ACS Management in 2018: An Update

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Cardiology Days 2018

September 22nd, 2018

Cardiac Sciences Program
Programme des sciences cardiaques

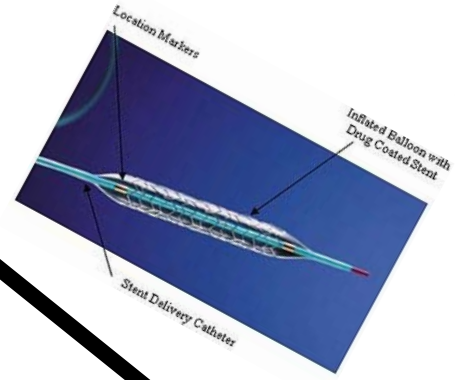
University of Manitoba
Rady Faculty of Health Sciences

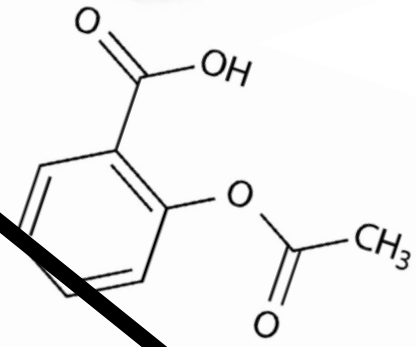
Disclosure Slide

- No relevant conflicts of interest
- No commercial relationships
- “Soapbox”

Outline and Objectives

- Define, compare and contrast “acute coronary syndrome (ACS)” and “myocardial infarction”
- Review the differential diagnosis, initial workup and management of these syndromes
- Appreciate that one of the most important facet of ACS care is recognizing what IS and what ISN’T ACS
- Review the management of ACS
- ACS Network





Acetylsalicylic acid

Acute Coronary Syndrome – Myocardial Infarction

ESC/ACCF/AHA/WHF Expert Consensus Document

Third Universal Definition of Myocardial Infarction

Kristian Thygesen, Joseph S. Alpert, Allan S. Jaffe, Maarten L. Simoons, Bernard R. Chaitman, and Harvey D. White: the Writing Group on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction

Acute Coronary Syndrome – Myocardial Infarction

Fourth universal definition of myocardial infarction (2018)

Kristian Thygesen* (Denmark), Joseph S. Alpert* (USA), Allan S. Jaffe (USA), Bernard R. Chaitman (USA), Jeroen J. Bax (The Netherlands), David A. Morrow (USA), Harvey D. White* (New Zealand): the Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/ American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction

What's new ?

- Must differentiate myocardial infarction from myocardial injury
- Highlighting peri-procedural myocardial injury after cardiac and non-cardiac procedures as discrete from myocardial infarction
- Consideration of electrical remodelling (cardiac memory) in assessing repolarization abnormalities with tachyarrhythmia, pacing, and rate-related conduction disturbance
- Use of cardiovascular magnetic resonance to define etiology of myocardial injury
- Use of CT coronary in suspected myocardial infarction

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J 2018;1–33.

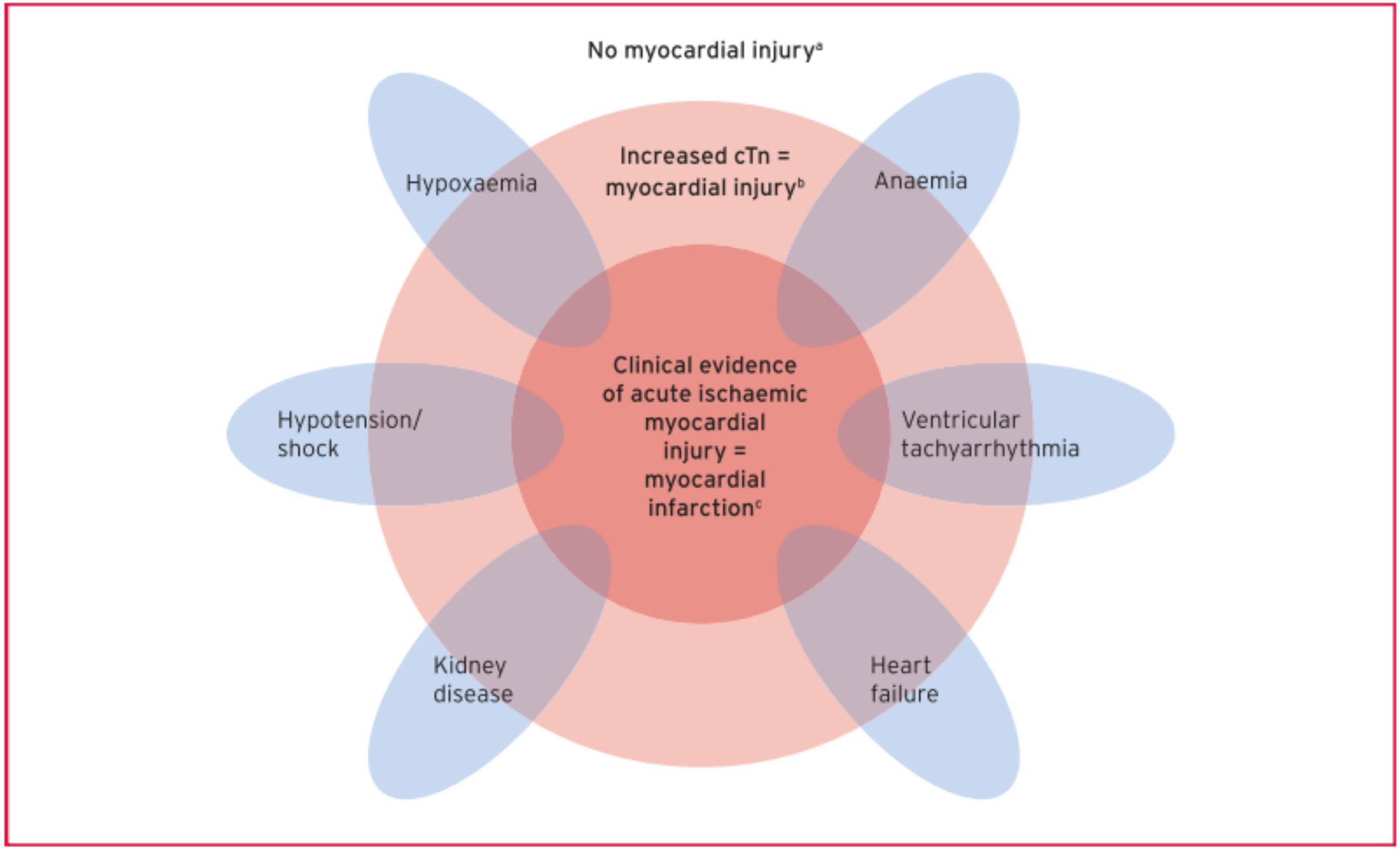
Myocardial injury

- Myocardial injury is elevated cardiac troponin values with at least one value above the 99th percentile
- Myocardial injury is considered acute if there is a rise and/or fall of TnT

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J 2018;1–33.

Acute Myocardial Infarction

- Should be considered when there is acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of TnT with at least one value above the 99th percentile AND AT LEAST one of the following:
  1. Symptoms of myocardial ischemia
  2. New ischemic ECG changes
  3. Development of pathologic Q waves
  4. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology
  5. Identification of a coronary thrombus by angiography or autopsy


No myocardial injury[a]
Increased cTn = myocardial injury[b]
Hypoxaemia
Anaemia
Clinical evidence of acute ischaemic myocardial injury = myocardial infarction[c]
Hypotension/ shock
Ventricular tachyarrhythmia
Kidney disease
Heart failure
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Who cares??

- ALL established therapies over the past 40 years focus on **myocardial infarction**
- Our entire paradigm of ACS management is mostly centred around **disruption of a coronary plaque**
- There is minimal/no established therapy at this time for myocardial injury

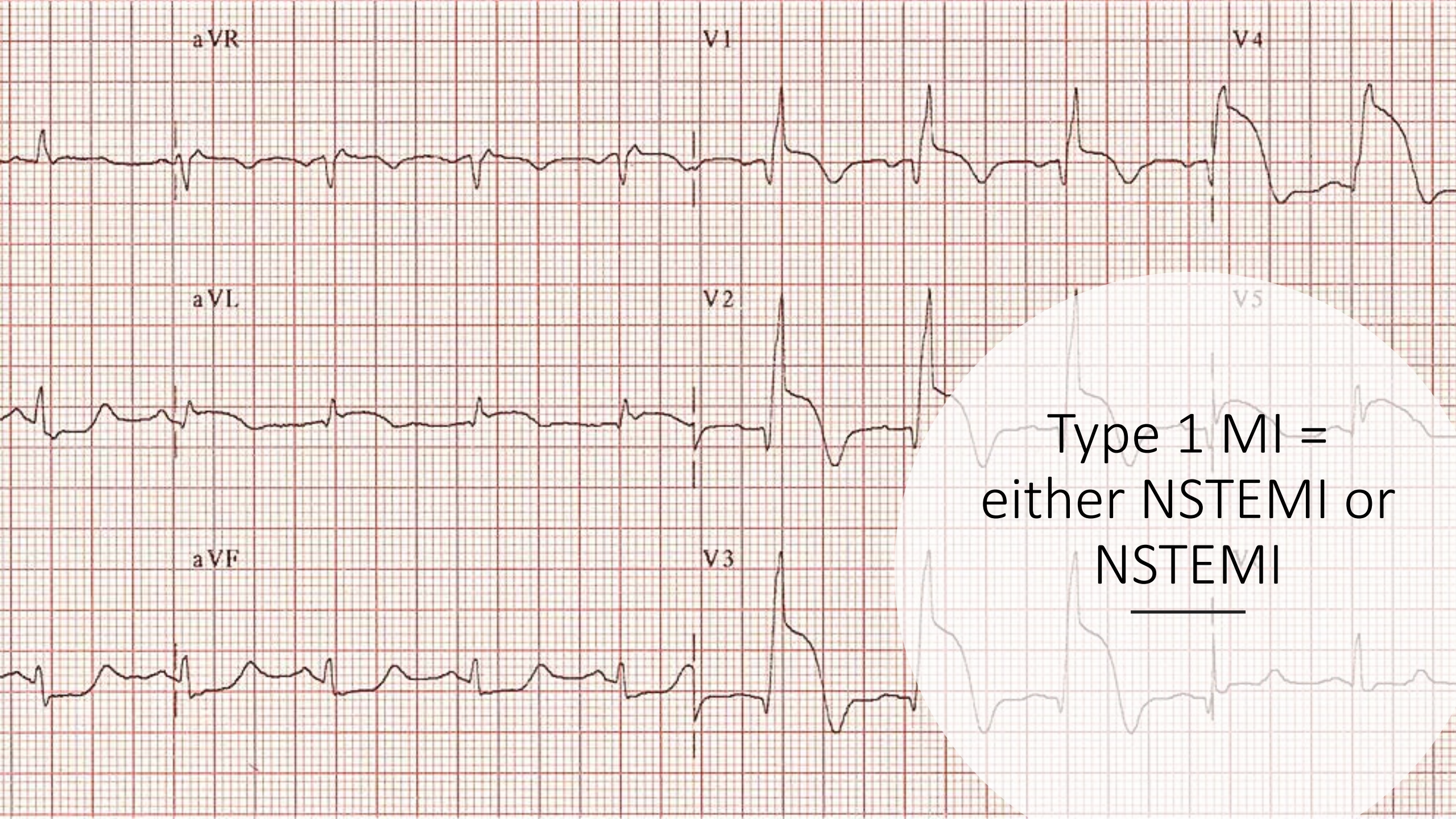
Myocardial Infarction Type 1

Clinical classification of myocardial infarction

- Type 1
  - MI caused by atherothrombotic coronary artery disease and usually precipitated by atherosclerotic plaque disruption (rupture OR erosion)

Plaque rupture/erosion with occlusive thrombus

Plaque rupture/erosion with non-occlusive thrombus



Type 1 MI = either NSTEMI or NSTEMI

Type 1 MI – STEMI vs NSTEMI (complete coronary occlusion vs incomplete)

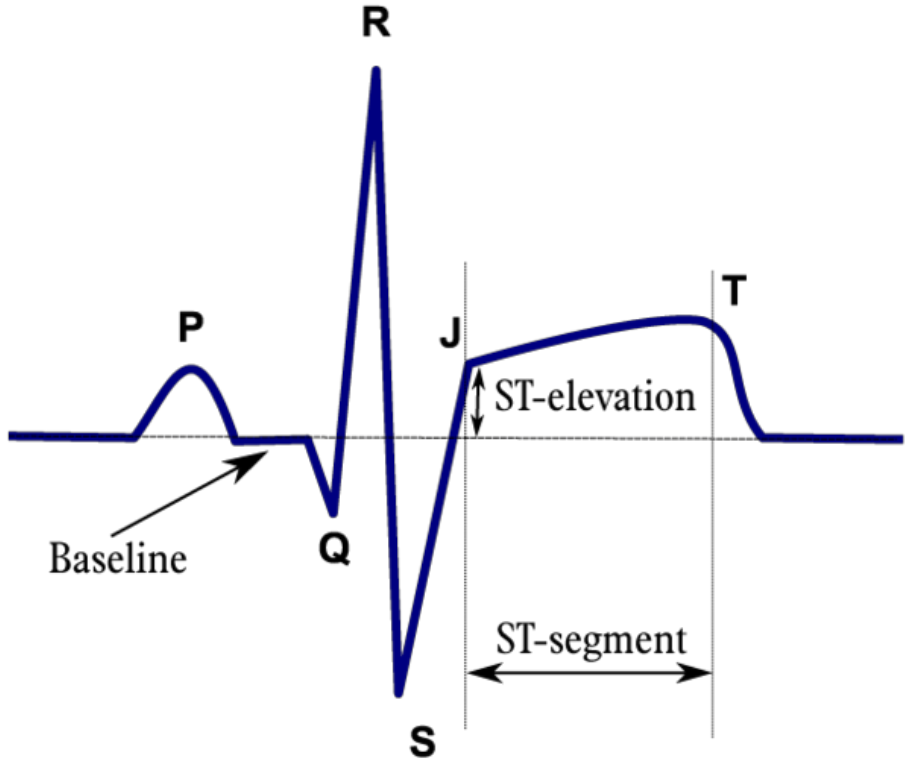
This is a crucial point: In the case of ST-elevation myocardial infarction:

Every 10-minute delay in reperfusion of STEMI is associated with 3.3 more deaths per 100 patients (high risk patients)

Scholz et al. EHJ 2018.

What is significant ST elevation?

- Measured at the J-point, compared to T-P segment
- ≥ 1 mm in contiguous leads EXCEPT V2-V3
  - ≥ 2 mm men > 40 years
  - ≥ 2.5 mm men < 40 years
  - ≥ 1.5 mm women
- **Look for reciprocal changes (PAIL)**

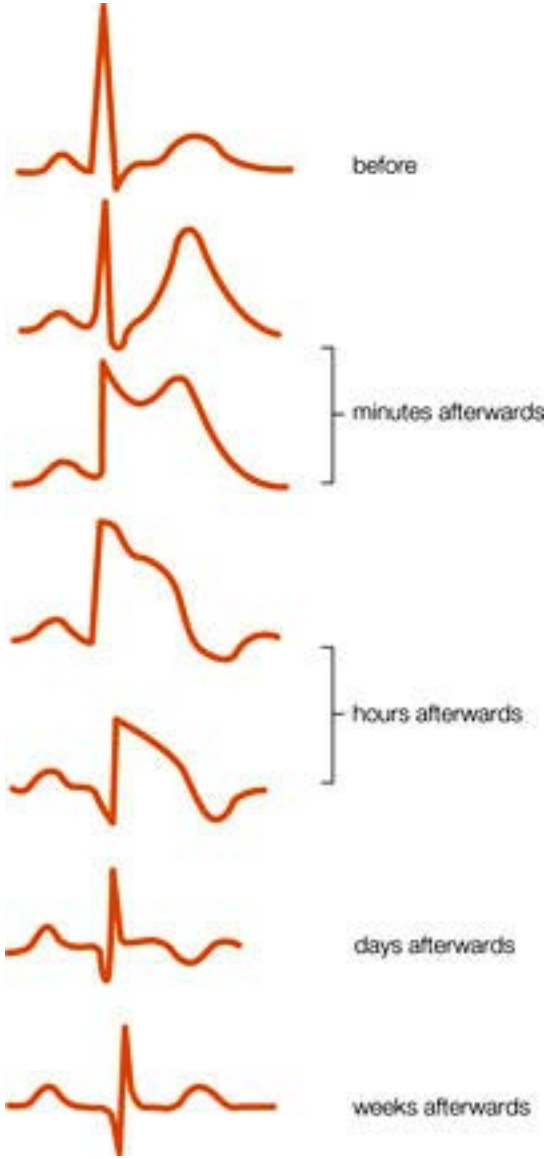


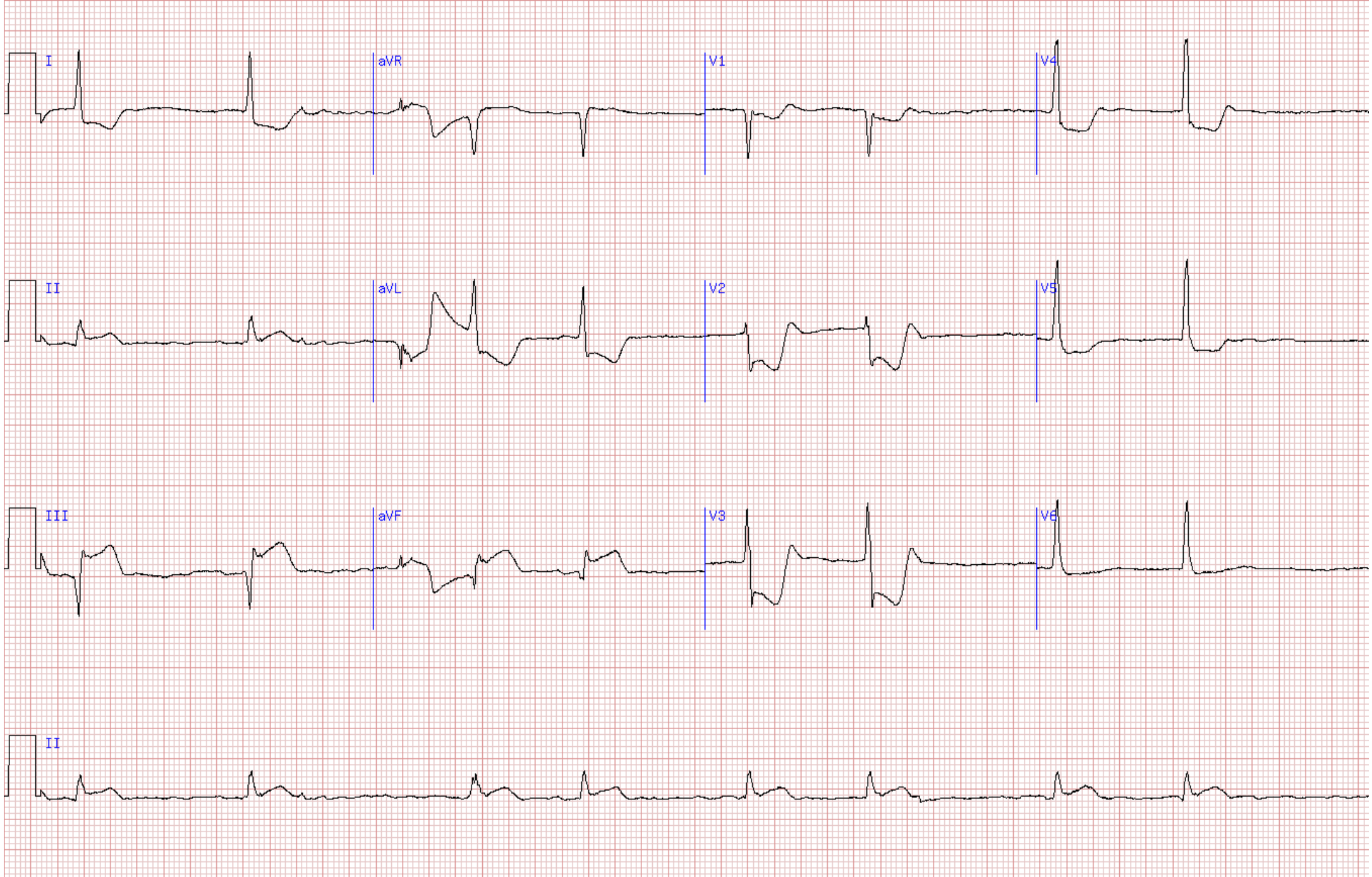
How to measure ST elevation?

1. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). Eur Heart J 2018;1–33.

ST-elevation MI - Evolution

- Hyperacute T wave – tall, peaked, symmetric
- J-point elevation, ST elevation
- Merges with T wave "tombstone"
- ST segments resolve, Q wave develops, loss of R wave amplitude
- What if persistent ST elevation?
- T wave becomes inverted

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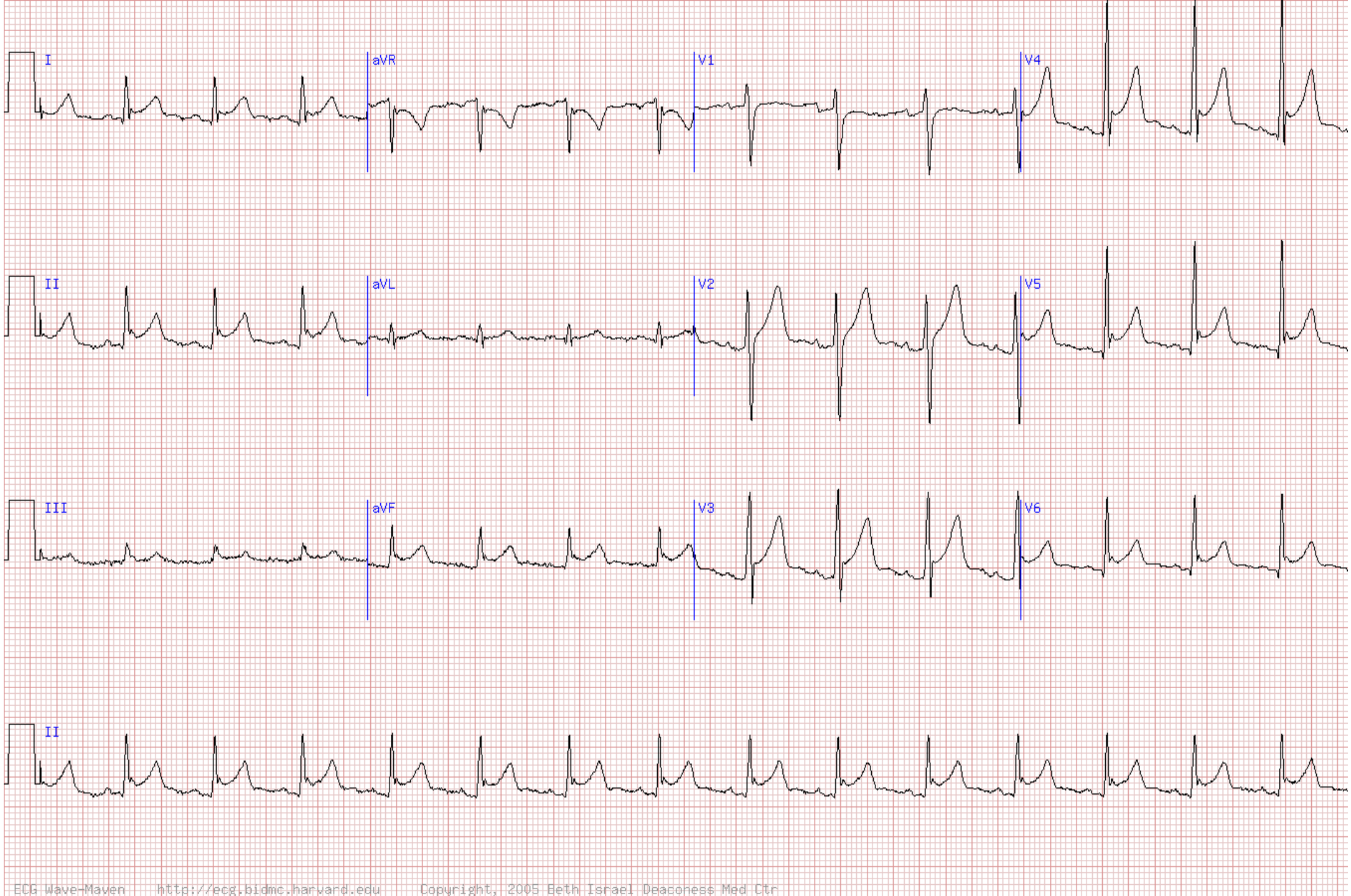
ECG Wave-Maven http://ecg.bidmc.harvard.edu Copyright, 2005 Beth Israel Deaconess Med Ctr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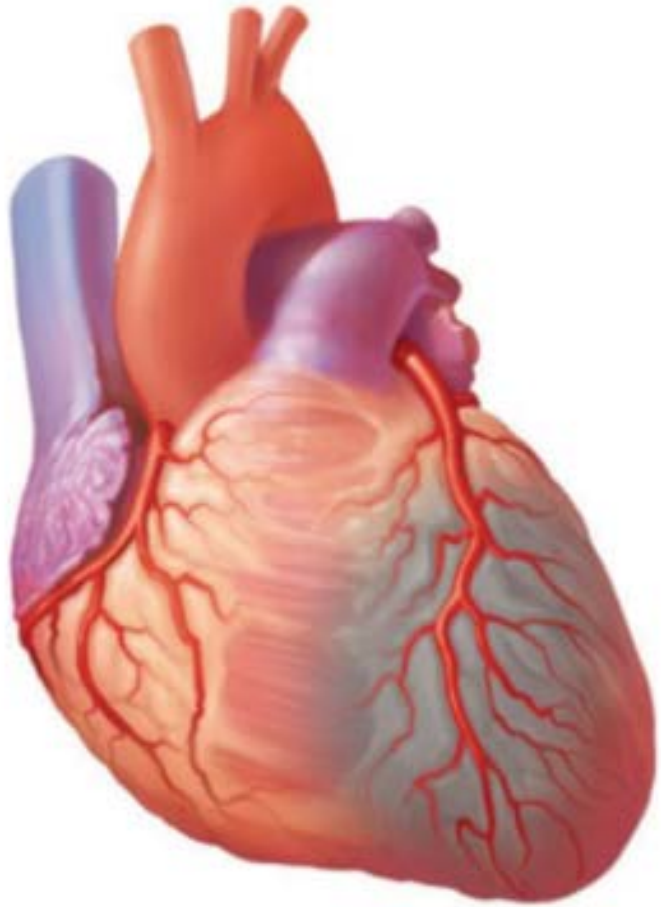
Myocardial Infarction Type 2

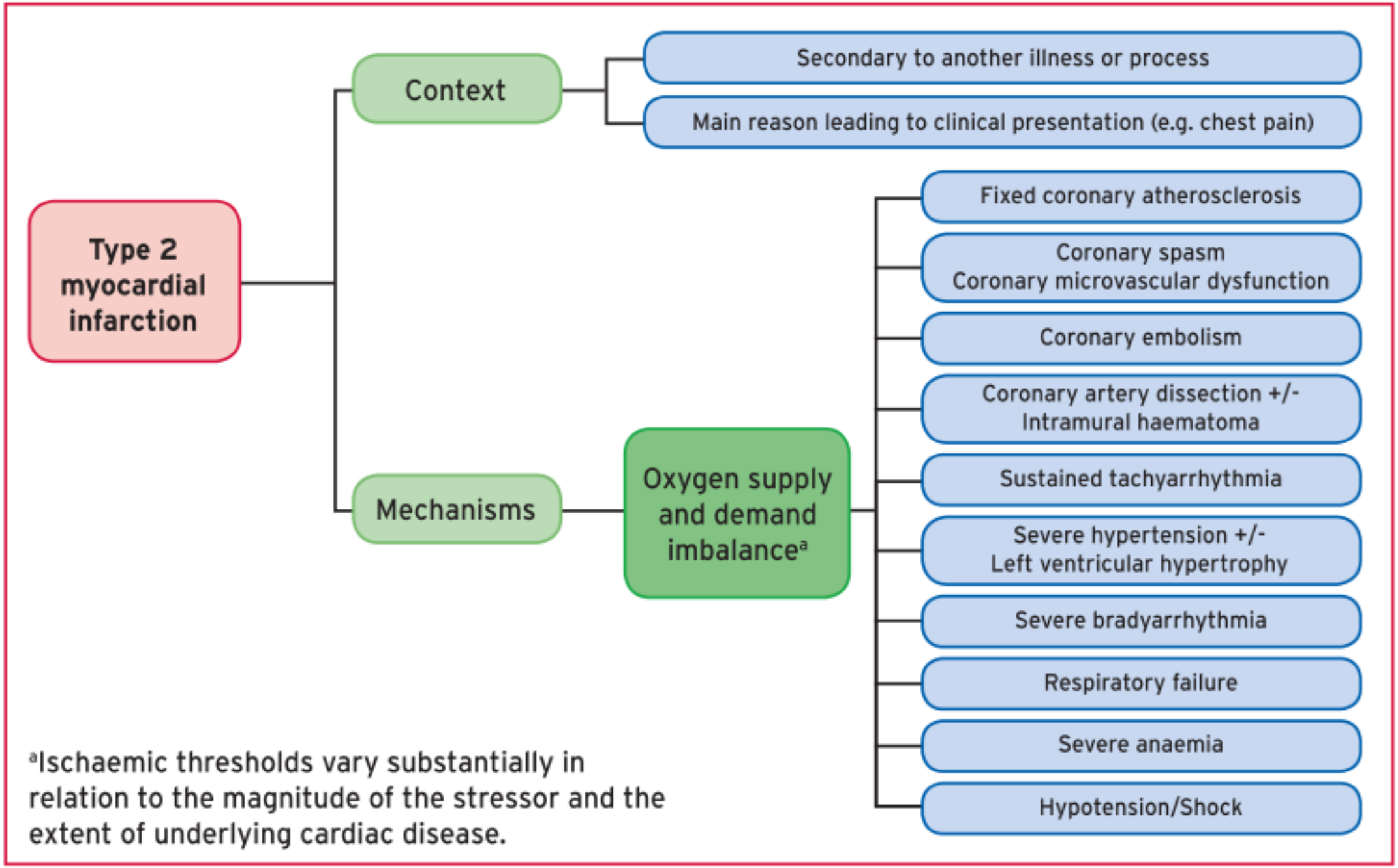


Atherosclerosis and oxygen supply/demand imbalance

Vasospasm or coronary microvascular dysfunction

Non-atherosclerotic coronary dissection

Oxygen supply/demand imbalance alone


Type 2 myocardial infarction
Context
Secondary to another illness or process
Main reason leading to clinical presentation (e.g. chest pain)
Mechanisms
Oxygen supply and demand imbalance[a]
Fixed coronary atherosclerosis
Coronary spasm
Coronary microvascular dysfunction
Coronary embolism
Coronary artery dissection +/-
Intramural haematoma
Sustained tachyarrhythmia
Severe hypertension +/-
Left ventricular hypertrophy
Severe bradyarrhythmia
Respiratory failure
Severe anaemia
Hypotension/Shock
[a]Ischaemic thresholds vary substantially in relation to the magnitude of the stressor and the extent of underlying cardiac disease.
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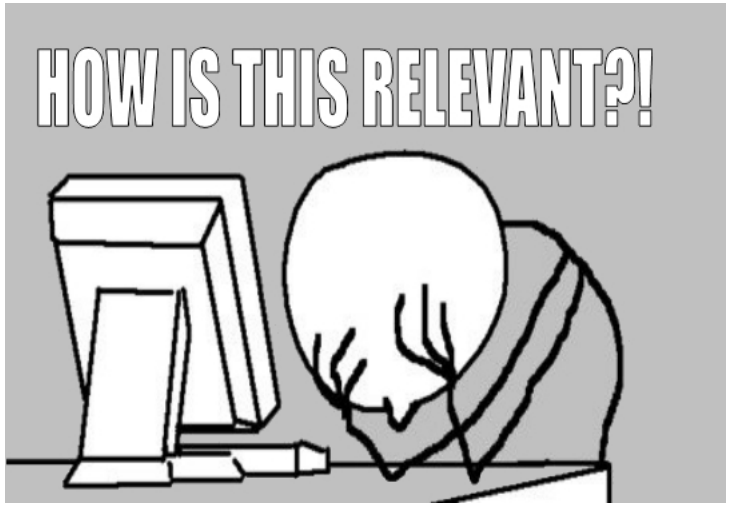
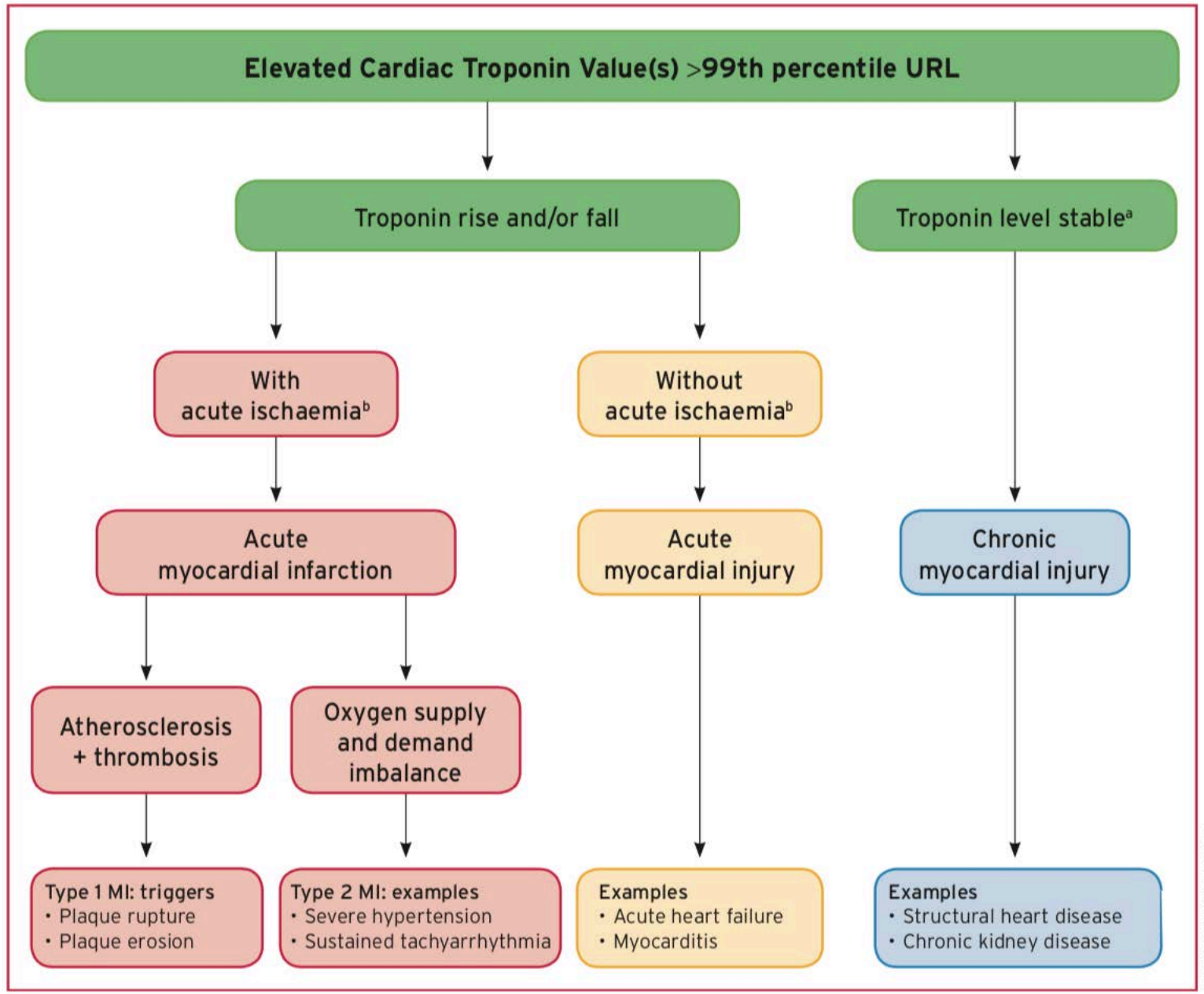
Beyond type 2 MI – Myocardial injury and troponin elevation without ischemia

- Cardiac conditions
  - Heart Failure
  - Myocarditis
  - Cardiomyopathy
  - Takotsubo
  - Coronary revascularization
  - Any cardiac procedure
  - Catheter ablation
  - Defibrillation
  - Cardiac Contusion
- Systemic conditions
  - Sepsis, severe infections
  - Chronic kidney disease
  - Stroke
  - Subarachnoid hemorrhage
  - Pulmonary embolism
  - Pulmonary hypertension
  - Infiltrative diseases, sarcoid, amyloid
  - Chemotherapy
  - Critical illness
  - Strenuous exercise

Other types of Myocardial infarction

- Type 3
  - Death presumably due to acute myocardial infarction (ECG changes, VF) without biomarkers available
- Type 4a
  - Associated with a percutaneous coronary intervention (coronary dissection etc)
- Type 4b
  - Related to stent thrombosis
- Type 4c
  - Related to stent restenosis
- Type 5
  - Related to CABG




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
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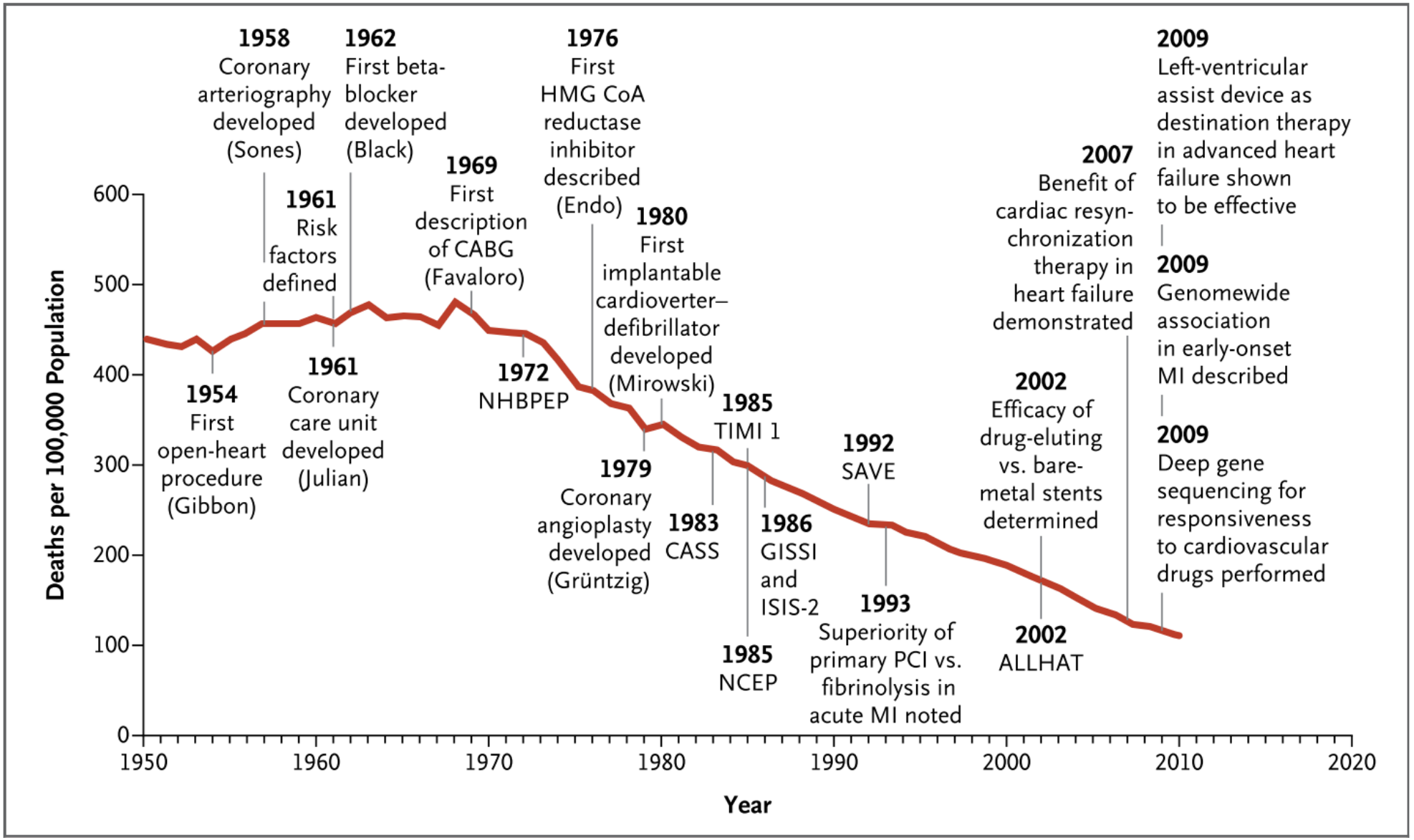
How does unstable angina (UA) fit in? (ACS = UA, NSTEMI and STEMI)

- Thought that it would become more rare with high sensitivity troponins
- Still represents ~ 10-15% of non-ST elevation ACS
- Diagnosis relies on good clinical story +/- ECG changes
  - Rapidly accelerating angina symptoms over 48 hours (crescendo angina)
  - New onset angina with minimal exertion or at rest
  - Rapid increase in frequency/severity of angina over 2-4 weeks

Take away points from this section…

- Not all troponin elevation represents an acute coronary syndrome
- Not all troponin elevation is related to the heart at all!
- Acute coronary syndrome (UA, NSTEMI, STEMI) is a *clinical* diagnosis, supported by biochemical and electrocardiographic criteria
- Once recognized, PROMPT treatment (especially for STEMI) of ACS is paramount

Treatment of ACS 2018



Nabel and Braunwald. NEJM. 2012

Reducing mortality from ACS may well have more to do with improving our systems of care than new drugs

Improving recognition of ACS, prompt triage, treatment and appropriate activation of the cardiac catheterization lab (cath lab)

About 2.5 years ago we started working on the goal to improve ACS outcome in MB by creating an ACS Network

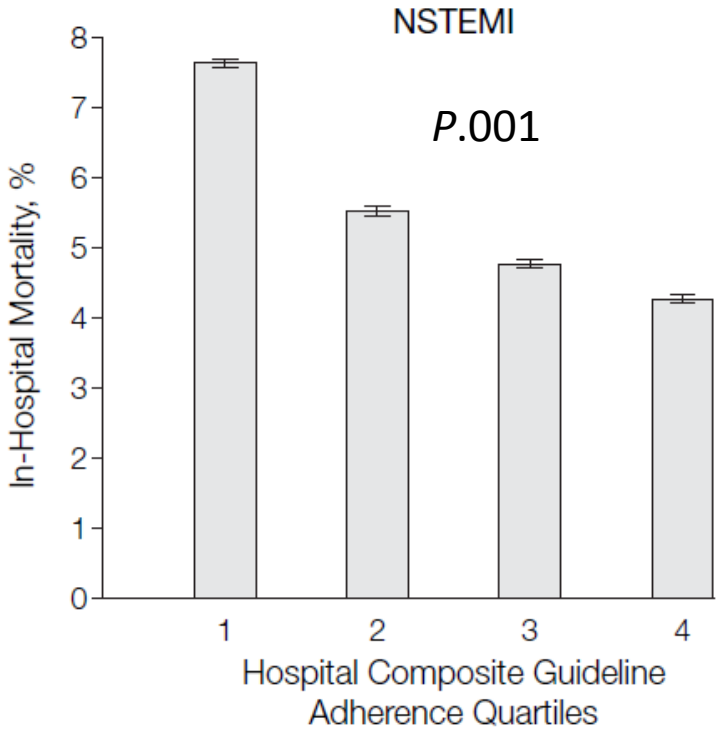
Dr. Lorraine Avery

Dr. Randy Fransoo

Dr. John Ducas

Why an ACS Network?

- ***Guideline adherence*** in ACS highly correlated with ***outcomes***



350 US centers, 64,775 patients
9 ACC/AHA class I guideline-recommended treatments

Significant association between care process and outcomes

JAMA. 2006;295:1912

Why an ACS Network?

AHA Guidelines 2013

All communities should create and maintain a **regional system of STEMI care that includes assessment and continuous quality improvement of EMS and hospital-based activities**.

ESC Guidelines 2012

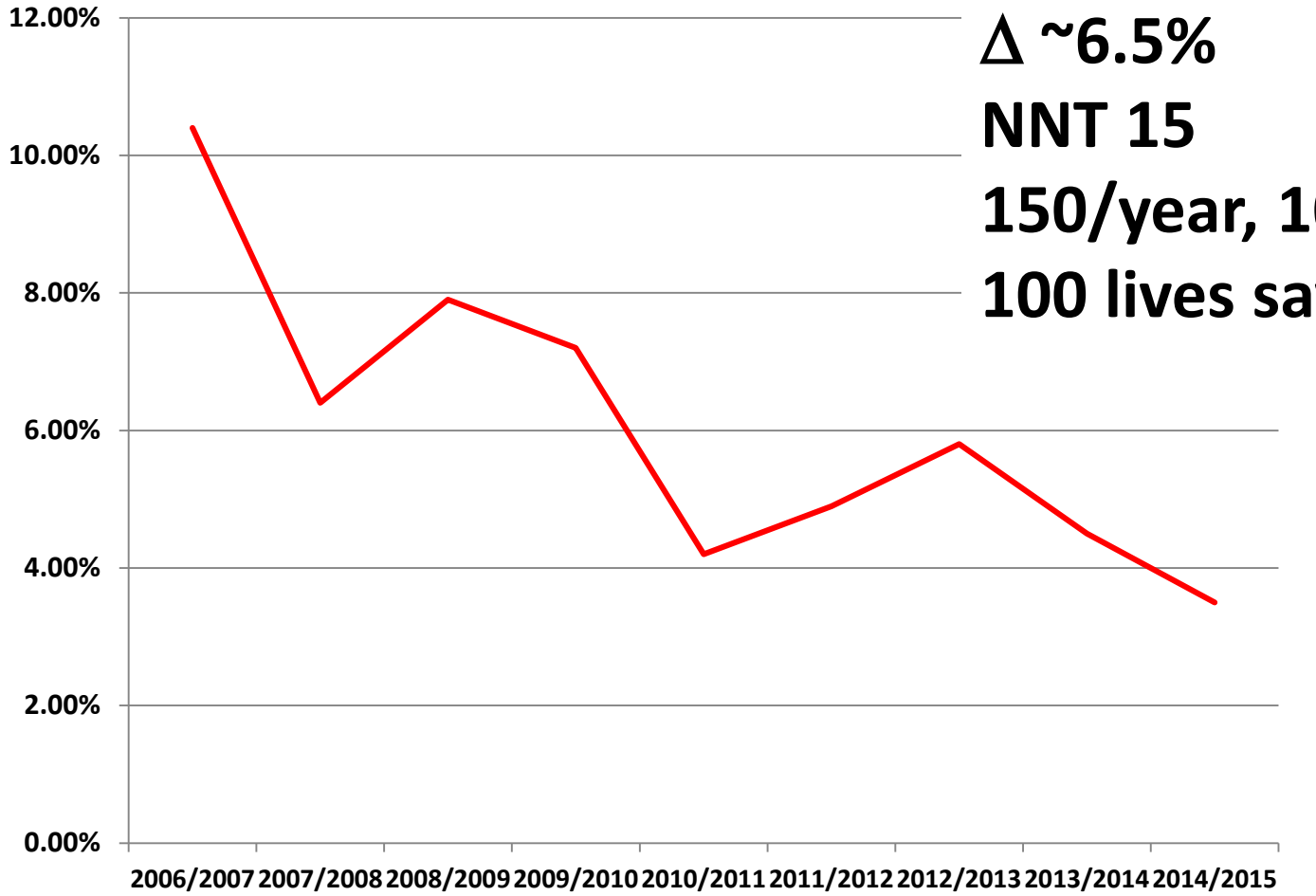
The prehospital management of STEMI patients must be based on regional networks designed to deliver reperfusion therapy expeditiously and effectively, with efforts made to make primary PCI available to as many patients as possible.	I	B
All hospitals and EMSs participating in the care of patients with STEMI must record and monitor delay times and work to achieve and maintain the following quality targets: • first medical contact to first ECG ≤10 min; • first medical contact to reperfusion therapy; • for fibrinolysis ≤30 min;	I	B

Why an ACS Network?

WRHA CODE-STEMI PROGRAM

In Hospital Mortality

150 / yr



Δ ~6.5%

NNT 15

150/year, 10/yr

100 lives saved!!!

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STEMI Diagnosis*

If diagnosis UNCERTAIN, call Outside Call Cardiologist (204-237-2053) or local specialist to discuss.
If diagnosis CERTAIN, DO NOT delay treatment by calling! Follow as below.

Can this patient arrive at SBGH in < 100 min FROM FIRST MEDICAL CONTACT?

YES, MD to:

1. ***First***, arrange Immediate Appropriate Transfer for Primary PCI*
2. ***Then***, page the Interventional Cardiologist on Call (204-237-2053)

Drugs prior to transport: *(if no contraindication)*

1. ASA 160 mg
2. Clopidogrel 600 mg ***OR*** ticagrelor 180 mg
3. **Bolus** IV Heparin 70 u/kg ***OR*** IV enoxaparin 0.5 mg/kg

NO, MD to:

First, administer immediate Fibrinolysis *(if no contraindication)*

< 75 years

1. TNK 0.5 mg/kg *max 50 mg*
2. Enoxaparin 30 mg IV & 1 mg/kg subcut *max 100 mg*
3. Clopidogrel 300 mg PO

> 75 years

1. TNK 0.5 mg/kg *max 50 mg*
2. Enoxaparin 0.75 mg/kg subcut *max 80 mg*
3. Clopidogrel 75 mg PO

Then, MD to follow steps below:

1. Arrange Immediate transfer for coronary angiography
2. Call Outside Call Cardiologist (204-237-2053) to discuss patient destination
3. Complete Cath Lab Referral form fax (204-258-1089) or & send with patient
4. Instruct transport staff to page Interventional Cardiologist on Call (204-237-2053) if **ongoing pain 30 minutes from SBGH**

Outside Call Cardiologist:

1. Direct patient destination as below
2. Inform Interventional Cardiologist on call

Expected Arrival Work Day 06:30 - 18:00

Patient to Y2 Pre & Post

Expected Arrival Off Hours

Patient to SBH ER & inform ER (call 204-237-2260)

***WRHA STEMI**

- Call 204-986-8410
- State there is a 'STEMI RED' patient requiring transport
- Provide the IFTC with the following information:
  - Ward/Room and transporting facility name
  - Patient's name
  - Does the patient require transvenous pacing, inotropes or vasopressors or is this patient intubated?
- The above information determines the appropriate level of transport staff
- If patient requires transvenous pacing, inotropes or vasopressors then an Advanced Care Level Respiratory Therapist will be dispatched

***Non WRHA**

- As per local/regional guidelines, consider STARS, Lifeflight as appropriate

FMC: FMC is the time of triage at the hospital or arrival of a paramedic at the side of the patient for emergency medical services (EMS) users)

Absolute Contraindications

As determined by asking the patient the following series of questions:

- Have you ever had a bleed into your brain?
- Have you ever had a brain aneurysm, a brain tumor, or recent brain or spine surgery (within the past two months)?
- Have you had any significant head or facial trauma within the past three months?
- Have you had a stroke within the past three months?
- Have you had recent major bleeding, or major surgery or a biopsy
- Are you currently pregnant or within one week post-delivery?

As determined when there is a high index of suspicion by the clinician

- Physician suspects acute aortic dissection
- Physician suspects acute pericarditis

Relative Contraindications

As determined by the clinician

- Any measurement of a blood pressure on this encounter: Systolic BP > 180 mmHg and/or diastolic BP > 110 mmHg
- Traumatic or prolonged CPR

Enoxaparin Contraindications

- Refer to contraindications for fibrinolytics (as above)
- Allergy or hypersensitivity to heparin, pork products or to enoxaparin

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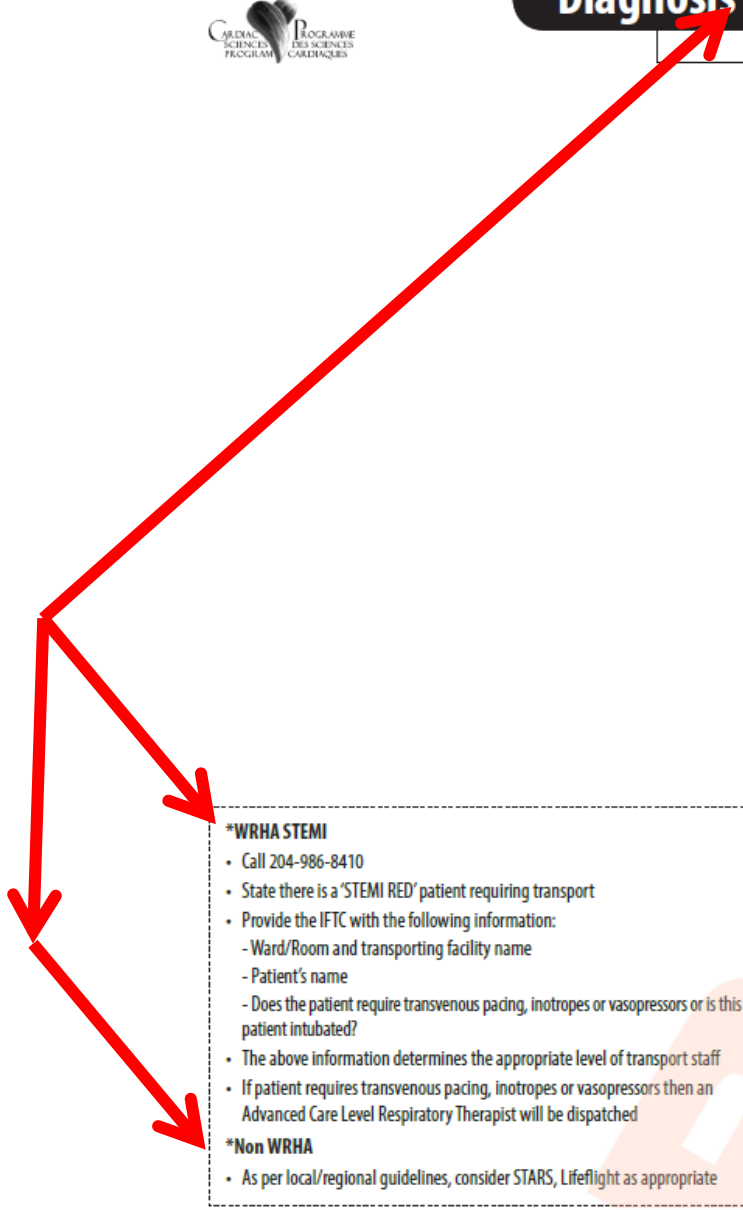

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Can this patient arrive at SRGH in < 100 min FROM FIRST MEDICAL CONTACT?

NO, MD to:

First, administer immediate Fibrinolysis *(if no contraindication)*

< 75 years	**> 75 years**
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FMC: FMC is the time of triage at the hospital or arrival of a paramedic at the side of the patient for emergency medical services (EMS) users)

RECOMMENDATIONS FOR REVASCULARIZATION IN ACUTE CORONARY SYNDROMES

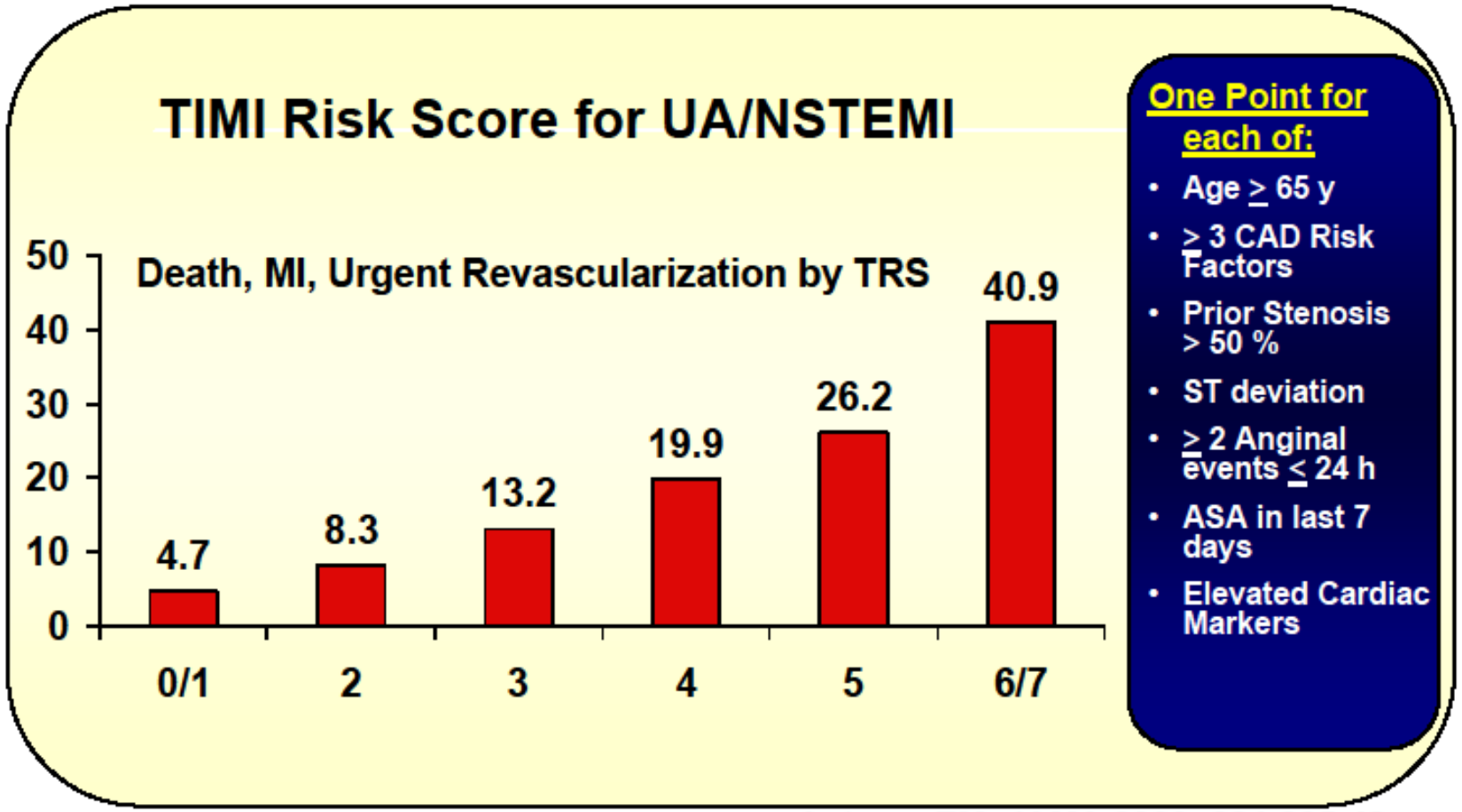
- We recommend early culprit-lesion revascularization with PCI, with minimal delay, in patients with STEMI. (Strong recommendation, high-quality evidence.)
- We recommend early culprit-lesion revascularization with PCI or early complete revascularization with CABG in most patients with acute coronary syndromes other than STEMI depending on relative stability and anatomy. (Strong recommendation, moderate-quality evidence.)

Values and Preferences: When a culprit lesion has been treated by PCI on a background of multivessel CAD, and there is uncertainty about the residual multivessel disease, eg, residual ischemia and anatomic complexity, it is reasonable to treat the culprit lesion and delay decisions on nonculprit lesions in a staged PCI procedure until the acute threat has resolved.

CJC 30 (2014) 1482e1491

Recommended Standards for the Evaluation/Treatment of Suspected Cardiac Chest Pain

TIMI 11B



Antman EM, JAMA 2000; 284:835-42

Recommended Standards for the Evaluation/Treatment of Suspected Cardiac Chest Pain

Focus on…. *NSTEMI Triage for timely cath*

	Standard	Data Elements
11	ACS (NSTE-ACS) patients (TIMI Risk Score 3 or higher) without contraindication, should receive ASA, a P2Y12 Inhibitor (ticagrelor preferred), an anticoagulant (heparin, enoxaparin or fondaparinux), a statin and a beta blocker with appropriate loading doses within ***target of*** 90 minutes of first medical contact.	• Time of first medical contact • TIMI Score • Time of administration of each medication
12	ACS (NSTE-ACS) patients (TIMI Risk Score 3 or higher) excluding unstable (#7 above) or very high risk (#8 above), without contraindications, should receive a coronary angiography within ***target of*** 72 hours of first medical contact.	• Time of first medical contact. • TIMI Score • Time to device/left coronary visualization

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Chest Pain with Cardiac Features

- First Medical Contact (FMC) Time documented (FMC is the time of triage at the hospital or arrival of a paramedic at the side of the patient for emergency medical services (EMS) users)
- Vital Signs recorded
- 12 lead ECG (target: performed within 10 minutes and interpreted within 5 minutes)

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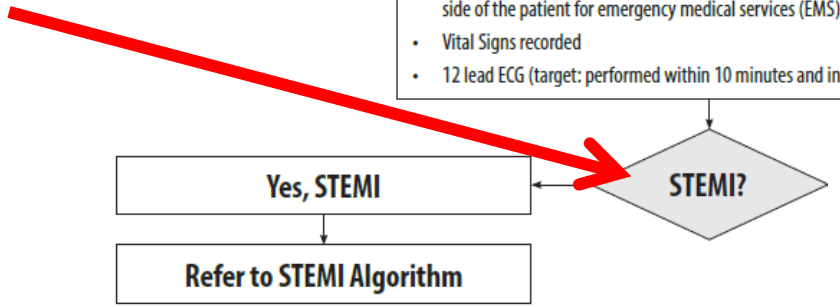

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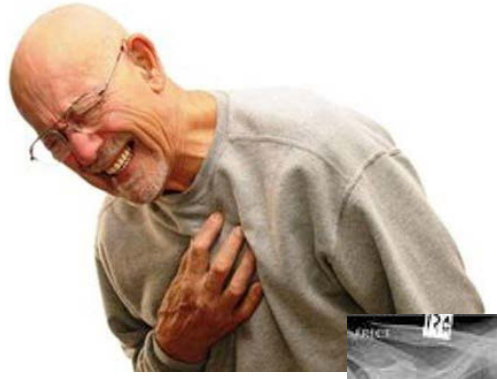
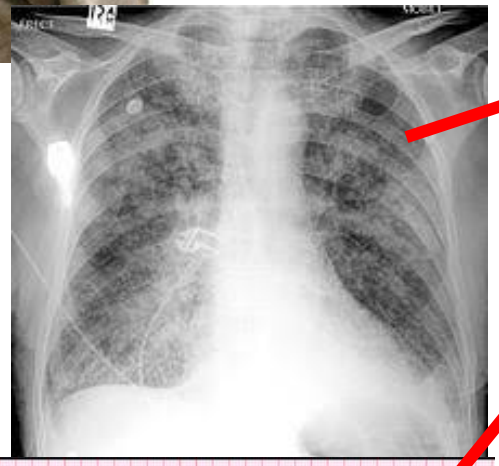
STEMI?

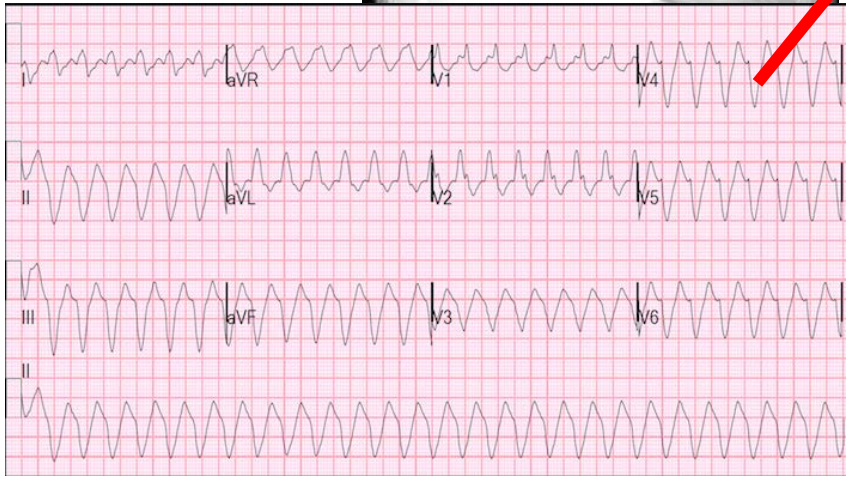
No, NSTEMI/Unstable angina

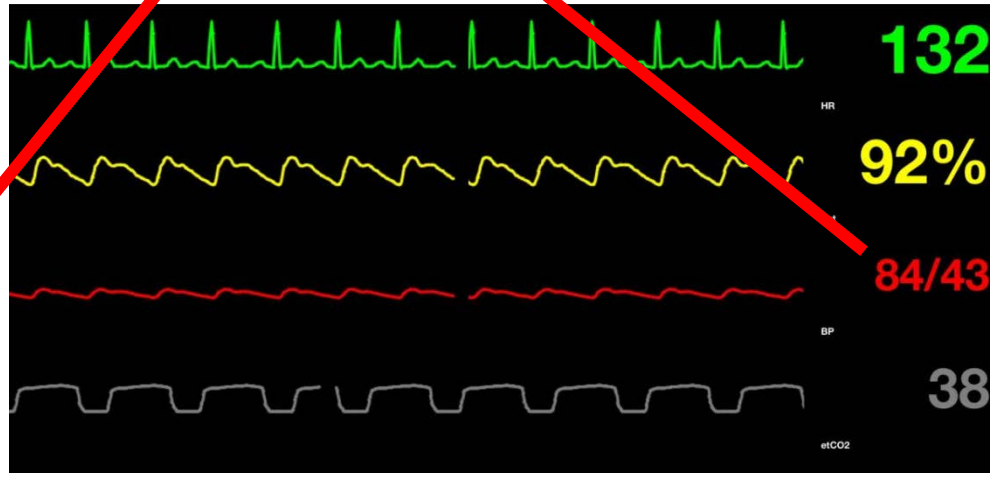
Is patient unstable? Presence of:

- Refractory Angina
- Heart Failure
- Life Threatening Arrhythmia
- Hemodynamic Instability









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Chest Pain with Cardiac Features

- First Medical Contact (FMC) Time documented (FMC is the time of triage at the hospital or arrival of a paramedic at the side of the patient for emergency medical services (EMS) users)
- Vital Signs recorded
- 12 lead ECG (target: performed within 10 minutes and interpreted within 5 minutes)

STEMI?

No, NSTEMI/Unstable angina

Is patient unstable? Presence of:
- Refractory Angina
- Heart Failure
- Life Threatening Arrhythmia
- Hemodynamic Instability

Unstable

Yes

1. Call Outside Call Cardiologist (204-237-2053) or local specialist to discuss patient
2. Complete Cath Lab Referral Form send with patient or Fax (204-258-1089)
3. *Administer treatment for likely ACS
4. *Arrange appropriate transport*

Target transfer to cath lab less than 120 minutes from FMC

***Treatment for Likely ACS (unstable angina or NSTEMI):**
if no contraindications
- ASA
- Ticagrelor or Clopidogrel
- High Dose Statin
- Beta-blocker
- LMW Heparin or Unfractionated Heparin
- Telemetry/patient monitoring

Doses as per local protocol

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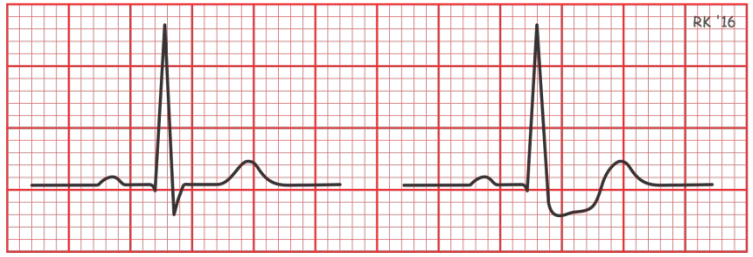
Unstable

No

Is patient High Risk? Presence of:
- Recurring Chest Pain
- Dynamic ST-T changes



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TIMI SCORE CALCULATIONS	
TIMI RISK SCORE FOR UA & NSTEMI	
CRITERIA	POINTS
HISTORICAL	
☐ Age 65 years or more	1
☐ 3 or more Risk Factors for CAD	1
☐ Known CAD (stenosis 50% or more)	1
☐ Aspirin use in past 7 days	1
PRESENTATION	
☐ Recent (24 hours or less) severe angina	1
☐ ST segment deviation 0.5 mm or more	1
☐ Elevated Cardiac Markers	1
RISK SCORE = TOTAL	0 - 7

Chest Pain with Cardiac Features

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No

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- Dynamic ST-T changes

No

TIMI Score?

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Chest Pain with Cardiac Features

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STEMI?

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Unstable

No

Is patient High Risk? Presence of:
- Recurring Chest Pain
- Dynamic ST-T changes

No

TIMI Score?

TIMI 3 or greater

1. Complete Cath Lab Referral Form & fax
2. *Administer treatment for likely ACS
3. If unsure call Outside Call Cardiologist (204-237-2053) to discuss patient

Target transfer to cath lab less than 72 hours from FMC

***Treatment for Likely ACS (unstable angina or NSTEMI):**
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- Beta-blocker
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Unstable

No

Is patient High Risk? Presence of:
- Recurring Chest Pain
- Dynamic ST-T changes

No

TIMI Score?

TIMI 2 or less

1. Repeat troponin 1-3 hour from baseline
2. Recalculate TIMI Risk Score

TIMI Score?

TIMI 2 or less

***Treatment for Likely ACS (unstable angina or NSTEMI):**
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- Beta-blocker
- LMW Heparin or Unfractionated Heparin
- Telemetry/patient monitoring

Doses as per local protocol

TIMI 2 or less

Consider:

- Pre discharge GXT if available
- Discharge home with follow-up Cardiology/Internal Medicine
- If unsure Call Outside Call Cardiologist (204-237-2053) or local specialist to discuss patient
- If smoker, consider NRT and referral to Smoker Helpline (www.smokerhelpline.ca)

CARDIAC SCIENCES PROGRAM

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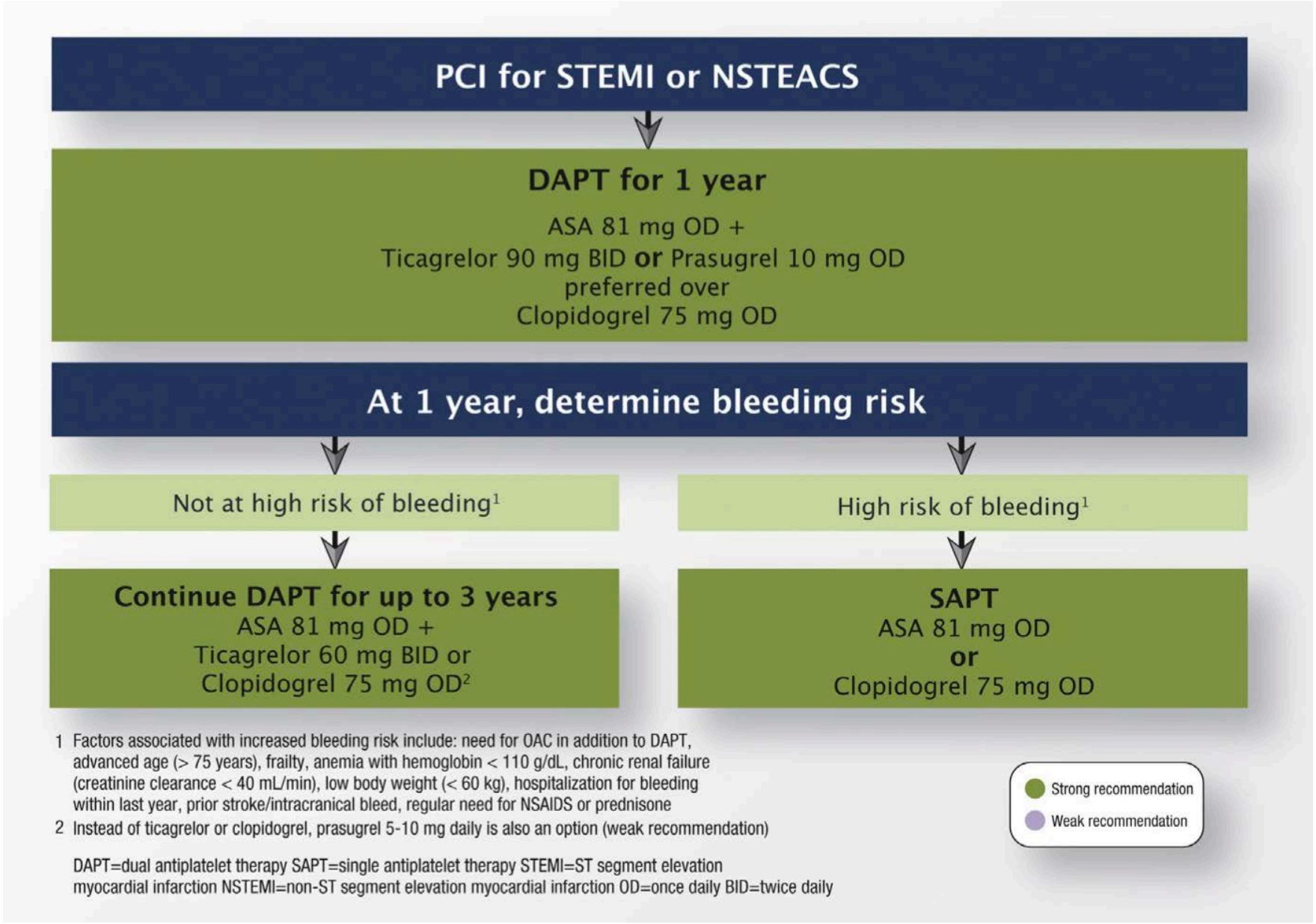
Take home points

- Not all troponin elevation represents an acute coronary syndrome
- Not all troponin elevation is related to the heart at all!
- Acute coronary syndrome (UA, NSTEMI, STEMI) is a *clinical* diagnosis, supported by biochemical and electrocardiographic criteria
- Once recognized, PROMPT treatment (especially for STEMI) of ACS is paramount
- Manitoba ACS Network Standards and care protocols are an excellent resource
- Never hurts to ask for advice if unsure

Thank you! Questions?

Acknowledgements:
Dr. Michael Love
Dr. John Ducas
ACS Network



CJC (2018) Antiplatelet Guidelines