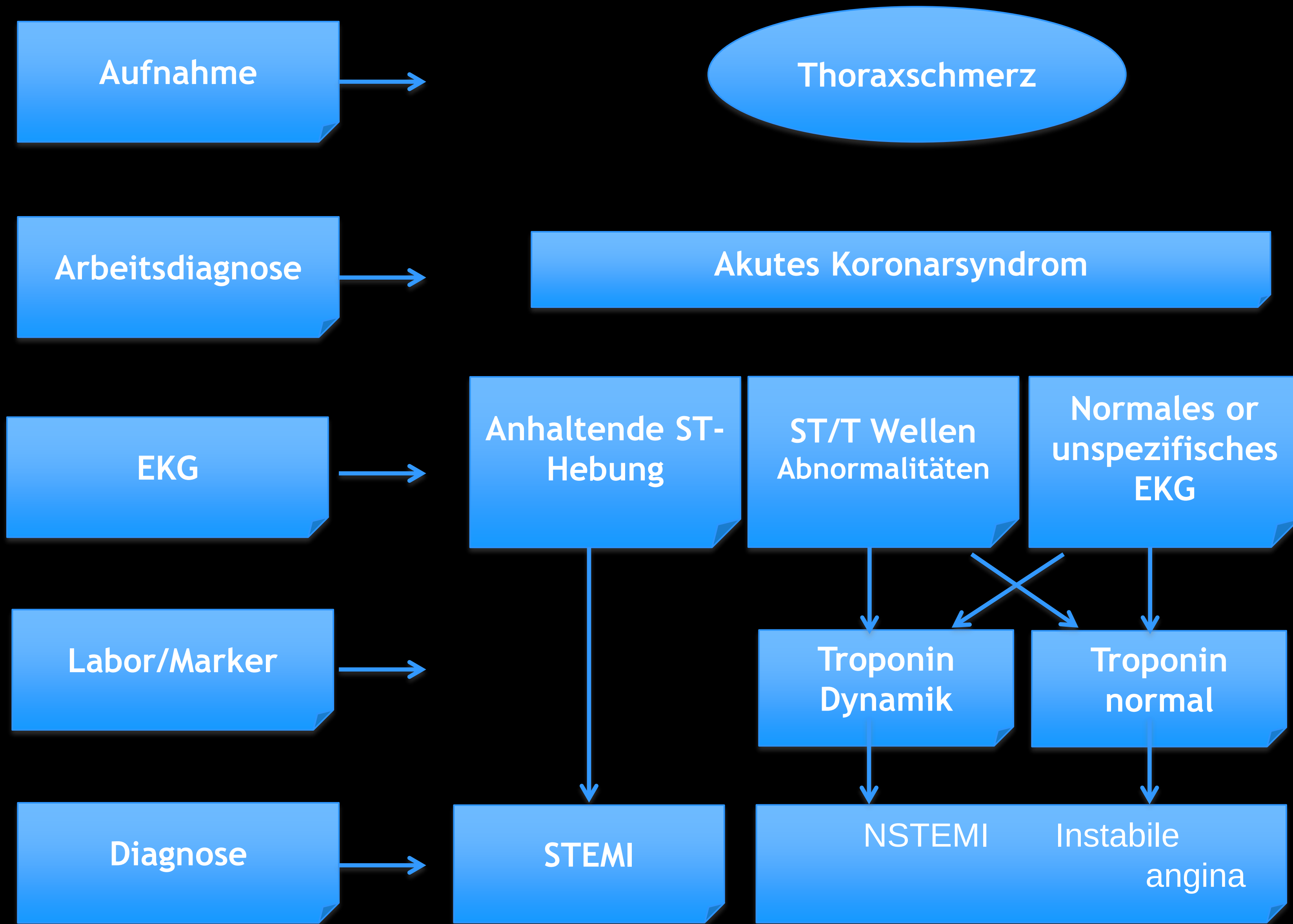


Bentzon JF et al. Circ Res. 2014;114:1852-1866

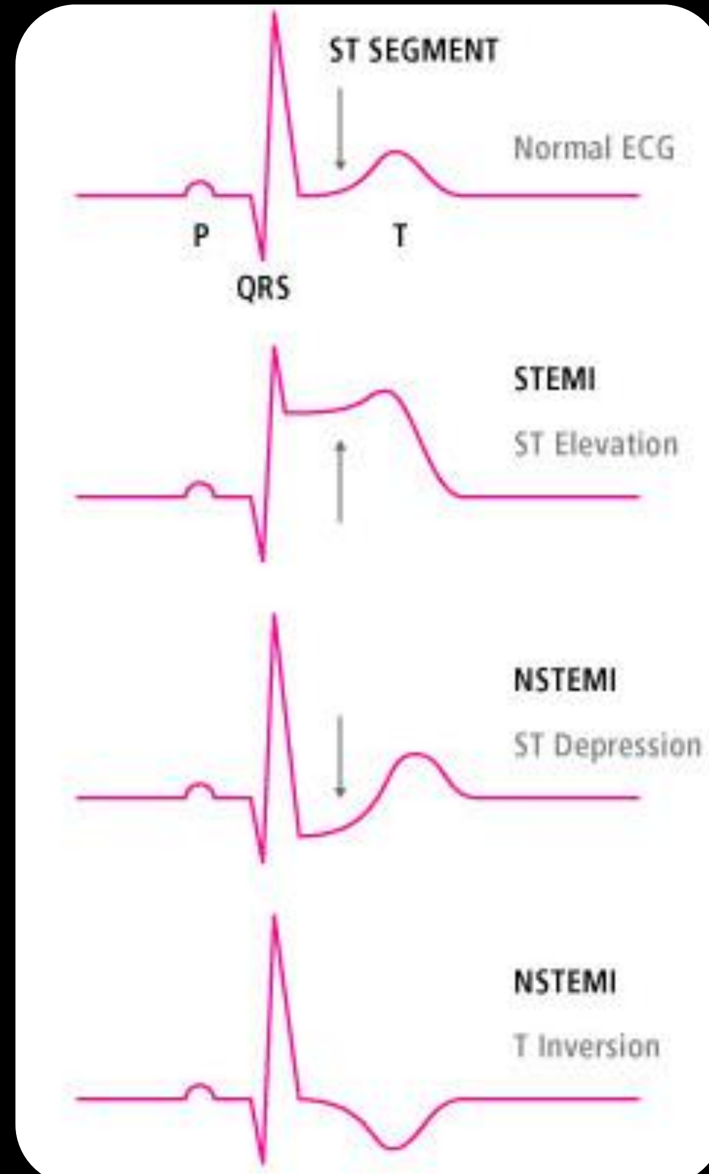


STEMI = ST-elevation myocardial infarction; NSTEMI = non-ST-elevation myocardial infarction; UA = unstable angina.

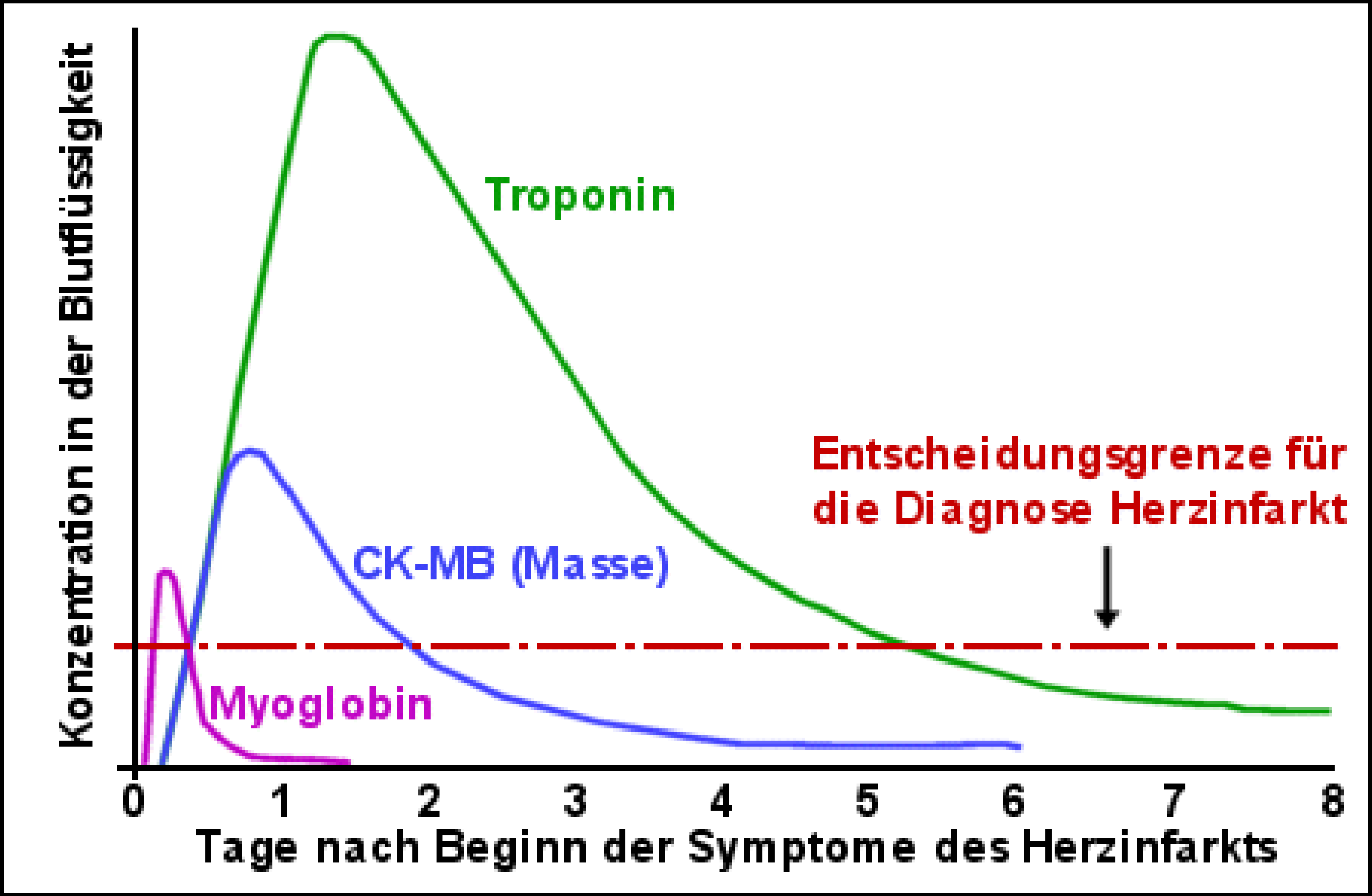
Spektrum des ACS



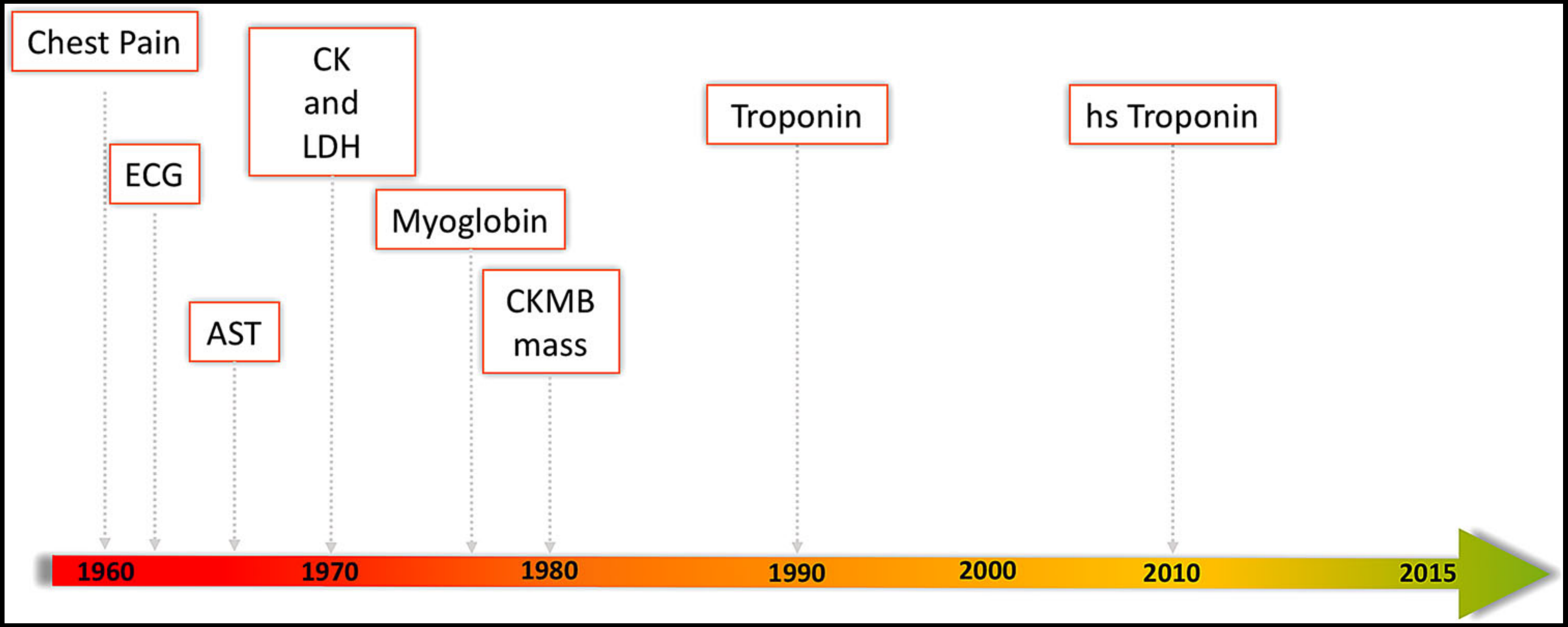
ESC 2011



ST SEGMENT
Normal ECG
P
QRS
T
STEMI
ST Elevation
NSTEMI
ST Depression
NSTEMI
T Inversion


Konzentration in der Blutflüssigkeit
Troponin
Entscheidungsgrenze für
die Diagnose Herzinfarkt
CK-MB (Masse)
Myoglobin
0
1
2
3
4
5
6
7
8
Tage nach Beginn der Symptome des Herzinfarkts

Kardiale Marker beim ACS



Pankaj G et al; Intern Emerg Med (2017) 12:147–155

HIGH-SENSITIVITY CARDIAC TROPONIN I (hs-cTnI) ASSAY

JAMA Cardiology | Brief Report

Validity of a Novel Point-of-Care Troponin Assay for Single-Test Rule-Out of Acute Myocardial Infarction

John W. Pickering, PhD; Joanna M. Young, PhD; Peter M. George, BSc, MBBS; Antony S. Watson, BBiomedSc; Sally J. Aldous, MBBS, ChB, PhD; Richard W. Troughton, MBChB, PhD; Christopher J. Pemberton, PhD; A. Mark Richards, MD, PhD; Louise A. Cullen, MBBS, PhD; Martin P. Than, MBBS

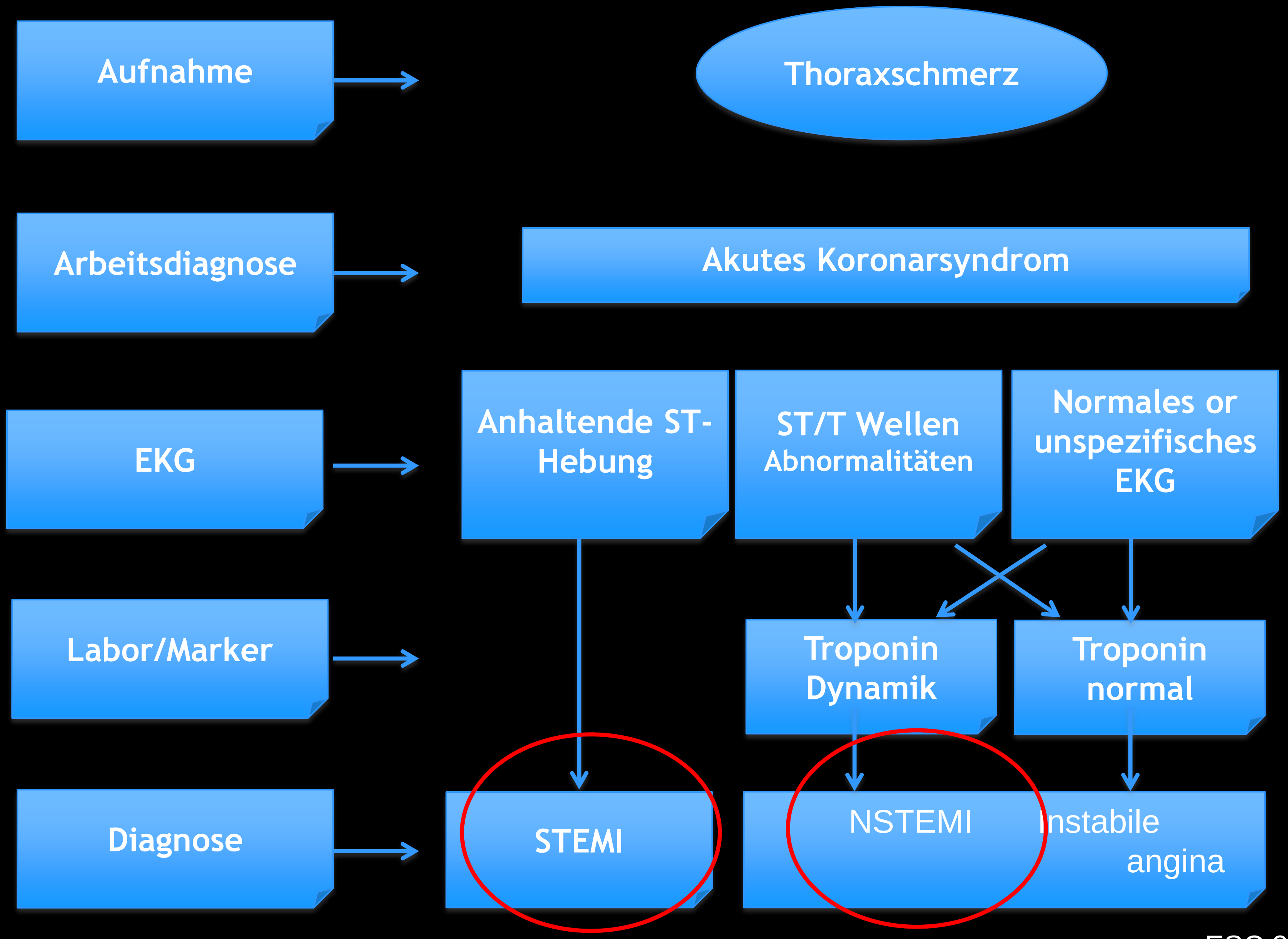
Supplemental cont

IMPORTANCE Emergency department (ED) investigations of patients with suspected acute myocardial infarction (AMI) are time consuming, partly because of the turnaround time of laboratory tests. Current point-of-care troponin assays shorten test turnaround times but lack precision at lower concentrations. Development of point-of-care troponin assays with greater analytical precision could reduce the decision-making time in EDs for ruling out AMI.

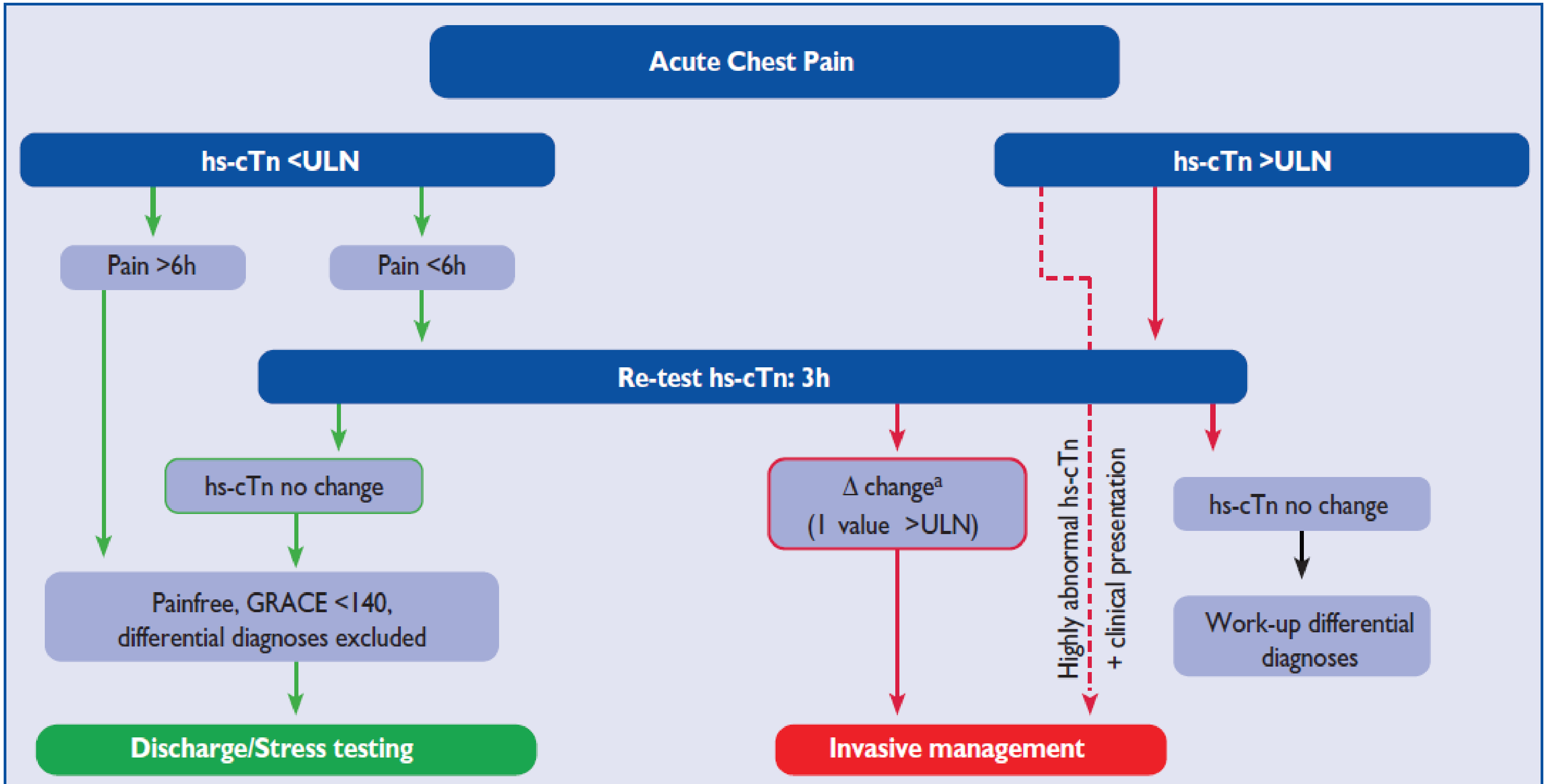
CONCLUSIONS AND RELEVANCE A novel point-of-care troponin assay that can produce a result 15 minutes after blood sampling had comparable discrimination ability to an hs-cTnI assay for ruling out AMI after a single blood test. Use in the ED may facilitate earlier decision making and could expedite the safe discharge of a large proportion of low-risk patients.

JAMA Cardiol. doi:10.1001/jamacardio.2018.3368
Published online October 17, 2018.

Spektrum des ACS



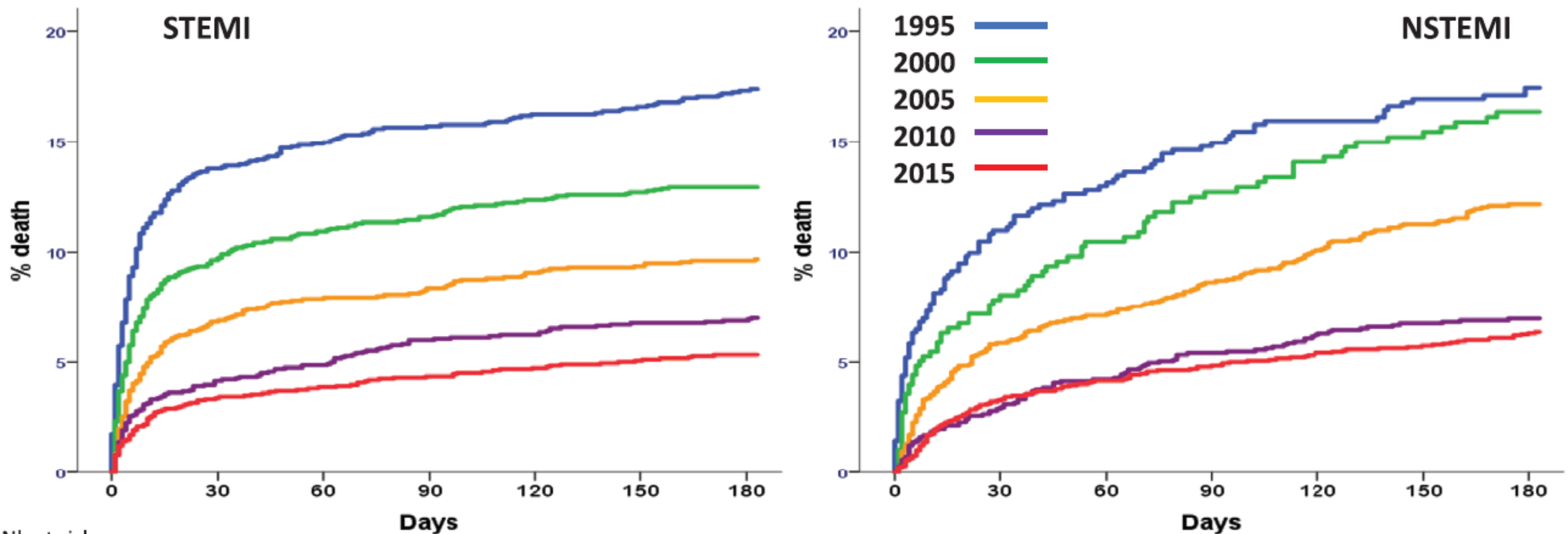
ESC 2011



GRACE = Global Registry of Acute Coronary Events score; hs-cTn = high sensitivity cardiac troponin; ULN = upper limit of normal, 99th percentile of healthy controls.
[a]Δ change, dependent on assay. Highly abnormal hsTn defines values beyond 5-fold the upper limit of normal.

Figure 2 0 h/3 h rule-out algorithm of non-ST-elevation acute coronary syndromes using high-sensitivity cardiac troponin assays.

20-Year Trends in STEMI and NSTEMI



Nb at risk

1995	1536	1268	1249	1233	616	525	507	497
2000	1844	1546	1484	1465	476	402	377	363
2005	1611	1485	1466	1457	1448	1346	1303	1273
2010	1716	1610	1587	1576	1363	1291	1263	1252
2015	1872	1780	1761	1736	1941	1835	1811	1779

Adjusted HR (95%CI) (reference 1995)

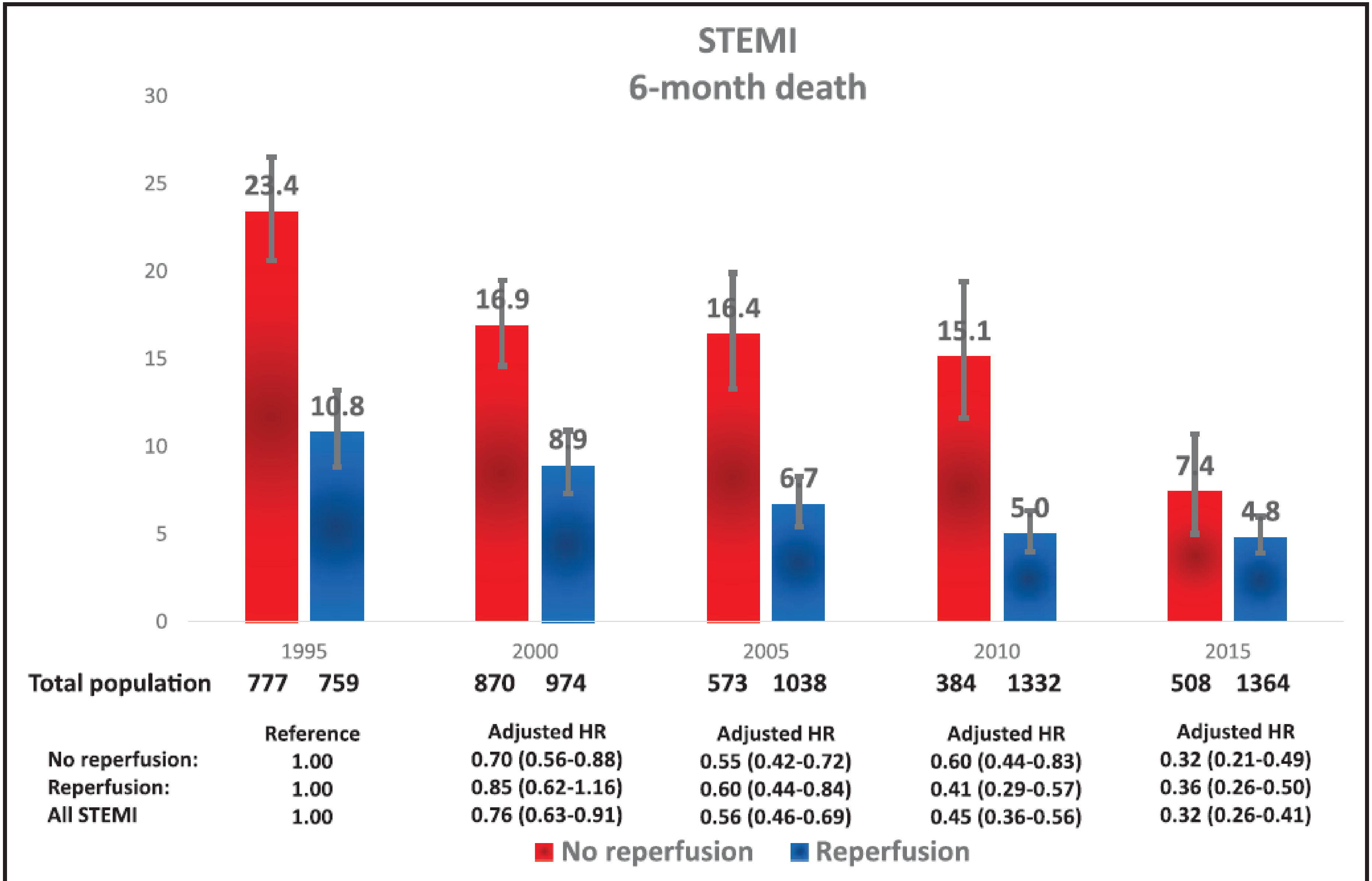
2000: 0.76 (0.63-0.91)
2005: 0.56 (0.46-0.69)
2010: 0.45 (0.36-0.56)
2015: 0.32 (0.26-0.41)

Adjusted HR (95%CI) (reference 1995)

2000: 0.92 (0.67-1.26)
2005: 0.66 (0.51-0.85)
2010: 0.40 (0.30-0.54)
2015: 0.40 (0.30-0.52)

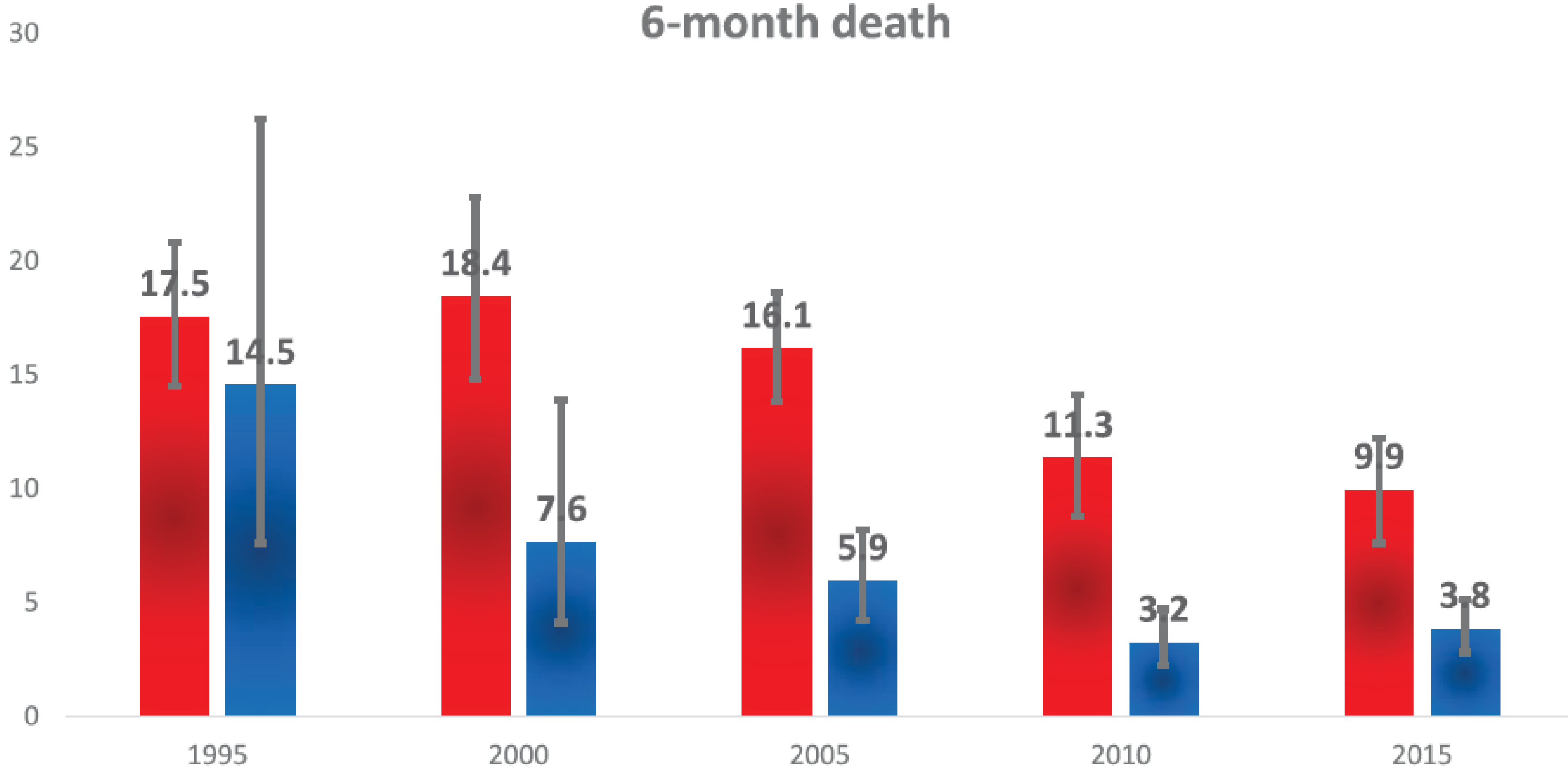
Figure 1. Cumulative 6-month mortality in patients with STEMI and NSTEMI by year of survey.
CI indicates confidence interval; HR, hazard ratio; NSTEMI, non–ST-segment–elevation myocardial infarction; and STEMI, ST-segment–elevation myocardial infarction.

Circulation. 2017;136:1908–1919.



Circulation. 2017;136:1908–1919.

NSTEMI
6-month death



	1995		2000		2005		2010		2015	
Total population	561	55	358	118	890	558	618	745	785	1156
	Reference		Adjusted HR		Adjusted HR		Adjusted HR		Adjusted HR	
No PCI ≤72 hours:	1.00		1.01 (0.73-1.39)		0.74 (0.56-0.97)		0.53 (0.38-0.73)		0.50 (0.36-0.68)	
PCI ≤72 hours:	1.00		0.28 (0.10-0.76)		0.19 (0.08-0.41)		0.09 (0.04-0.21)		0.11 (0.05-0.23)	
All NSTEMI:	1.00		0.92 (0.67-1.26)		0.66 (0.51-0.85)		0.40 (0.30-0.54)		0.40 (0.30-0.52)	

No PCI ≤ 72h PCI ≤ 72h

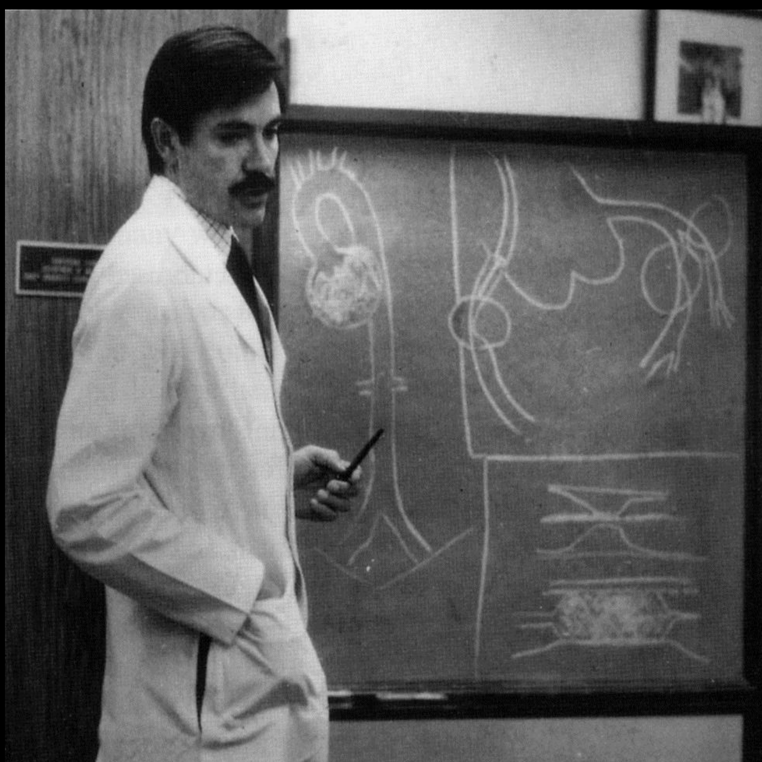
Circulation. 2017;136:1908–1919.

Ziel der Akut-PCI

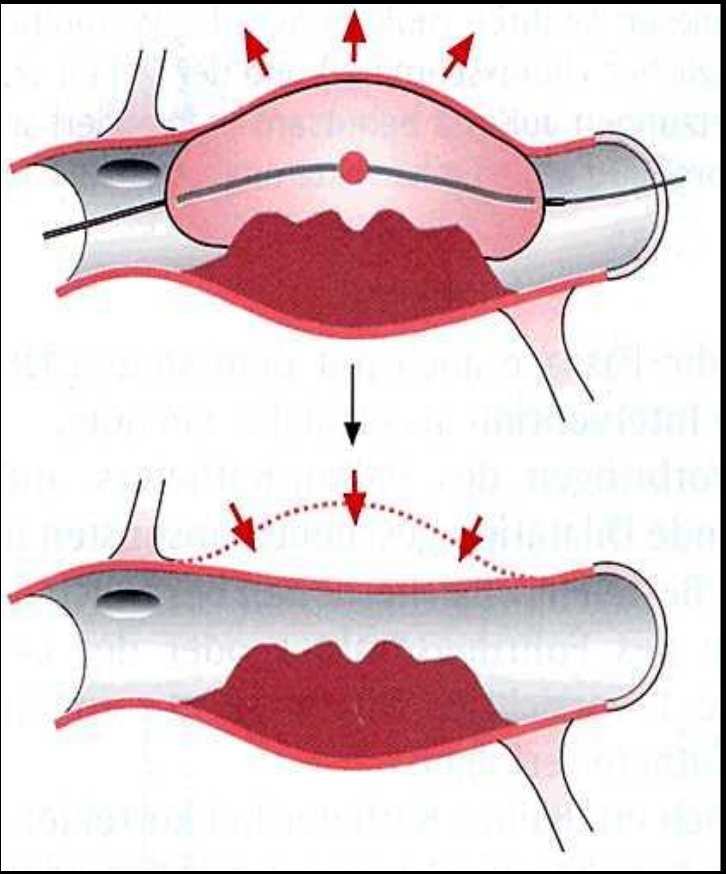
- Befreiung vom Schmerz
- Erhalt des Myokardes
- Reduktion/Verhinderung von Rhythmusstörungen
- Reduktion der Mortalität und Co-Morbiditäten
- Verhinderung von assoziierten Komplikationen
    - u.a. Thrombus, Klappendysfunktion, Ruptur (VSD), Remodeling/Aneurysma, usw.

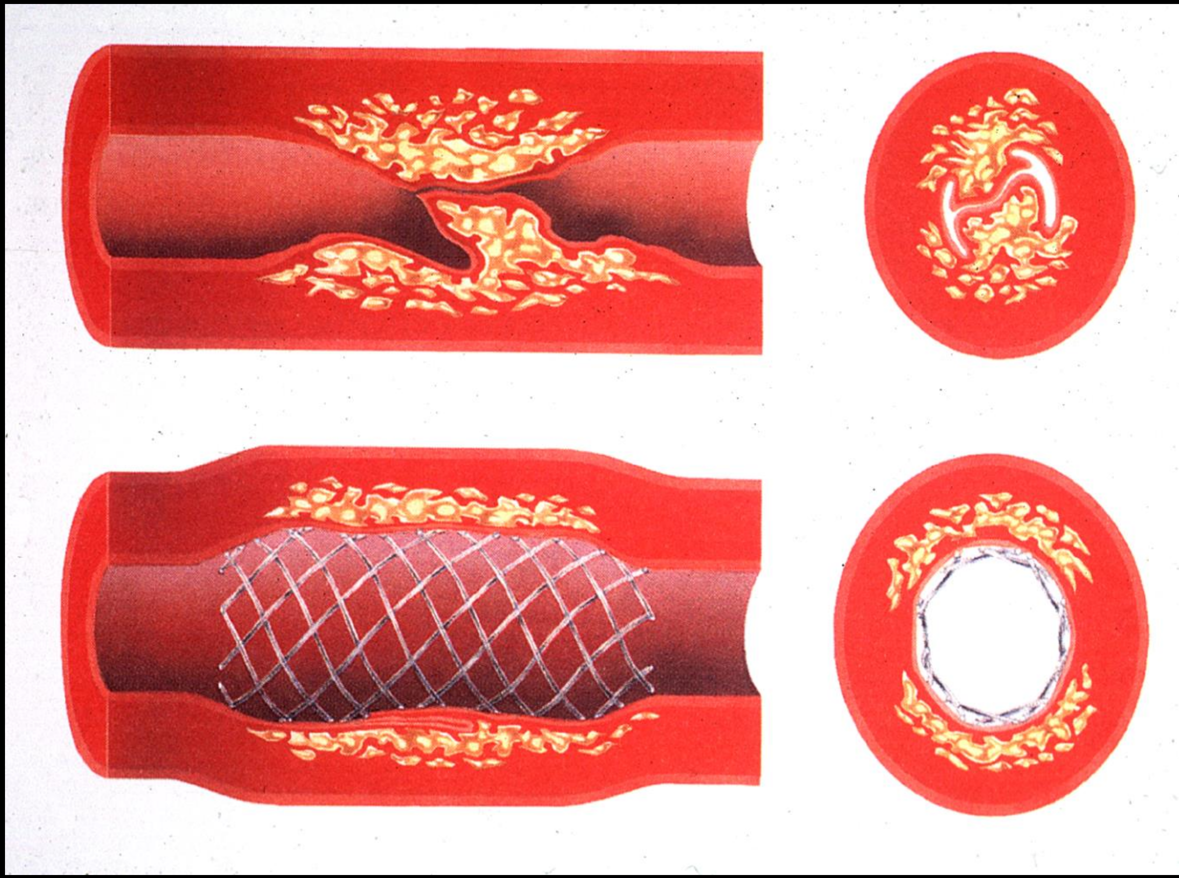
Perkutante koronare Angioplastie (PTCA)

Zürich 1977: Andreas Grüntzig

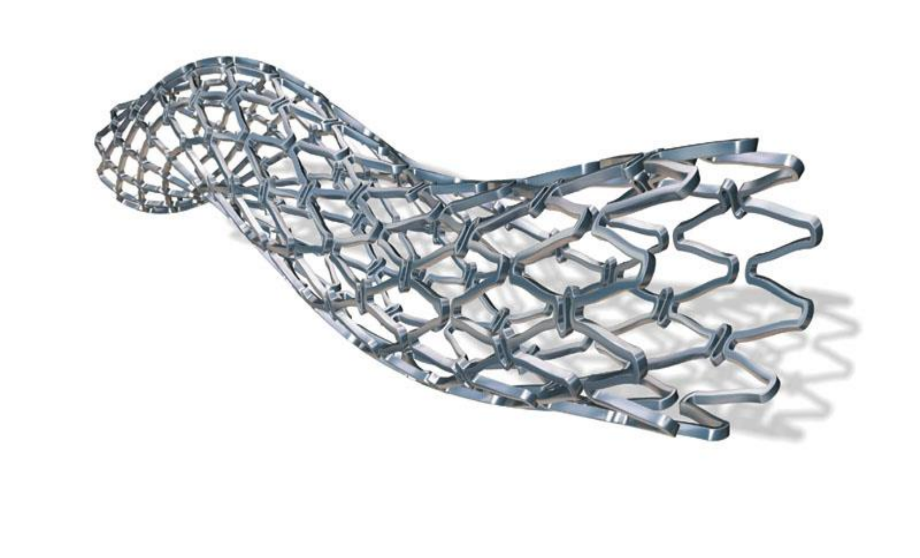


Prinzip der Ballonangioplastie (PTCA) und des Stentings





Stent



CHANGE IN RECOMMENDATIONS

2012 → **2017**

- **Radial access**[a] — MATRIX[143]
- **DES over BMS** — EXAMINATION[150, 151], COMFORTABLE-AMI[149], NORSTENT[152]
- **Complete Revascularization**[b] — PRAMI[168], DANAMI-3-PRIMULTI[170], CVLPRIT[169], Compare-Acute[171]
- **Thrombus Aspiration**[c] — TOTAL[159], TASTE[157]
- **Bivalirudin** — MATRIX[209], HEAT-PPCI[205]
- **Enoxaparin** — ATOLL[200,201], Meta-analysis[202]
- **Early Hospital Discharge**[d] — Small trials & observational data[259–262]
- Oxygen when SaO2 <95% — AVOID[64], DETO2X[66] — Oxygen when SaO2 <90%
- Dose i.V. TNK-tPA same in all patients — STREAM[121] — Dose i.V. TNK-tPA half in Pts ≥75 years

2017 NEW RECOMMENDATIONS

- Additional lipid lowering therapy if LDL >1.8 mmol/L (70 mg/dL) despite on maximum tolerated statins
  IMPROVE-IT[376], FOURIER[382]
- Complete revascularization during index primary PCI in STEMI patients in shock
  Expert opinion
- Cangrelor if $P2Y_{12}$ inhibitors have not been given
  CHAMPION[193]
- Switch to potent $P2Y_{12}$ inhibitors 48 hours after fibrinolysis
  Expert opinion
- Extend Ticagrelor up to 36 months in high-risk patients
  PEGASUS-TIMI 54[333]
- Use of polypill to increase adherence
  FOCUS[323]
- Routine use of deferred stenting
  DANAMI 3-DEFER[155]

I	IIa
IIb	**III**

2017 NEW / REVISED CONCEPTS

MINOCA AND QUALITY INDICATORS:
- New chapters dedicated to these topics.

TIME LIMITS FOR ROUTINE OPENING OF AN IRA[e]:
- 0–12h (Class I); 12–48h (Class IIa); >48h (Class III).

STRATEGY SELECTION AND TIME DELAYS:
- Clear definition of first medical contact (FMC).
- Definition of "time 0" to choose reperfusion strategy (i.e. the strategy clock starts at the time of "STEMI diagnosis").
- Selection of PCI over fibrinolysis: when anticipated delay from "STEMI diagnosis" to wire crossing is ≤120 min.
- Maximum delay time from "STEMI diagnosis" to bolus of fibrinolysis agent is set in 10 min.
- "Door-to-Ballon" term eliminated from guidelines.

ELECTROCARDIOGRAM AT PRESENTATION:
- Left and right bundle branch block considered equal for recommending urgent angiography if ischemic symptoms.

TIME TO ANGIOGRAPHY AFTER FIBRINOLYSIS:
- Timeframe is set in 2–24h after successful fibrinolysis.

PATIENTS TAKING ANTICOAGULANTS:
- Acute and chronic management presented.

CHANGE IN RECOMMENDATIONS

2012 → 2017

Radial access[a]
MATRIX[143]

DES over BMS
EXAMINATION[150, 151]
COMFORTABLE-AMI[149], NORSTENT[152]

Complete Revascularization[b]
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MATRIX[209], HEAT-PPCI[205]

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2012		2017
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I | IIa | IIb | III

2017 NEW / REVISED CONCEPTS

MINOCA AND QUALITY INDICATORS:

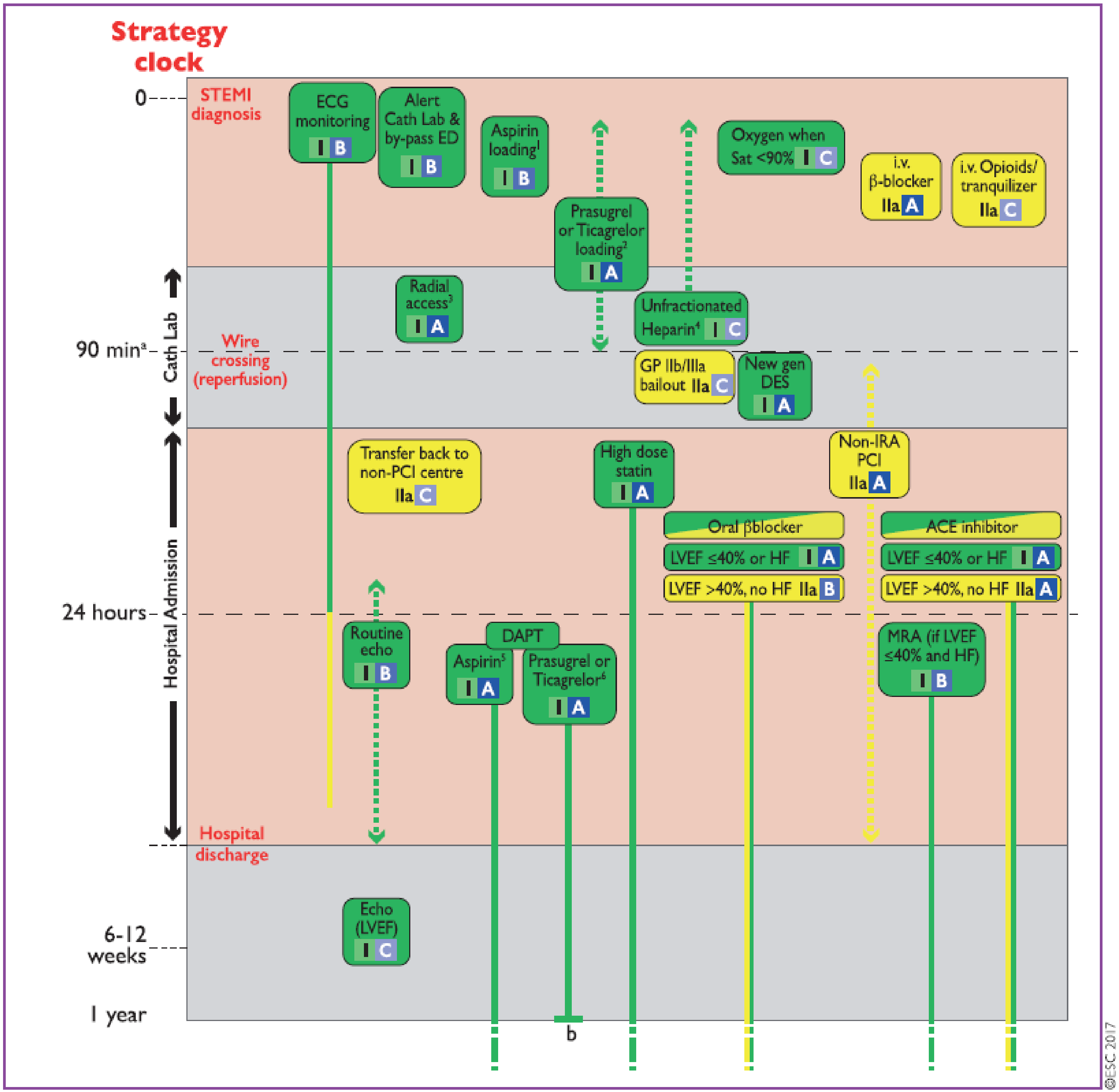
TIME LIMITS FOR ROUTINE OPENING OF AN IRA[e]:

Relief of hypoxaemia and symptoms

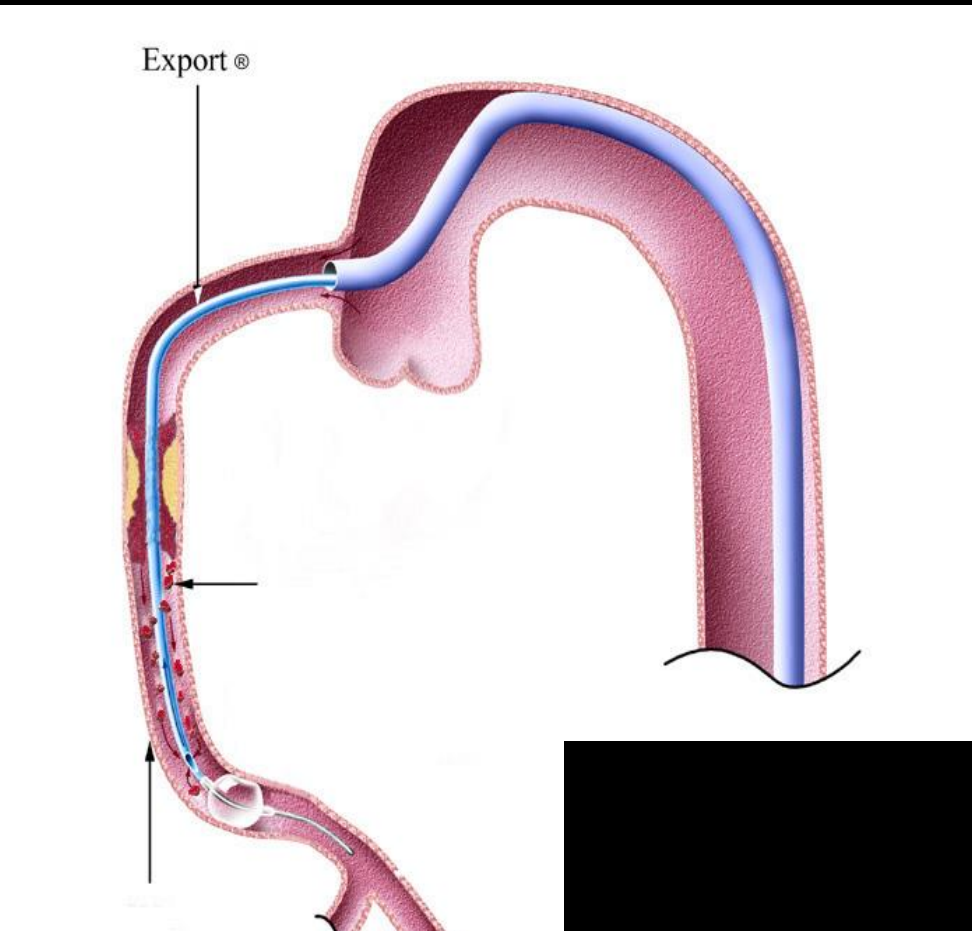
Recommendations	Class[a]	Level[b]
Hypoxia		
Oxygen is indicated in patients with hypoxaemia ($SaO_2 < 90\%$ or $PaO_2 < 60$ mmHg).	I	C
Routine oxygen is not recommended in patients with $SaO_2 \geq 90\%$.[64–66]	III	B
Symptoms		
Titrated i.v. opioids should be considered to relieve pain.	IIa	C
A mild tranquillizer (usually a benzodiazepine) should be considered in very anxious patients.	IIa	C

	Clopidogrel	Prasugrel	Ticagrelor	Cangrelor
Chemical class	Thienopyridine	Thienopyridine	Cyclopentyl-triazolopyrimidine	Stabilized ATP analogue
Administration	Oral	Oral	Oral	Intravenous
Dose	300–600 mg orally then 75 mg a day	60 mg orally then 10 mg a day	180 mg orally then 90 mg twice a day	30 μg/kg bolus and 4 μg/kg/min infusion
Dosing in CKD				
• Stage 3 (eGFR 30–59 mL/min/1.73m^2)	No dose adjustment	No dose adjustment	No dose adjustment	No dose adjustment
• Stage 4 (eGFR 15–29 mL/min/1.73m^2)	No dose adjustment	No dose adjustment	No dose adjustment	No dose adjustment
• Stage 5 (eGFR <15 mL/min/1.73m^2)	Use only for selected indications (e.g. stent thrombosis prevention)	Not recommended	Not recommended	No dose adjustment
Binding reversibility	Irreversible	Irreversible	Reversible	Reversible
Activation	Prodrug, with variable liver metabolism	Prodrug, with predictable liver metabolism	Active drug, with additional active metabolite	Active drug
Onset of loading dose effect[a]	2–6 hours[b]	30 min[b]	30 min[b]	2 min
Duration of effect	3–10 days	7–10 days	3–5 days	1–2 hours
Withdrawal before surgery	5 days[c]	7 days[c]	5 days[c]	1 hour
Plasma half-life of active $P2Y_{12}$ inhibitor[d]	30–60 min	30–60 min[e]	6–12 hours	5–10 min
Inhibition of adenosine reuptake	No	No	Yes	Yes ('inactive' metabolite only)

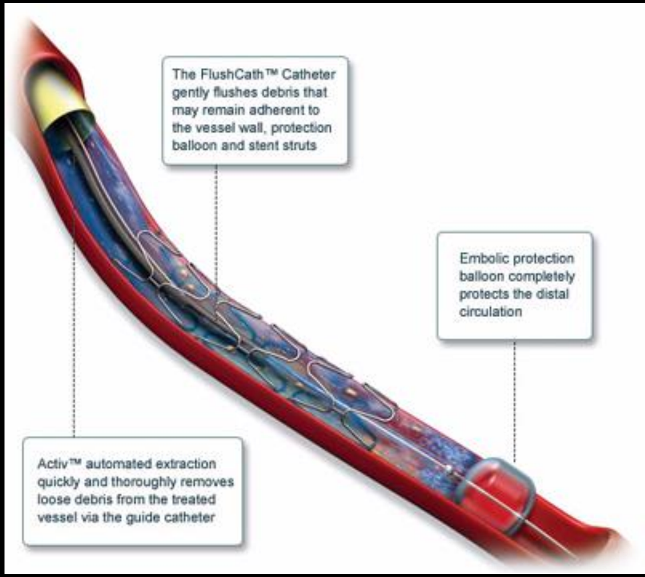
Task Force Members et al. Eur Heart J 2015;eurheartj.ehv320

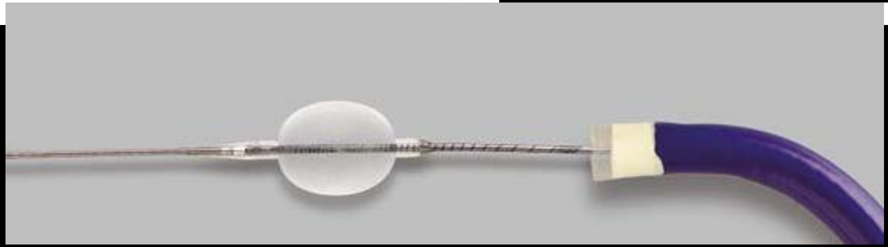


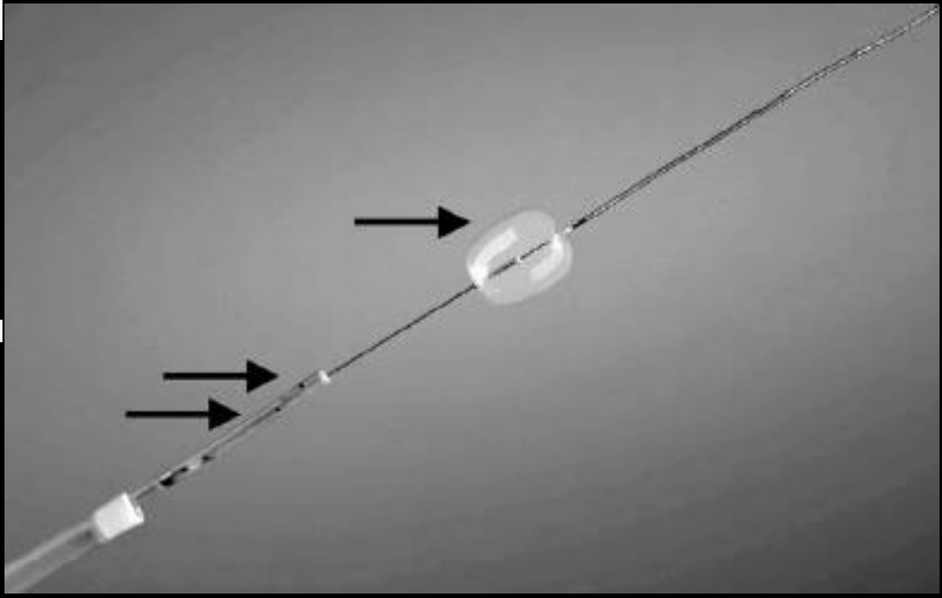
Strategy clock
0
STEMI diagnosis
ECG monitoring I B
Alert Cath Lab & by-pass ED I B
Aspirin loading1 I B
Prasugrel or Ticagrelor loading2 I A
Oxygen when Sat <90% I C
i.v. β-blocker IIa A
i.v. Opioids/ tranquilizer IIa C
Cath Lab
Radial access3 I A
Unfractionated Heparin4 I C
90 mina
Wire crossing (reperfusion)
GP IIb/IIIa bailout IIa C
New gen DES I A
Hospital Admission
Transfer back to non-PCI centre IIa C
High dose statin I A
Non-IRA PCI IIa A
Oral βblocker
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa B
ACE inhibitor
LVEF ≤40% or HF I A
LVEF >40%, no HF IIa A
24 hours
Routine echo I B
DAPT
Aspirin5 I A
Prasugrel or Ticagrelor6 I A
MRA (if LVEF ≤40% and HF) I B
Hospital discharge
Echo (LVEF) I C
6-12 weeks
1 year
b
©ESC 2017



Aspiration of debris from RCA







Aspirationsmaterial:

Vor dem Stenting

Nach Stenting

Mehrgefäss-KHK bei ACS

- Welches Gefäss?
- Welches Gefäss zuerst?
- Komplette «Revaskularisation»?

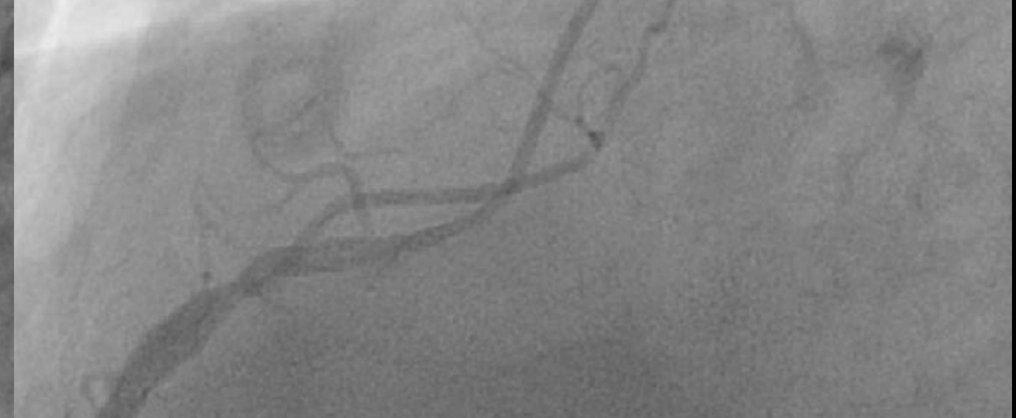
Culprit Lesion

- Two of the following morphological features of acute plaque rupture (ESC, GL 2015)
  - intraluminal filling defects consistent with thrombus (i.e.acute occlusion abruptly ending with a squared-off or convex upstream)
  - termination or an intraluminal filling defect in a patent vessel within or adjacent to a stenotic region with surrounding homogeneous contrast opacification
  - plaque ulceration (i.e. presence of contrast and hazy contour beyond the vessel lumen), plaque irregularity (i.e. irregular margins or overhanging edges)
  - Dissection or impaired flow

ACS

- 72 j Pat
- Beim Frühstück plötzlicher Schmerz
- Diskrete ST-Hebung V2-4, T-Wellen Abflachung inferior
- Trop leicht erhöht
- RF: Art. Hyp, Hyperlip, Adip, St. n. Zigarettenrauchen
- LV: apikale Hypokinesie/Frührelax






71 j Pat mit ACS und 2 G

- Was tun?
- Was zuerst?

Is Multivessel Intervention in STEMI Associated With Early Harm?

Analysis of 168,465 patients in the Nationwide Inpatient Sample (7.3% undergoing multivessel PCI) and a meta-analysis of 19 studies.

- In the Nationwide Inpatient Sample, multivessel PCI was associated with lower rate of in-hospital mortality (1.91% vs 5.32%; OR 0.28; 95% CI 0.24-0.32)
- In the meta-analysis, mortality did not differ based on completeness of revascularization (OR 0.87; 95% CI 0.65-1.17)
- Sequential analysis showed accumulated evidence did not support harm from multivessel PCI at any time point

Conclusion: After accounting for potential biases, accumulated observational evidence supports the safety—at least in the short term—of multivessel PCI in hemodynamically stable patients with STEMI.

Chatterjee S, et al. *Catheter Cardiovasc Interv.* 2016;Epub ahead of print.

Spätverlauf: atypische thorakale Schmerzen (inkl. relevante COPD)

09.06.2016

PCI beim älteren Patienten ?

z.B. > 85 j

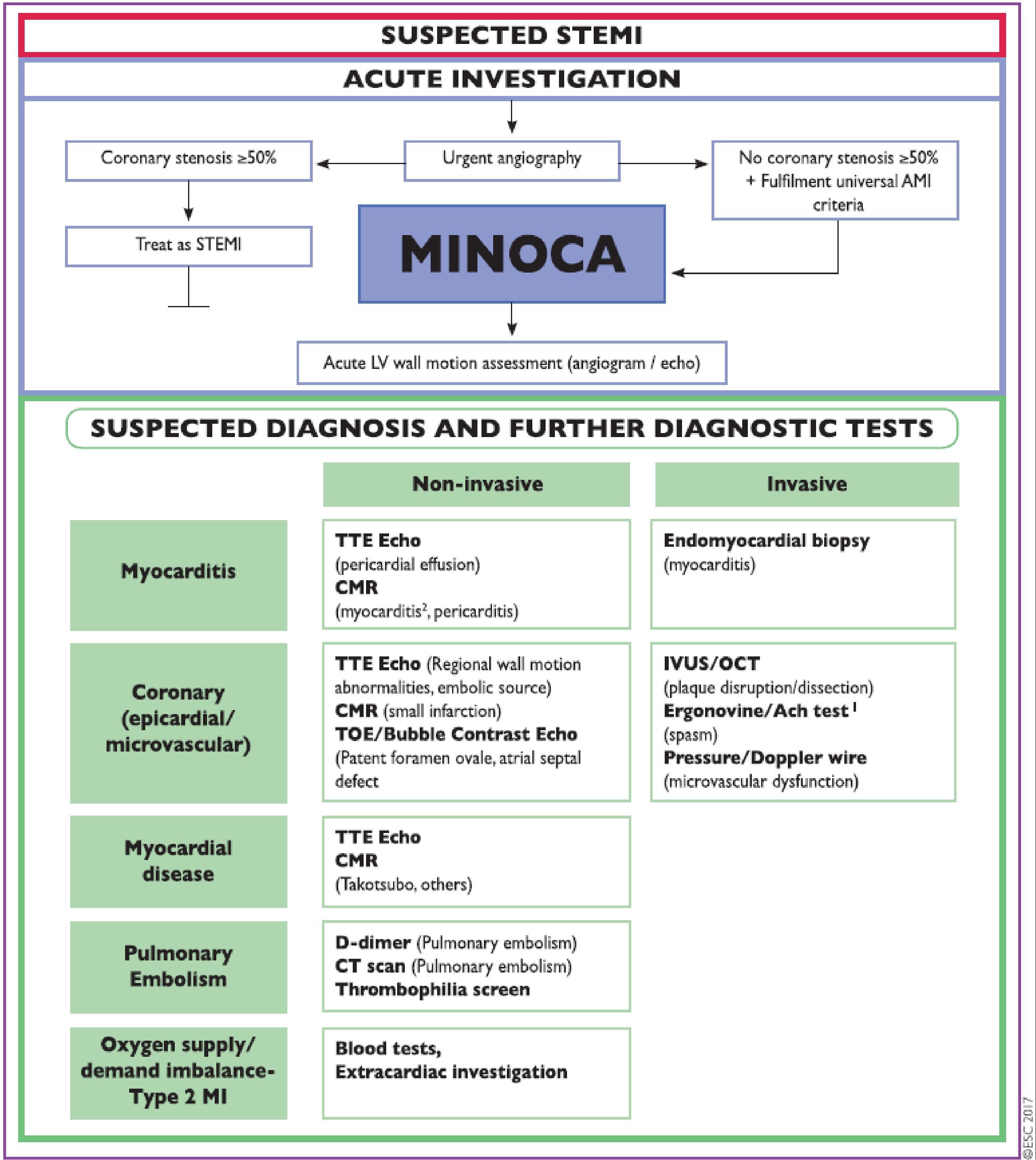
Management of Patients ≥ 85 Years of Age With ST-Elevation Myocardial Infarction

101 patients treated at a tertiary Australian hospital with conservative or invasive strategy at physician discretion, Nov 2011-July 2015.

Mortality	Conservative Strategy (n = 56)	Angiography/ PCI (n = 45)	*P* Value
In-hospital	32%	13%	0.03
30 Days	38%	13%	< 0.01
12 Months	57%	22%	< 0.01

Conclusion: Patients in their mid-to-late 80s and older are less likely to die after STEMI when treated with PCI rather than a conservative approach.

Yudi MB, et al. *Am J Cardiol.* 2016;Epub ahead of print.


SUSPECTED STEMI
ACUTE INVESTIGATION
Coronary stenosis ≥50%
Urgent angiography
No coronary stenosis ≥50% + Fulfilment universal AMI criteria
Treat as STEMI
MINOCA
Acute LV wall motion assessment (angiogram / echo)
SUSPECTED DIAGNOSIS AND FURTHER DIAGNOSTIC TESTS
Non-invasive
Invasive
Myocarditis
TTE Echo (pericardial effusion) CMR (myocarditis[2], pericarditis)
Endomyocardial biopsy (myocarditis)
Coronary (epicardial/ microvascular)
TTE Echo (Regional wall motion abnormalities, embolic source) CMR (small infarction) TOE/Bubble Contrast Echo (Patent foramen ovale, atrial septal defect
IVUS/OCT (plaque disruption/dissection) Ergonovine/Ach test[1] (spasm) Pressure/Doppler wire (microvascular dysfunction)
Myocardial disease
TTE Echo CMR (Takotsubo, others)
Pulmonary Embolism
D-dimer (Pulmonary embolism) CT scan (Pulmonary embolism) Thrombophilia screen
Oxygen supply/ demand imbalance- Type 2 MI
Blood tests, Extracardiac investigation
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Zusammenfassung

- Die Akut-PCI ist die bewährte Behandlungsmethode beim ACS

  → hohe Erfolgsrate, rasche Befreiung von Schmerzen, verbessertes Überleben

- Nahezu jeder Patient ist ein Kandidat für die Akut-PCI
- Ziel ist die rasche Intervention
  - Reduktion der Verzögerung durch Patient
  - Verkürzung der Zeit zwischen Erstkontakt Arzt-Notfall/Kardiologe
  - Kurze in-Hospital Zeit (Door-to-needle)