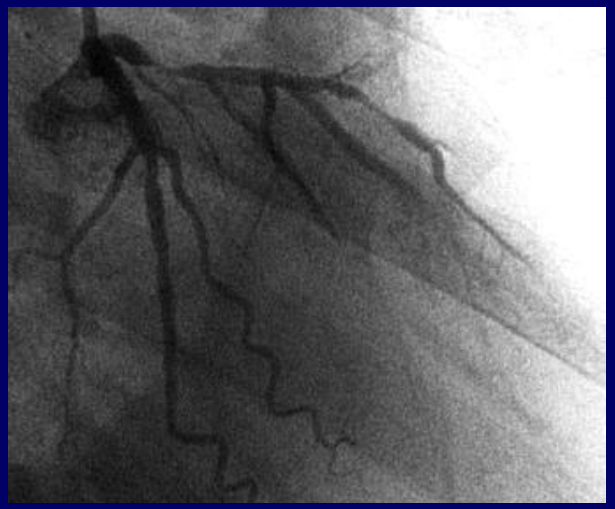
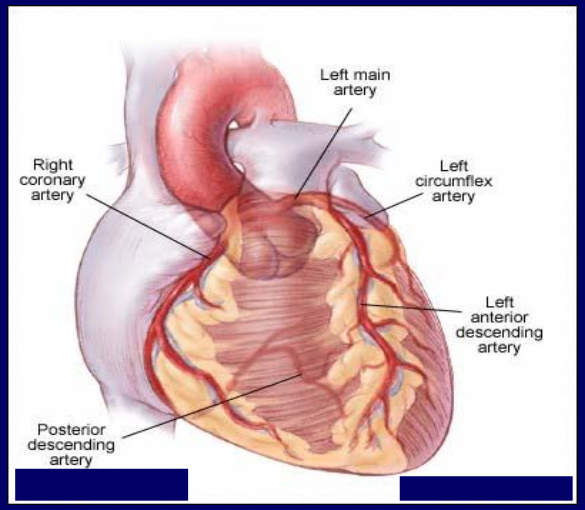
Life after a heart attack



John Elliott



Associate Professor of Medicine
Interventional Cardiologist
Christchurch

UNIVERSITY of OTAGO
Te Whare Wānanga o Otāgo

Topics to discuss today

- Life before a heart attack -screening
- Delays to presentation
- Diagnosing a heart attack
- Trends in
  - in-hospital treatments,
  - investigations and
  - prognosis
- Ongoing care after discharge

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- Delays to presentation
- Diagnosing a heart attack
- Trends in
  - in-hospital treatments,
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  - prognosis
- Ongoing care after discharge
- Rupert

Life before a heart attack

- Rupert is a 46 year old builder
- Smokes 25/day since he played as lock for Otago
- Enjoys 3 jugs a night
- BMI 32
- He thinks he is healthy because he can still do what he did 10 years ago
- Father died suddenly aged 47. Mother well, Older brother has diabetes.

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- Comments?

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- BMI 32
- He thinks he is healthy because he can still do what he did 10 years ago
- Father died suddenly aged 47. Mother well, Older brother has diabetes
- Comments?
- He probably has the arteries of a 65 year old

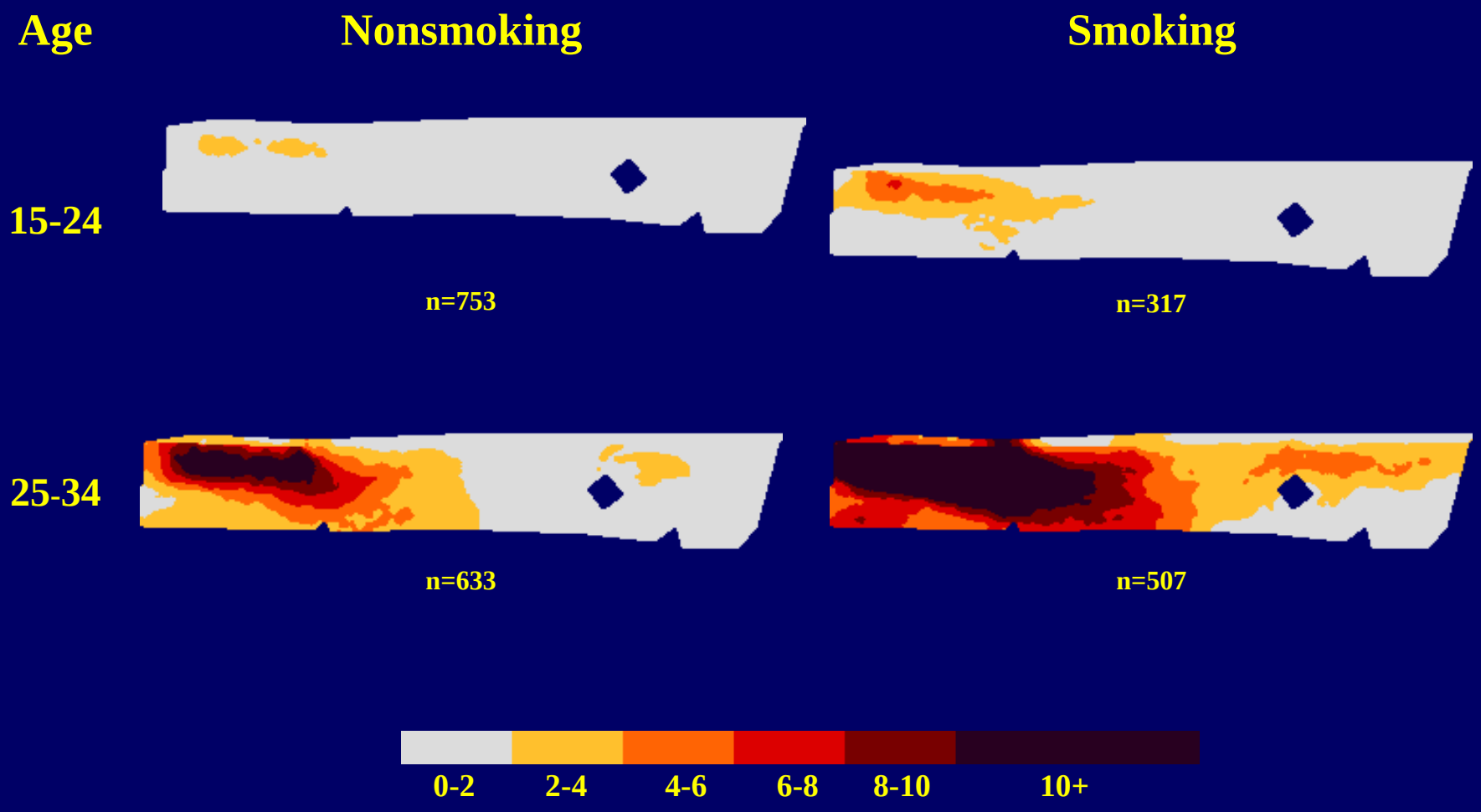
Life before a heart attack

- We all have atherosclerosis
- It begins at school
- It progresses more rapidly if risk factors present as a teenager or young adult

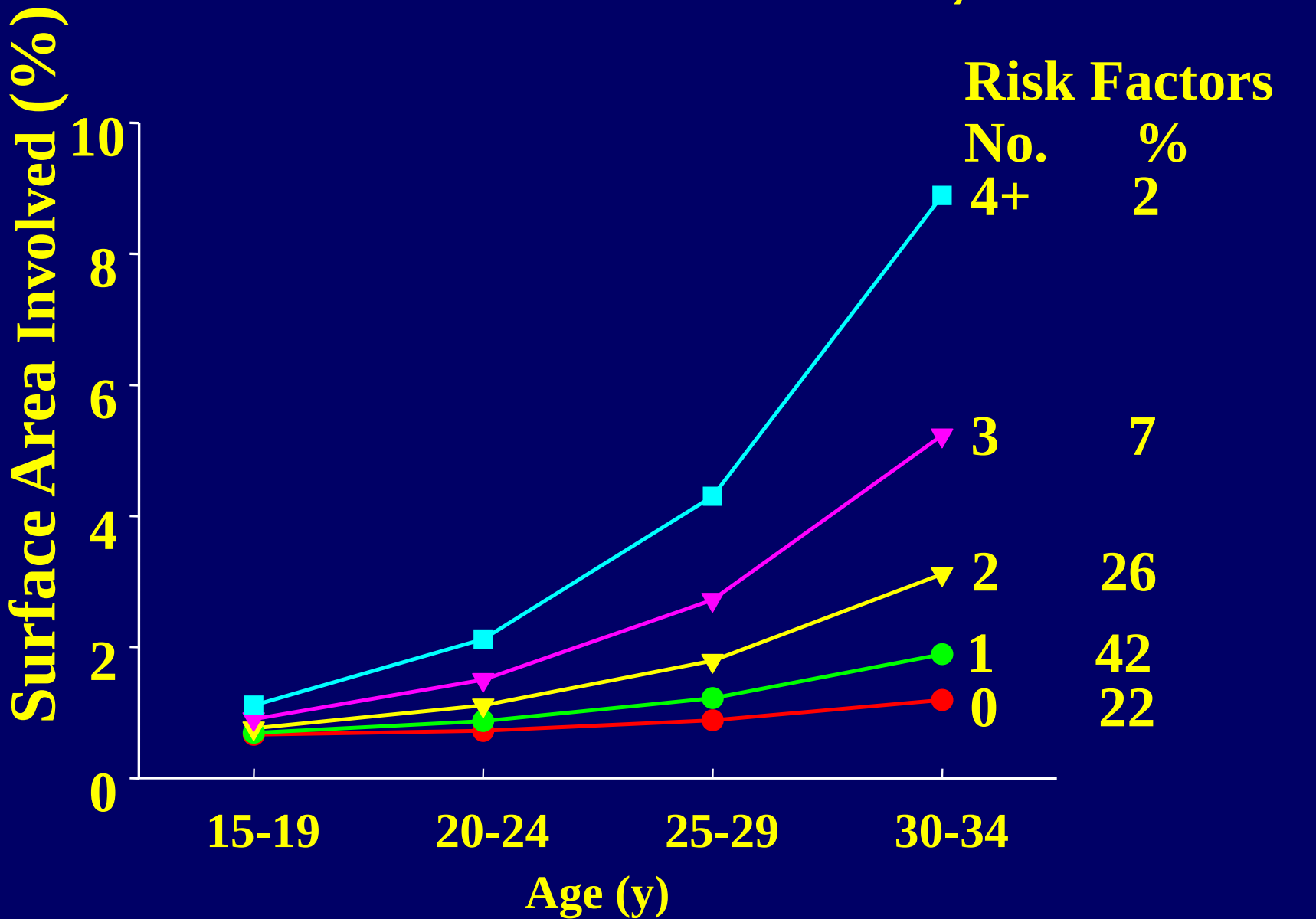
PDAY Study: Pathobiological Determinants of Atherosclerosis in Youth

- Study of atherosclerosis in 15-34 year old persons autopsied in forensic laboratories in USA
- Organized in 1985 by 14 centers
- Collected 3,000 cases (arteries and risk factor data), 1987-1994
- Grading and analyses in central labs

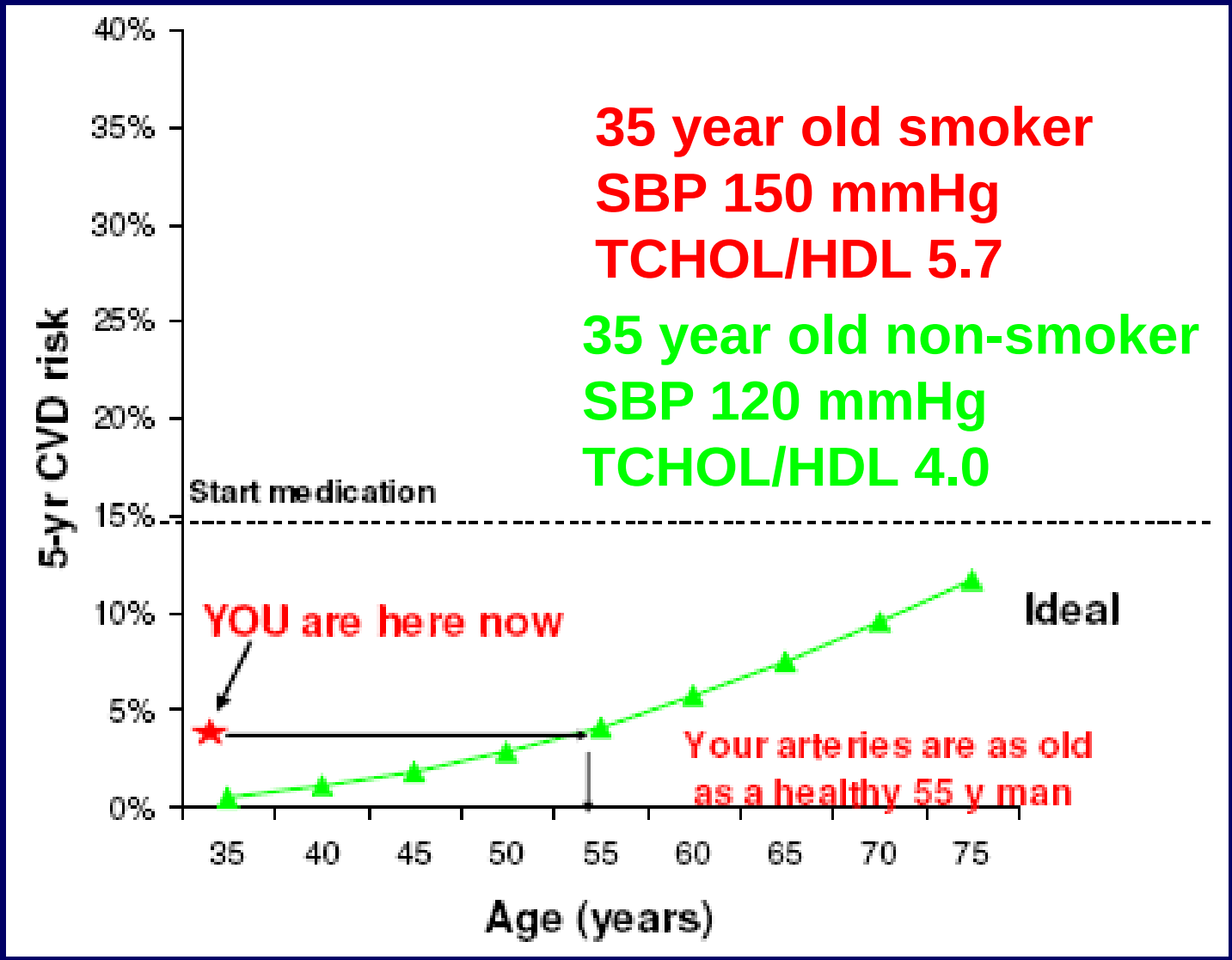
Prevalence Map of Raised Lesions of Abdominal Aorta by Age and Smoking



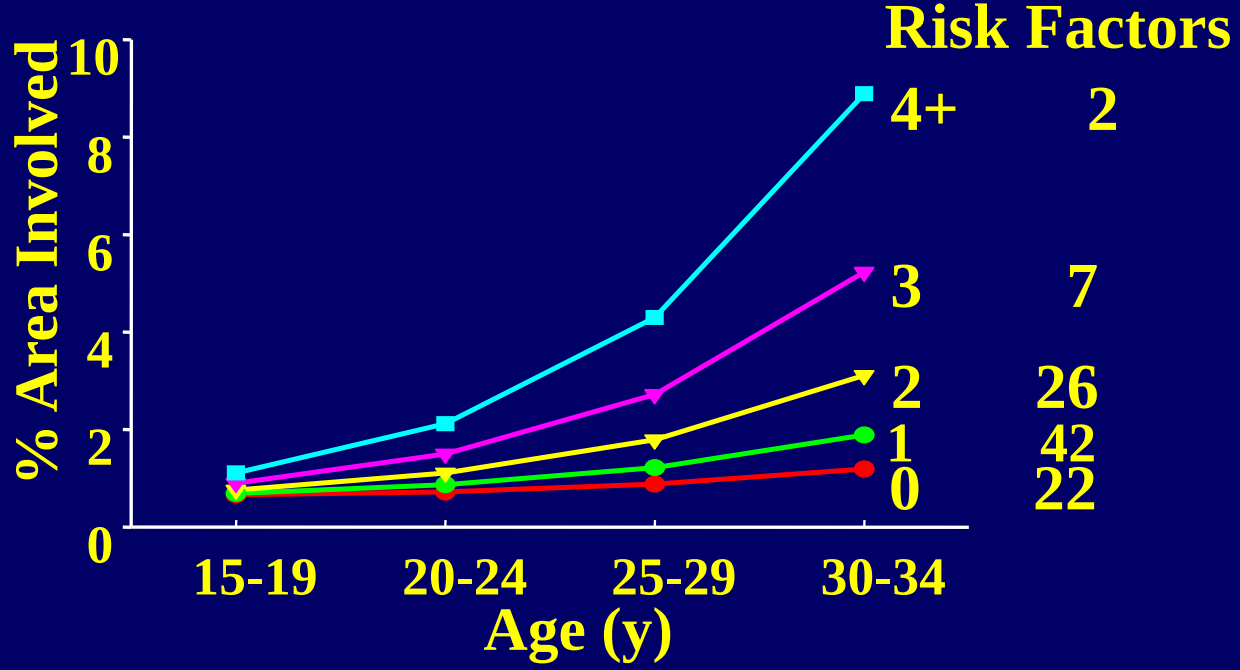
Right Coronary Artery Raised Lesions by Age and Number of Risk Factors, Men



Risk of cardiovascular events greater at a young age if you smoke etc

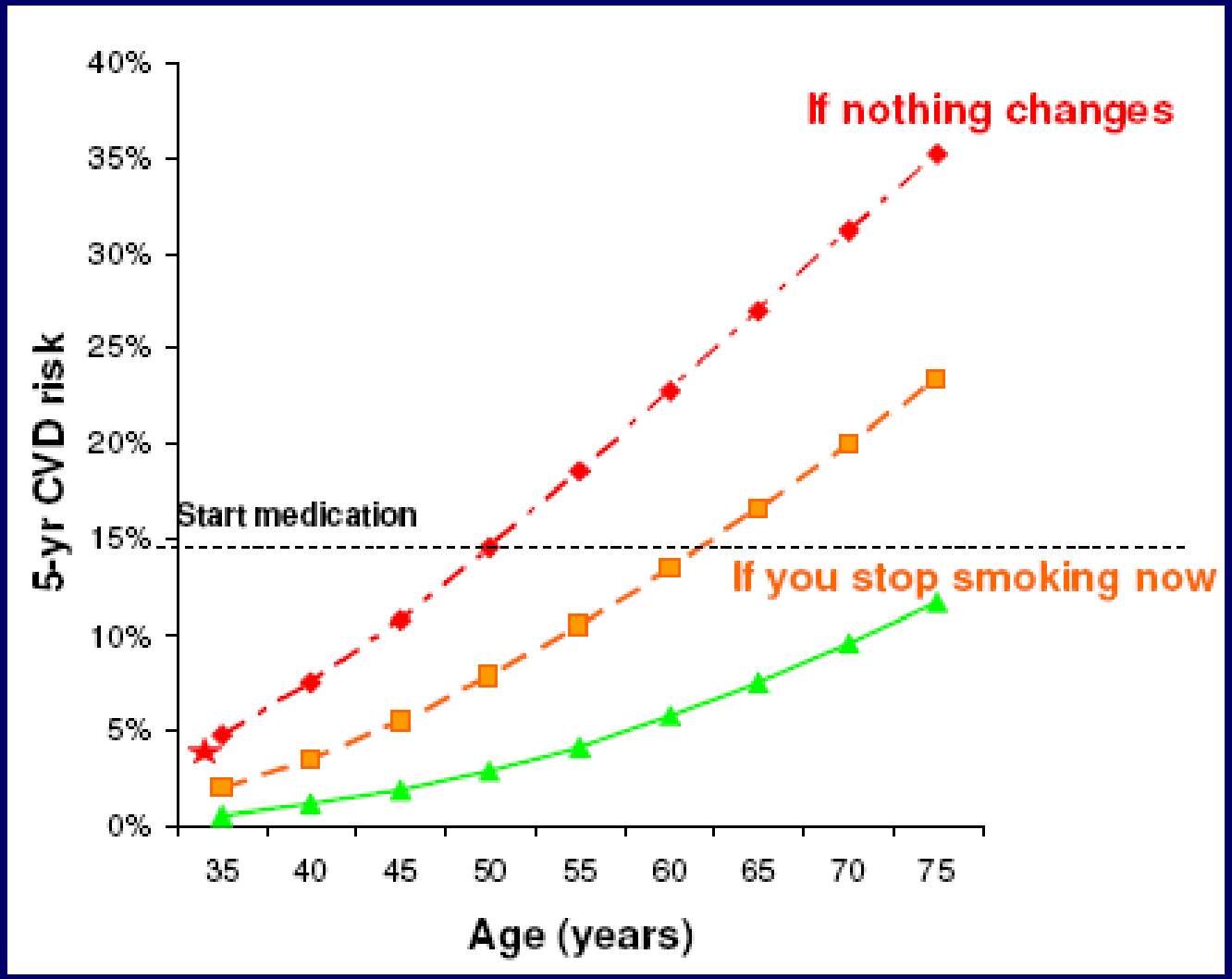


Remember the PDAY study.
In young men, the area of right coronary artery covered by raised lesions is proportional to the number of risk factors.

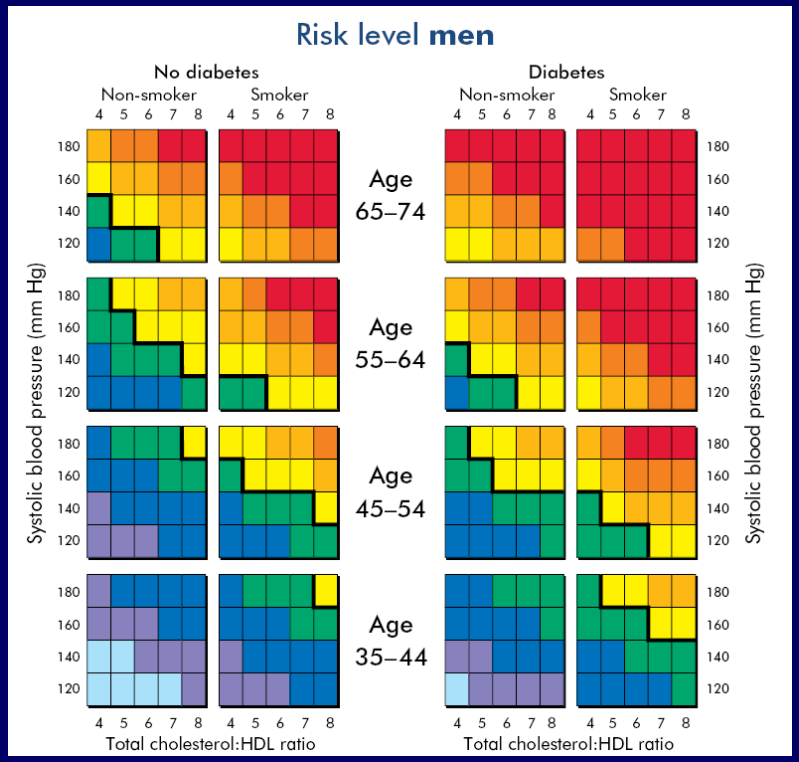


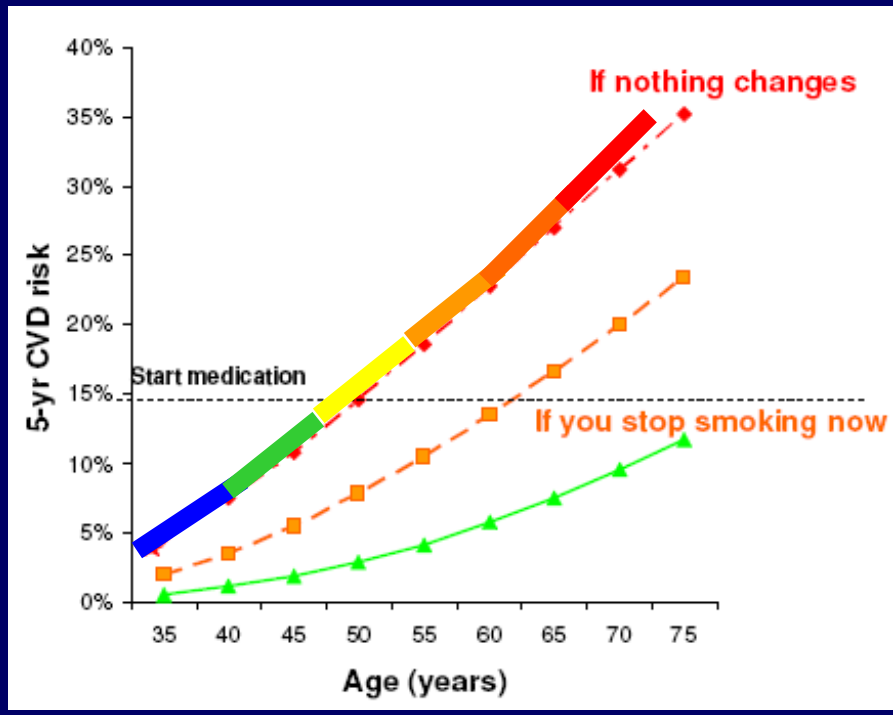
↑ plaque at earlier age, so more plaque to rupture

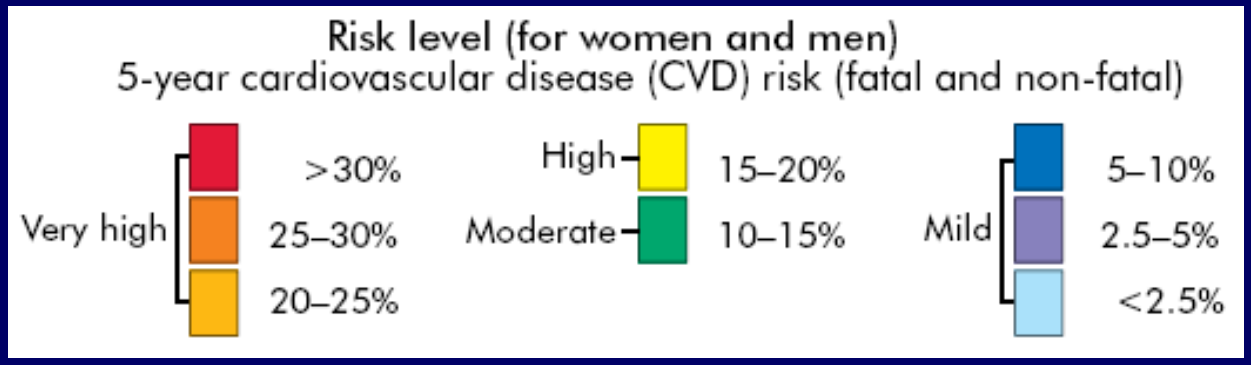
Increase in risk of an event increases with age, a steeper line if smoker etc.



At “low” risk when young but dead by 60







Minimise atherosclerotic plaque

- Prevention should have begun at school
- Screening at age of 46 has missed the boat.
- His arteries are loaded

Life before a heart attack

- “Sudden death is Nature’s way of telling us to slow down…”

  Woody Allen

Life before a heart attack

- "Sudden death is Nature's way of telling us to slow down…"

  Woody Allen

- If he has a heart attack, Rupert has a 50% chance of dying before reaching medical assistance

Zheng ZJ, Croft JB, Giles WH, Mensah, GA. Sudden cardiac death in the United States, 1989 to 1998. *Circulation*, 2001;104:2158–2163.

Rupert

- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- Probably has the arteries of a 65 year old

Rupert

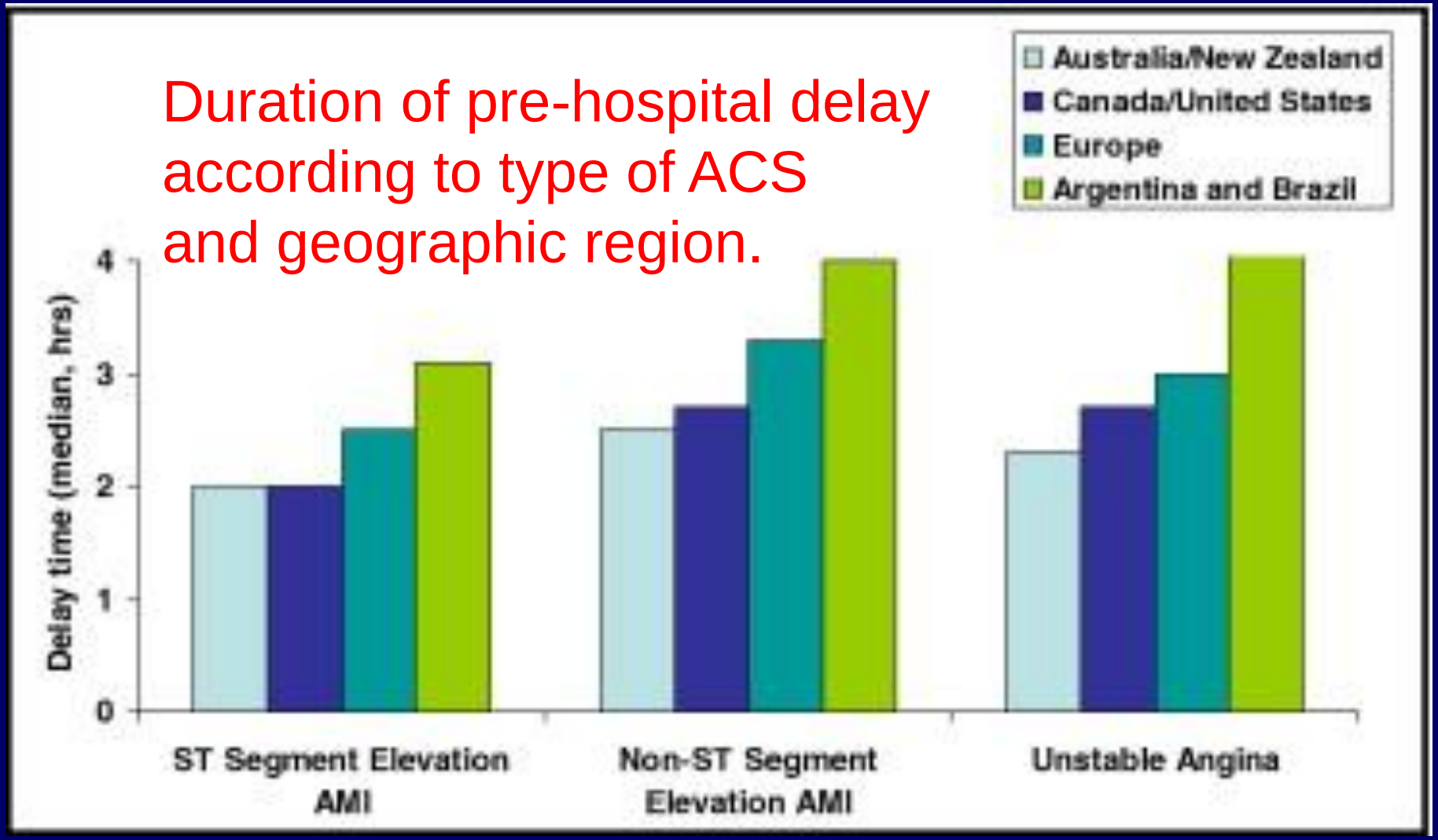
- Rupert
- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- Probably has the arteries of a 65 year old
- Watching Canterbury beat Otago during an afternoon ITM game
- Develops retrosternal chest pain started in his chest at 3pm, went down his arm and into his lower jaw.
- Quickeze hasn't helped.
- Wife called the ambulance at 530pm.

Death after the pain begins

- Often a long delay between symptom onset and first medical contact
- An opportunity to die

Delay to presentation

Duration of pre-hospital delay according to type of ACS and geographic region.



GRACE Study. Goldman et al

Delay to presentation

Variable	n	Time to contact health professional	Time to arrive at hospital
All patients	100	90 minutes (0–9600)	228 minutes (33–9630)
Telephoned hospital	4	81 minutes (5–150)	170 minutes (90–248)
Presented directly to hospital	8	72 minutes (5–2385)	72 minutes (5–2385)
Telephoned GP	14	15 minutes (0–1740)	305 minutes (34–2505)
Presented to GP	33	300 minutes (0–4140)	485 minutes (115–4340)
Telephoned ambulance		39 minutes	112 minutes (0–1072)

Table 4. Activation of emergency medical services (EMS) Median time taken in minutes (range) for patients to contact a health professional, or to arrive at hospital from the time of symptom onset. Values are given for all patients, and by mode of contacting a health professional. Note that 2 patients contacted a health professional through private consultations and arrived at hospital 155 and 9630 minutes after symptom onset.

Tanner et al NZMJ, 21-April-2006, Vol 119 No 1232

Case 1:46M

- Builder, ex Otago lock, smoker, Dad died at 47 of MI
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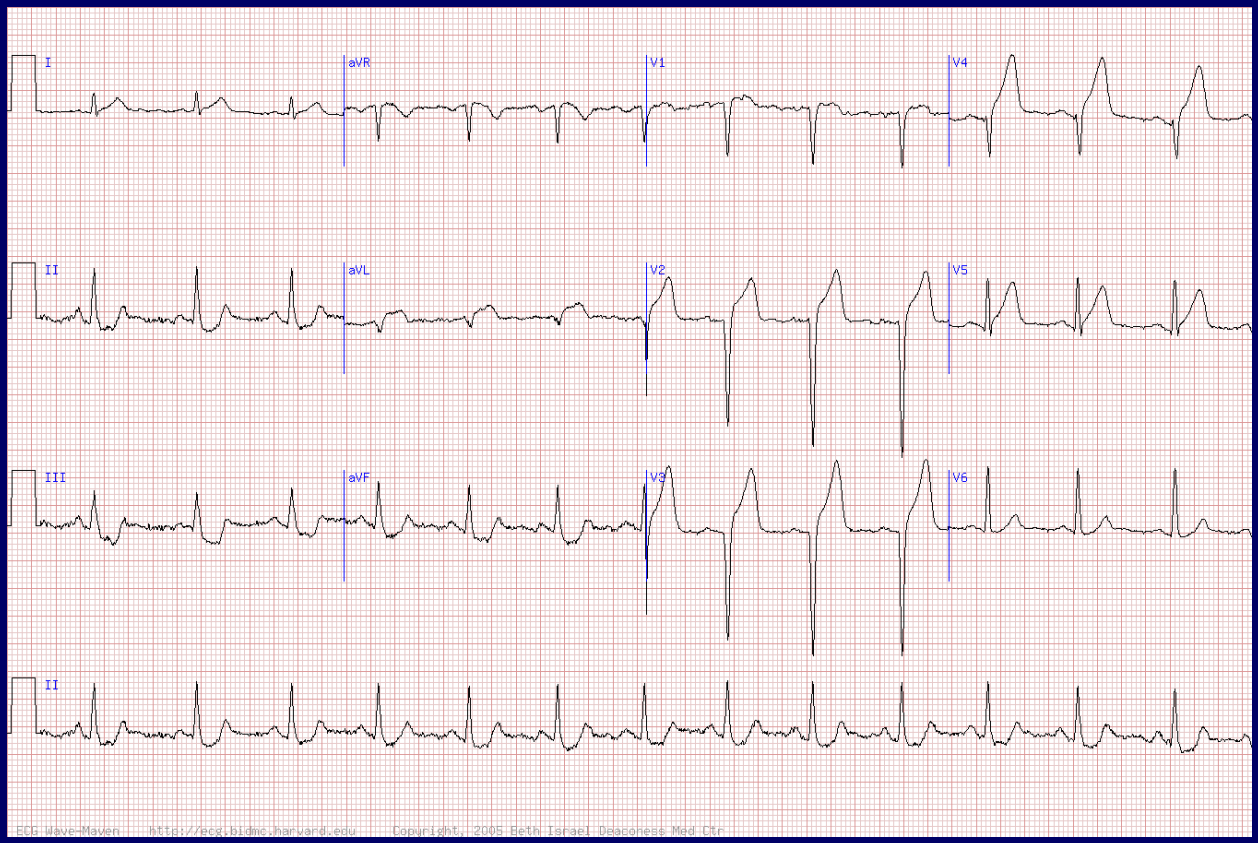
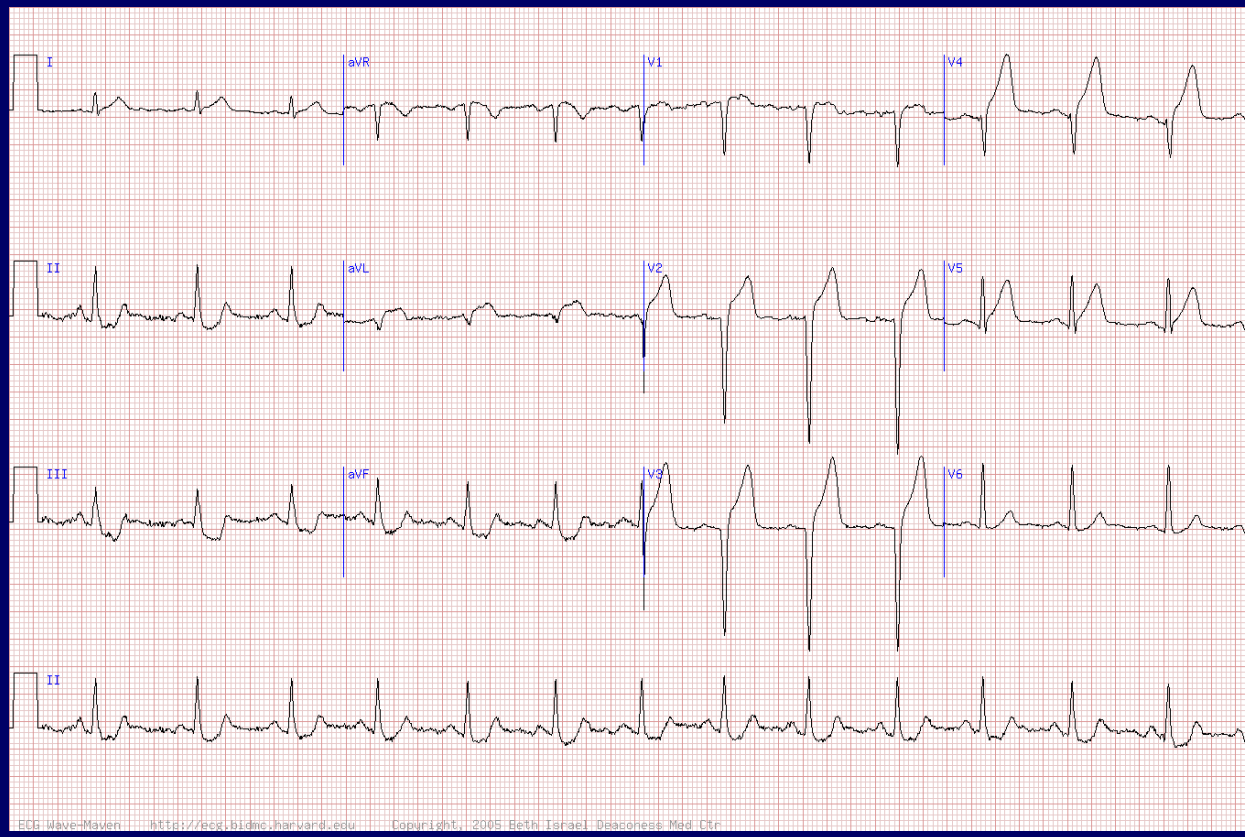
Case 1:46M

- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- ED at 5:52pm
- ECG at 6pm



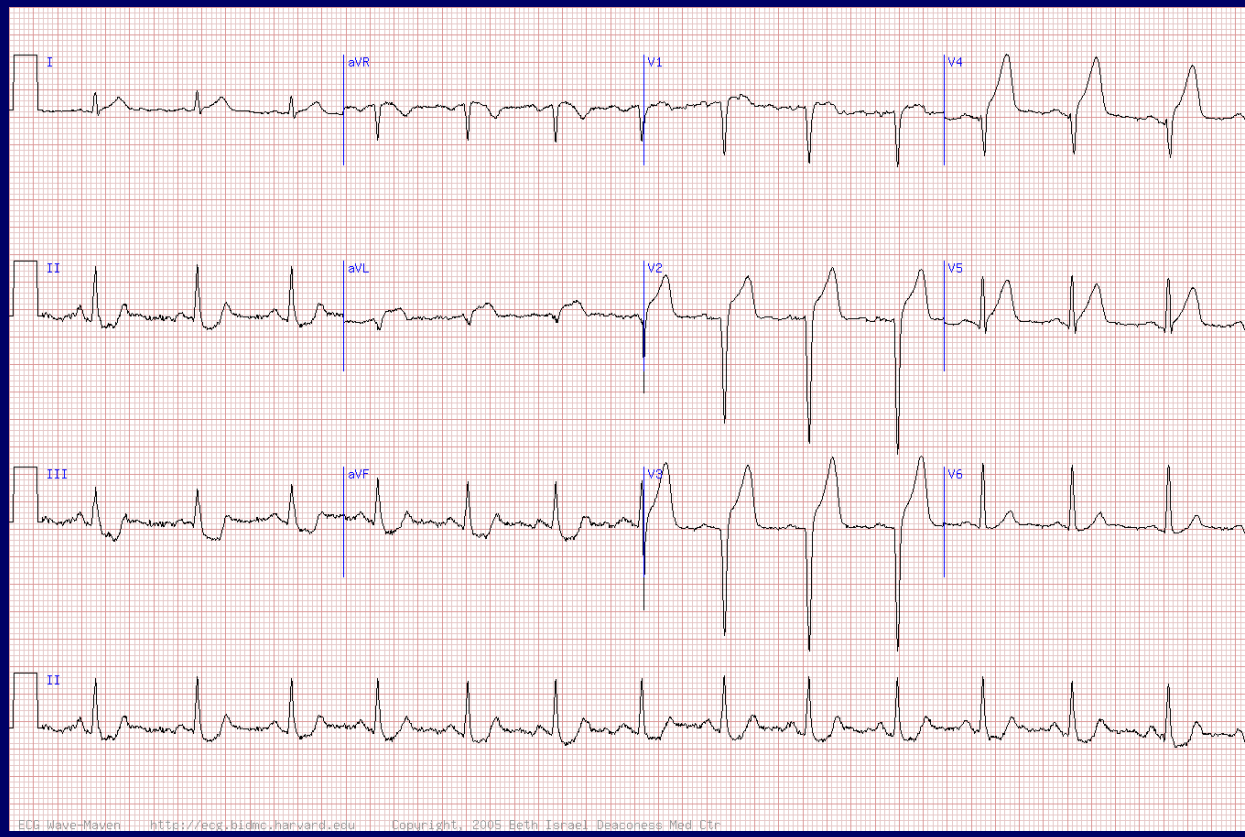
Case 1:46M

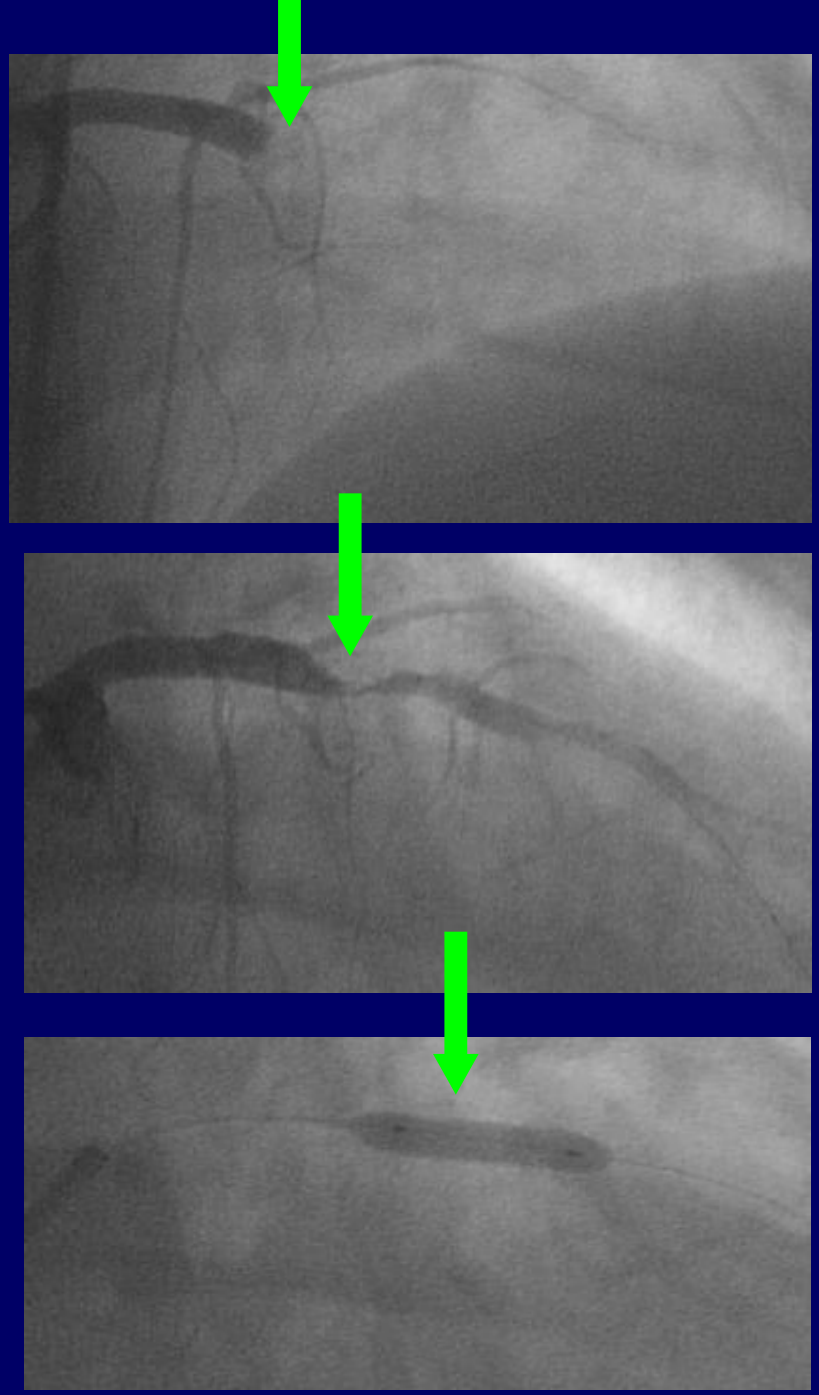
- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- Anterior STEMI:
- What next?



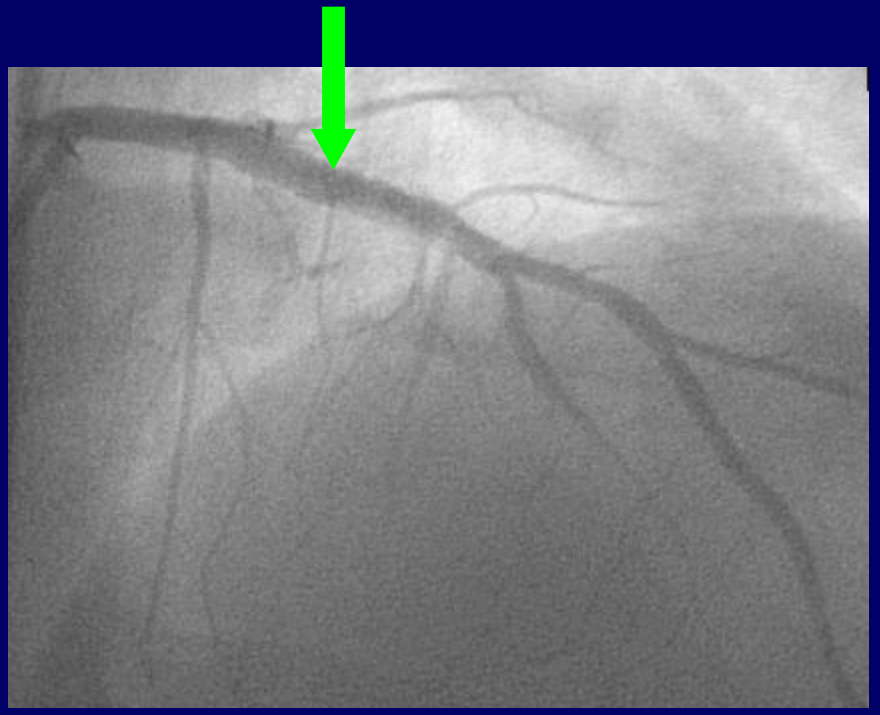
Case 1:46M

- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- Anterior STEMI:
- Aspirin
- Clopidogrel
- Either
- Angiogram or
- Thrombolysis
- hsTNT 58
- CK 103



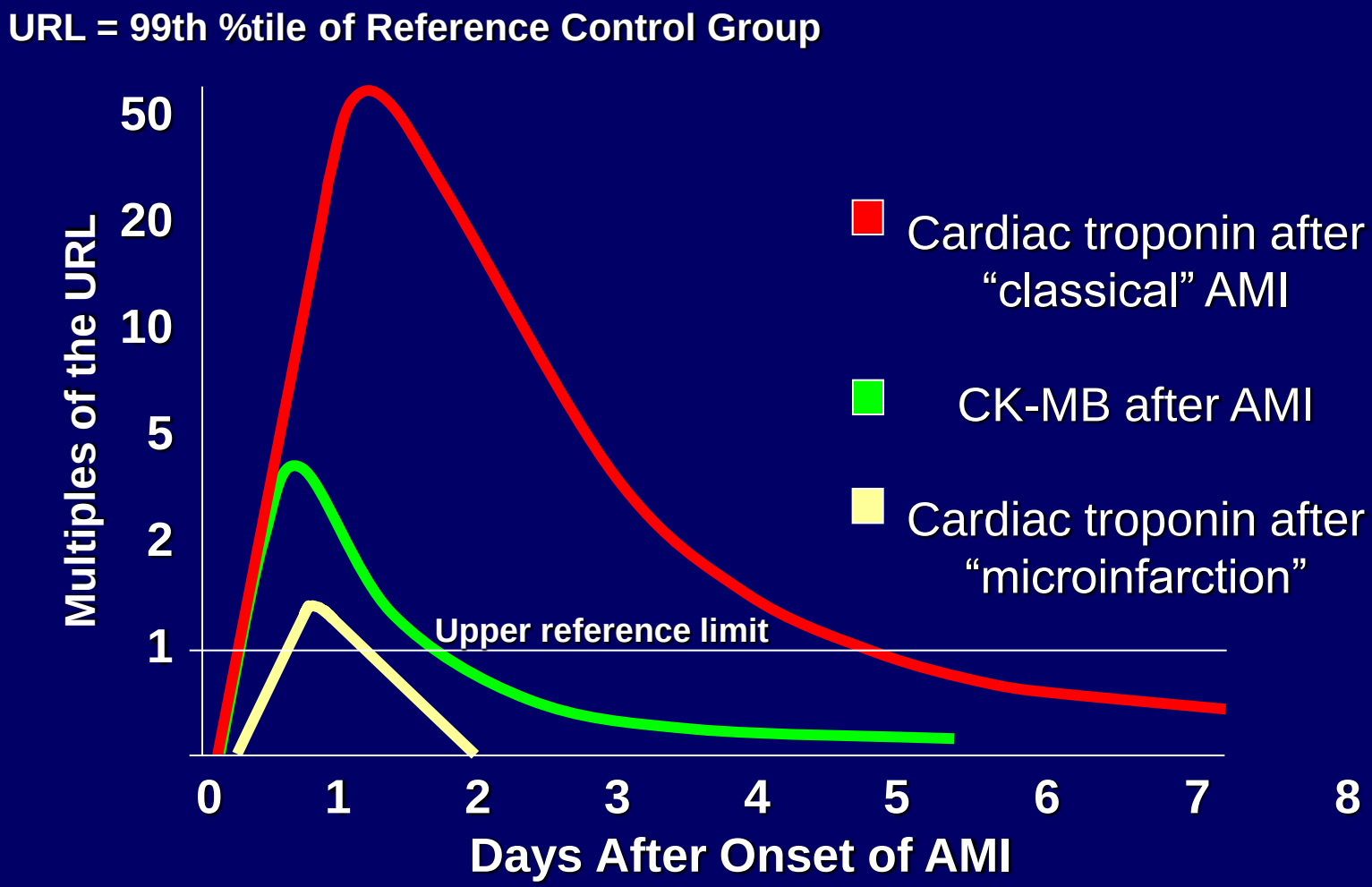


One drug eluting stent to occuded LAD for acute anterior STEMI



Team called at 610
Artery open at 7pm
Other vessels OK

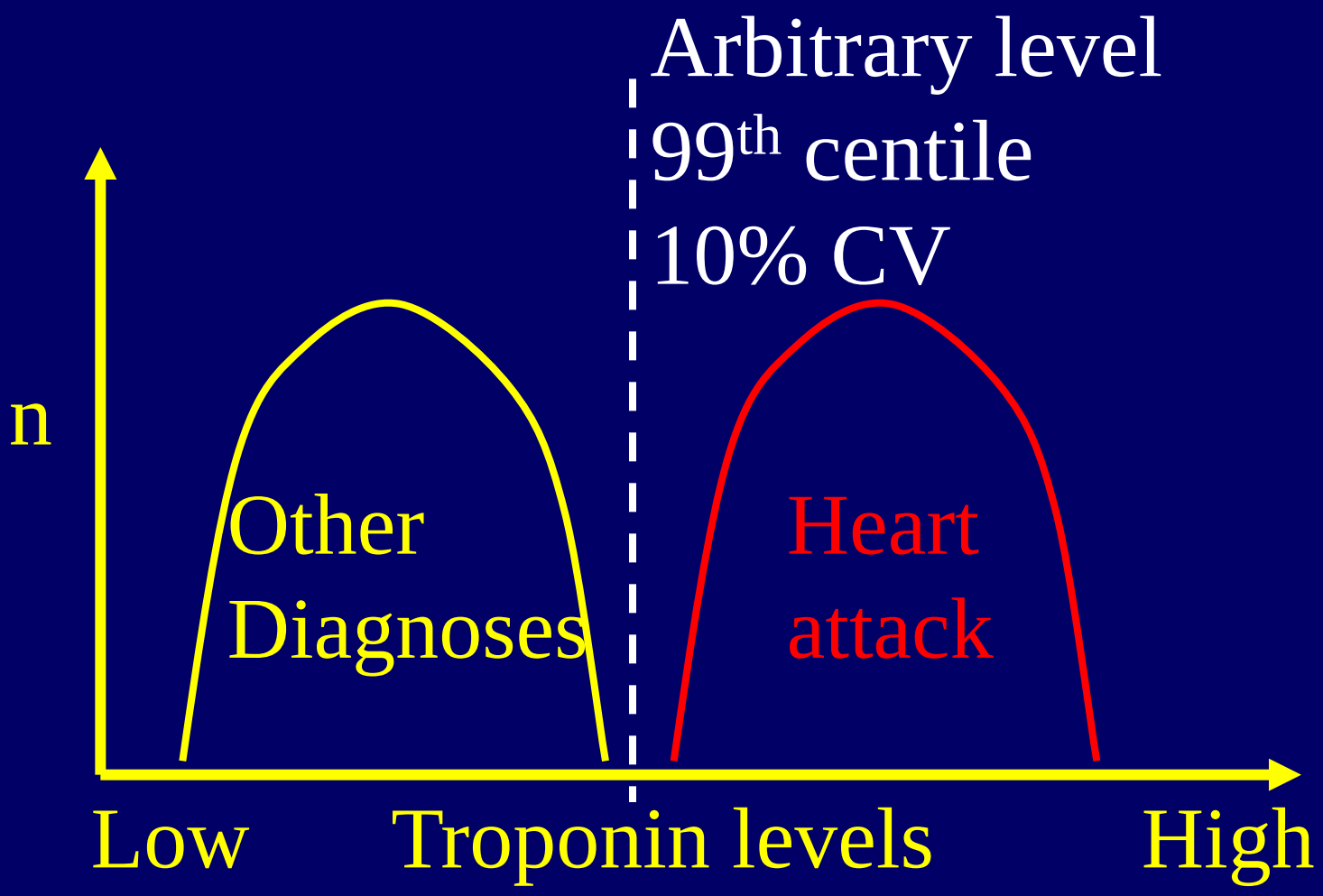
Cardiac Markers in ACS



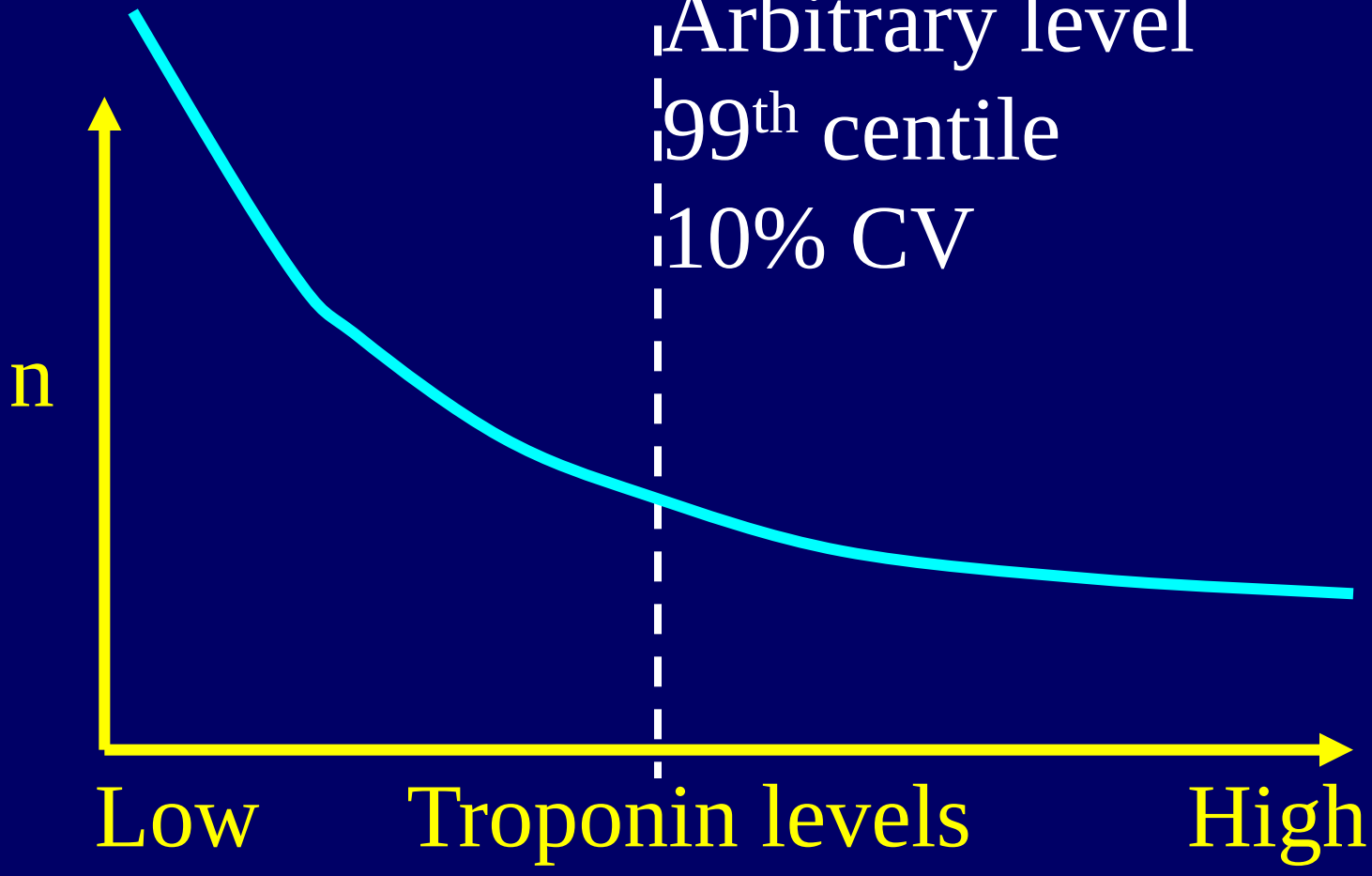
Modified from:
ESC/ACC Comm "MI redefined…" JACC 36: 959,2000

Wu AH et al. Clin Chem 1999;45:1104.

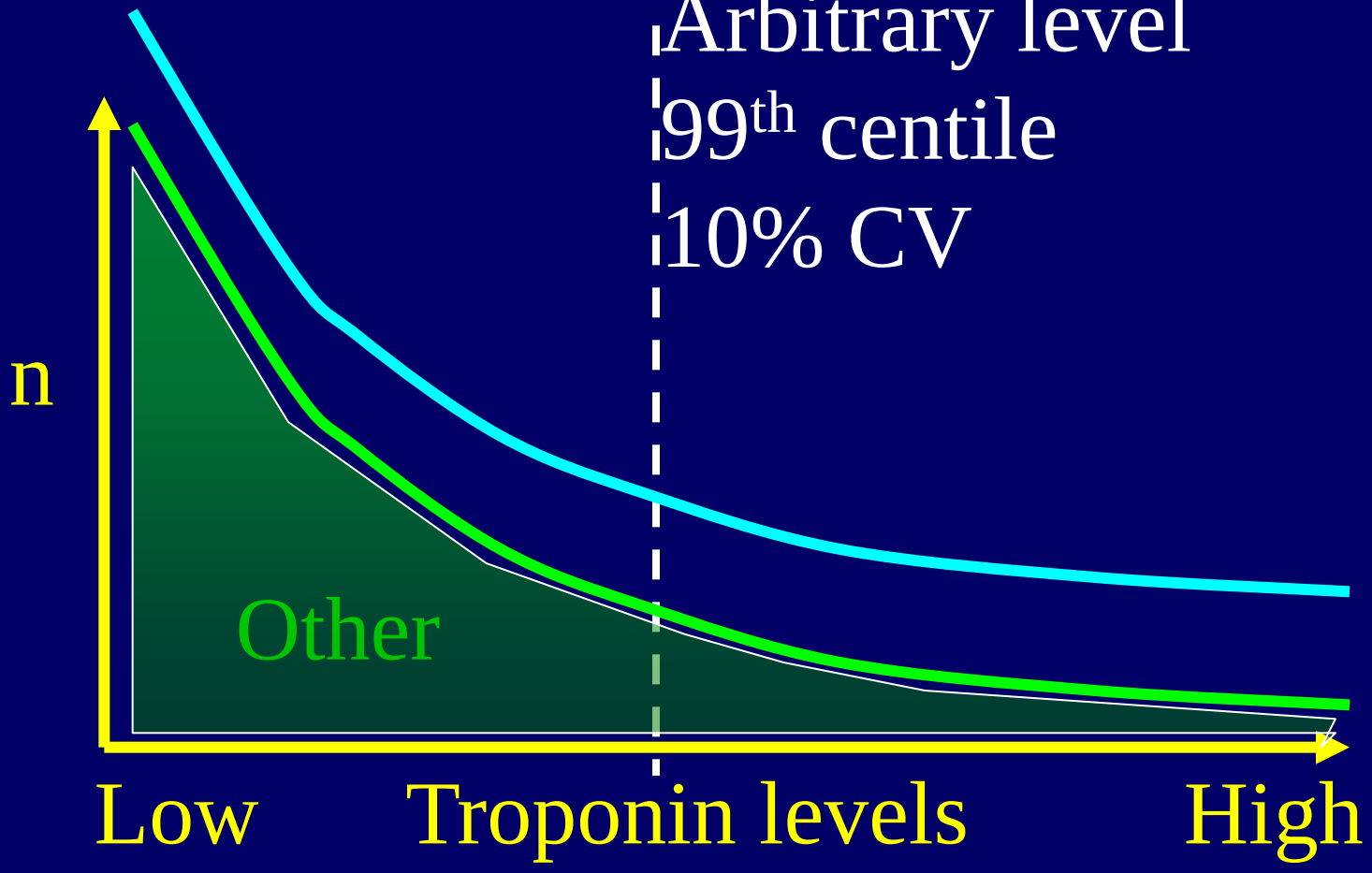
Diagnosis : The dream



Reality


Arbitrary level
99th centile
10% CV
n
Low
Troponin levels
High

Reality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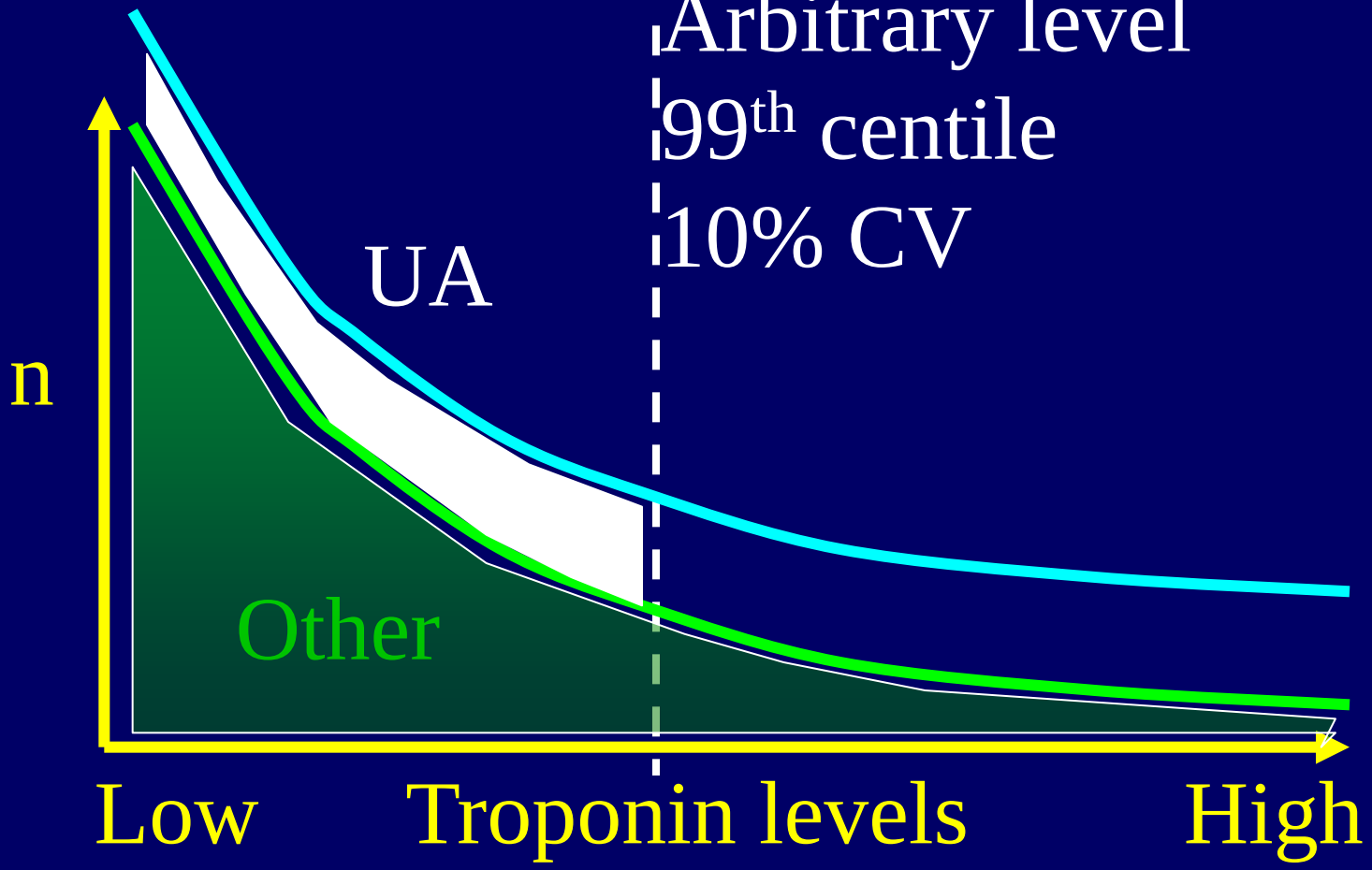
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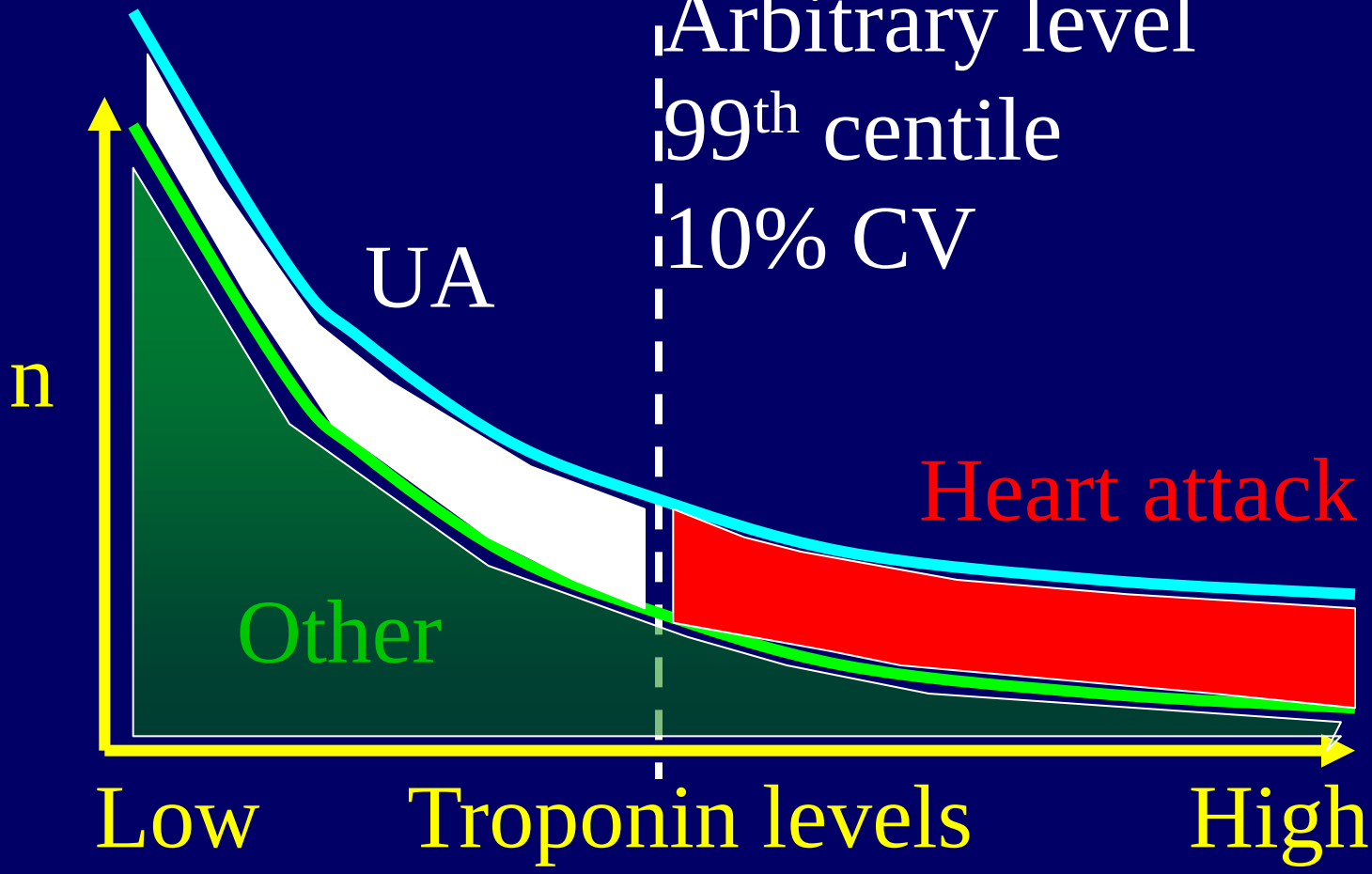
Reality


Arbitrary level
99th centile
10% CV
UA
n
Heart attack
Other
Low
Troponin levels
High

The troponin is raised – what next?

- Find a synapse
- Think

Differential diagnosis of rise in troponins

Table 2 Elevations of troponin in the absence of overt ischemic heart disease

Cardiac contusion, or other trauma including surgery, ablation, pacing, etc.
Congestive heart failure—acute and chronic
Aortic dissection
Aortic valve disease
Hypertrophic cardiomyopathy
Tachy- or bradyarrhythmias, or heart block
Apical ballooning syndrome
Rhabdomyolysis with cardiac injury
Pulmonary embolism, severe pulmonary hypertension
Renal failure
Acute neurological disease, including stroke or subarachnoid haemorrhage
Infiltrative diseases, e.g. amyloidosis, haemochromatosis, sarcoidosis, and scleroderma
Inflammatory diseases, e.g. myocarditis or myocardial extension of endo-/pericarditis
Drug toxicity or toxins
Critically ill patients, especially with respiratory failure or sepsis
Burns, especially if affecting >30% of body surface area
Extreme exertion

The troponin is up
Differential diagnosis

PLEASE

- PE
- LVH
- Extremes or BP/HR
- A
- Spasm
- Exercise

THINK

- Trauma/toxins
- Heart failure
- Inflam/infection
- Neuro –CVA,SAH
- Kidney disease

5 types of myocardial infarction

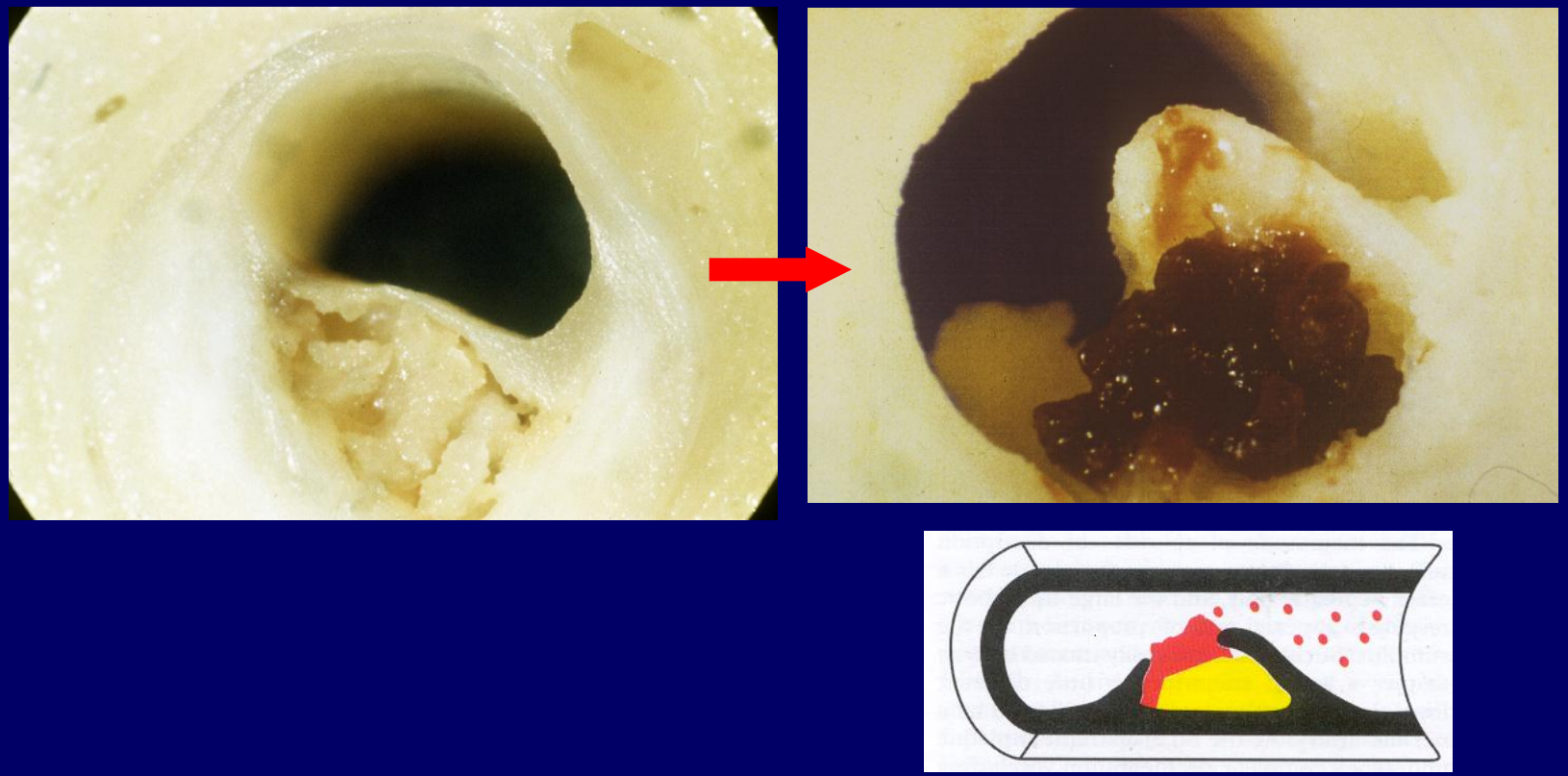
(2007)

Table 1 Clinical classification of different types of myocardial infarction

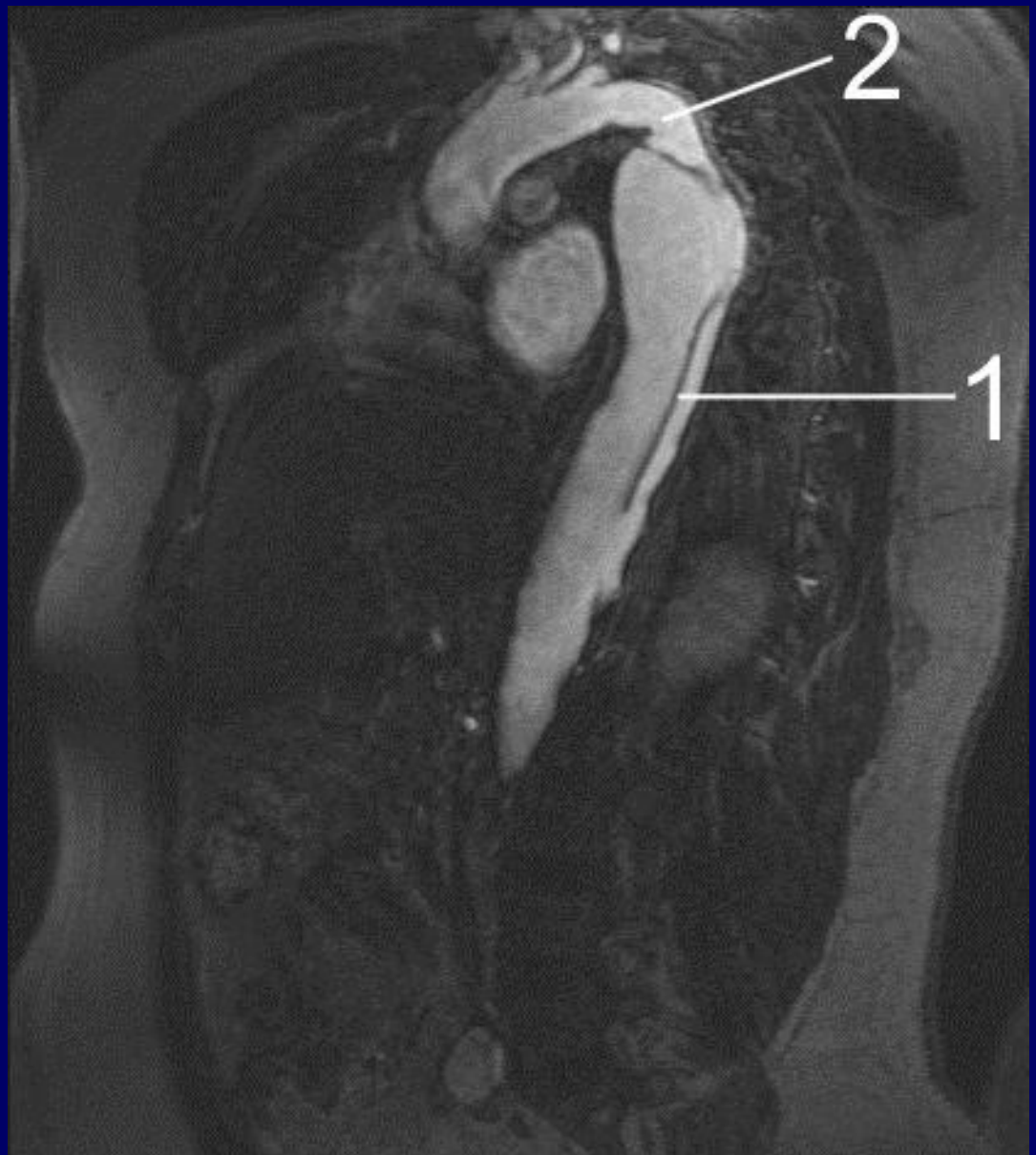
Type 1	Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection
Type 2	Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension
Type 3	Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischaemia, accompanied by presumably new ST elevation, or new LBBB, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood
Type 4a	Myocardial infarction associated with PCI
Type 4b	Myocardial infarction associated with stent thrombosis as documented by angiography or at autopsy
Type 5	Myocardial infarction associated with CABG

Type of myocardial infarction

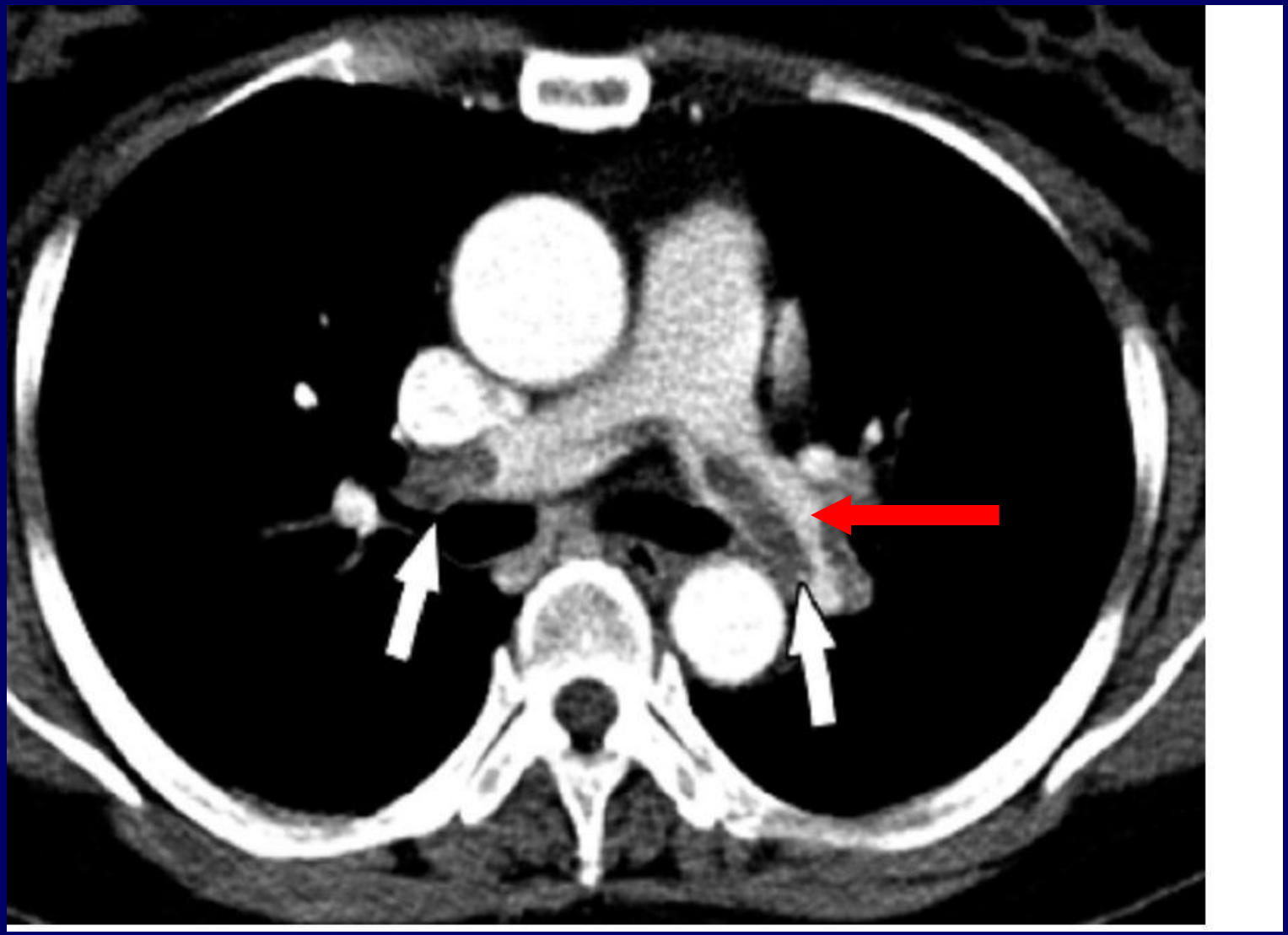
Type 1 **Spontaneous MI related to ischemia due to a primary coronary event, such as plaque erosion and/or rupture, fissuring, or dissection**



Aortic dissection



Pulmonary embolus



Consequences of not exploring differential diagnosis of raised troponins

Lets get back to Rupert

- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- Anterior STEMI: LAD blocked on acute angiogram stented with 1 drug eluting stent, other vessels OK
- Aspirin, Clopidogrel, Metoprolol, Atorvastatin, Cilazapril
- No recurrent pain, evolves anterior Q waves
- ECHO. Why?

Rupert

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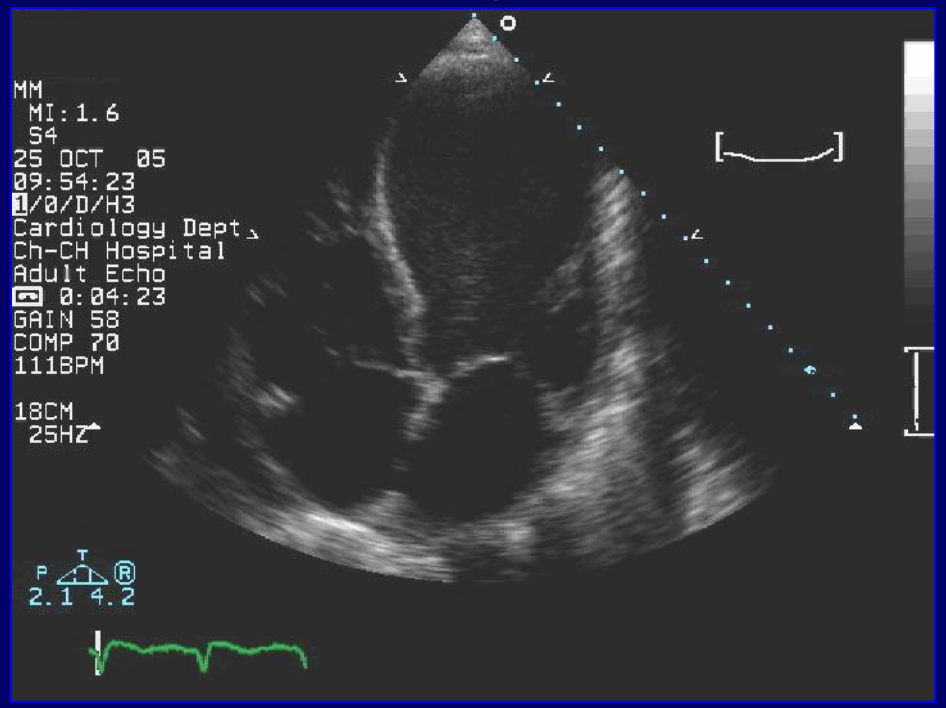

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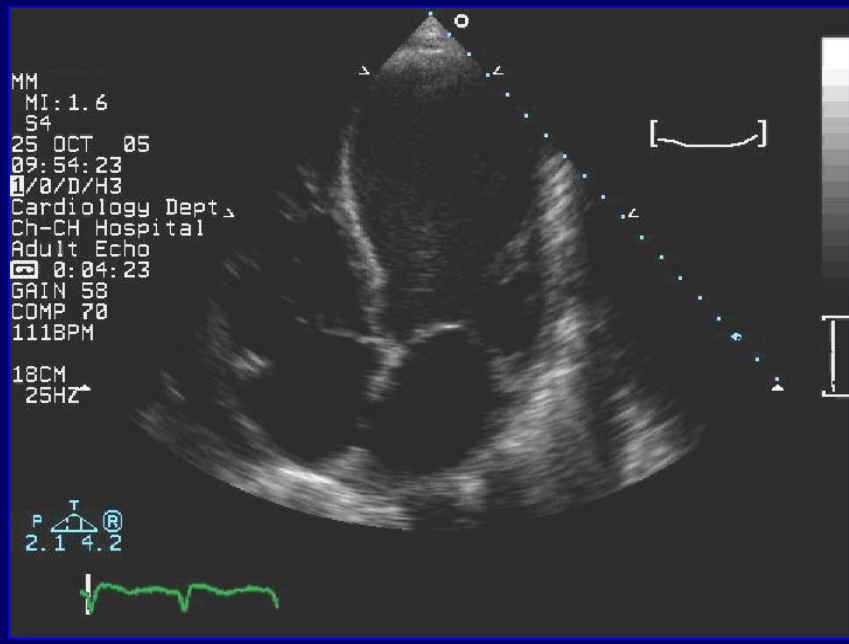
- ECHO: Left ventricle dilated
- LVEF 30% with anterior and apical hypokinesis.
- Valves OK



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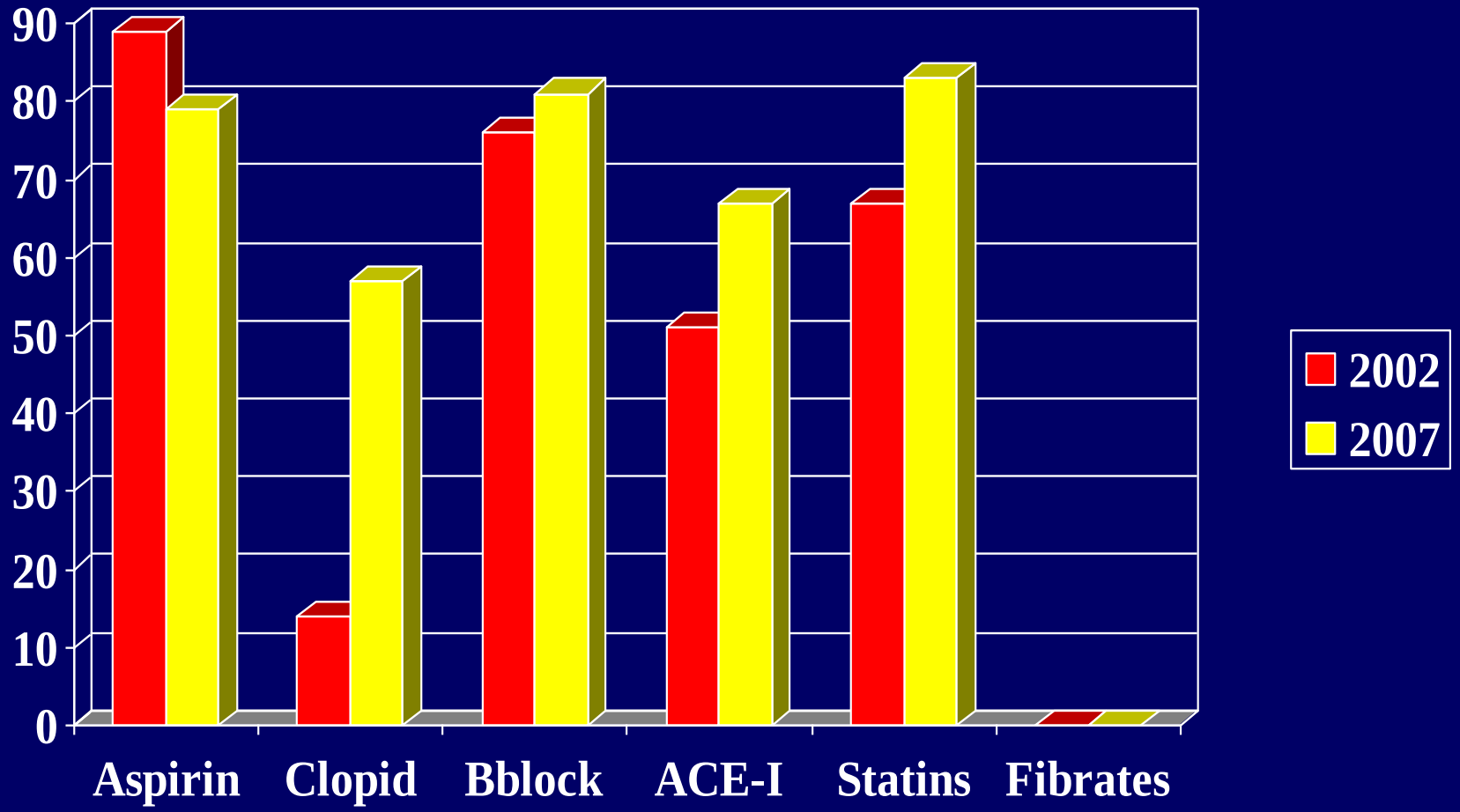
- No recurrent pain, evolves anterior Q waves
- ECHO: Left ventricle dilated and LVEF 30% with anterior and apical ischaemia. Valves OK
- Medications during admission – aspirin, clopidogrel, metoprolol, atorvastatin, cilazapril
- Medications at discharge –

Rupert

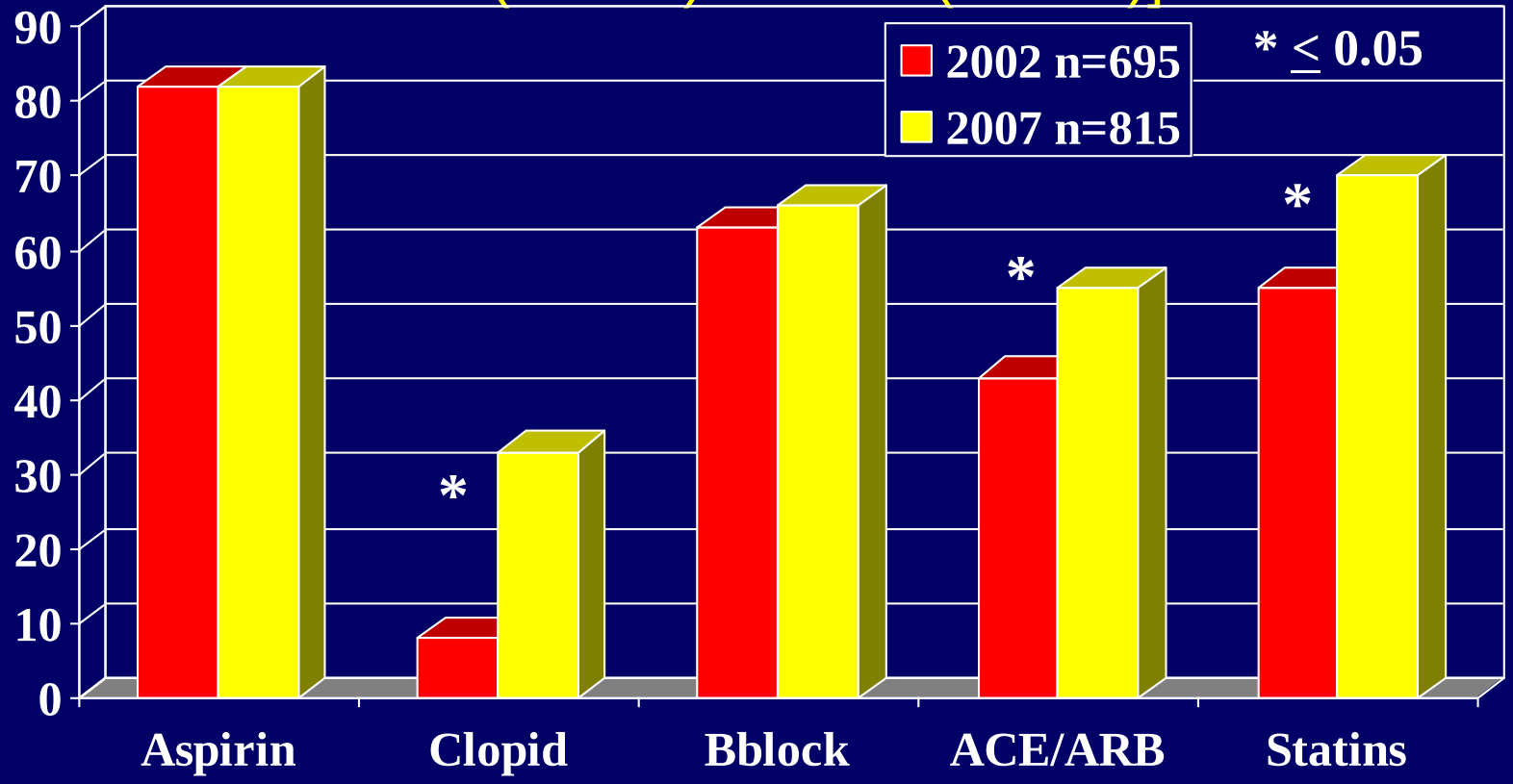
- Builder, ex Otago lock, smoker, Dad died at 47 of MI
- BMI 32, TCHOL 6.9, LDL 4.3, TG 4.2, HDL 0.8
- Anterior STEMI: LAD blocked on acute angiogram stented with 1 drug eluting stent, other vessels OK
- No recurrent pain, evolves anterior Q waves
- ECHO Left ventricle dilated and LVEF 30% with anterior and apical ischaemia. Valves OK
- Medications at discharge –
  - Aspirin 100mg and Clopidogrel 75mg
  - Atorvastatin 40mg mane, + dietary advice
  - Metoprolol 47.5mg mane
  - Cilazapril 1mg mane
  - Nicotine patch

1st & 2nd NZ ACS Audits: Discharge Medications – STEMI Pts

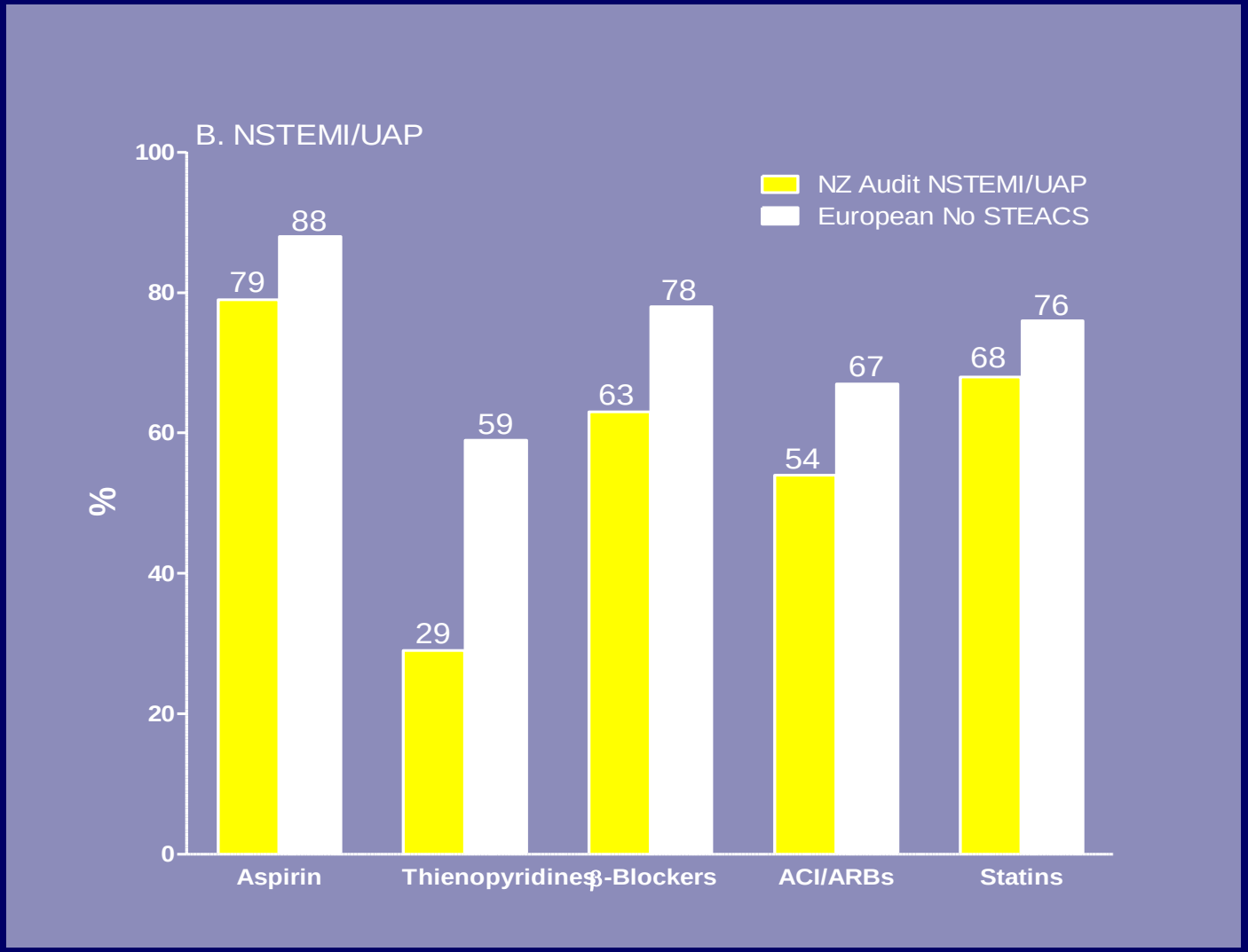


1st & 2nd NZ ACS Audits: Discharge Medications

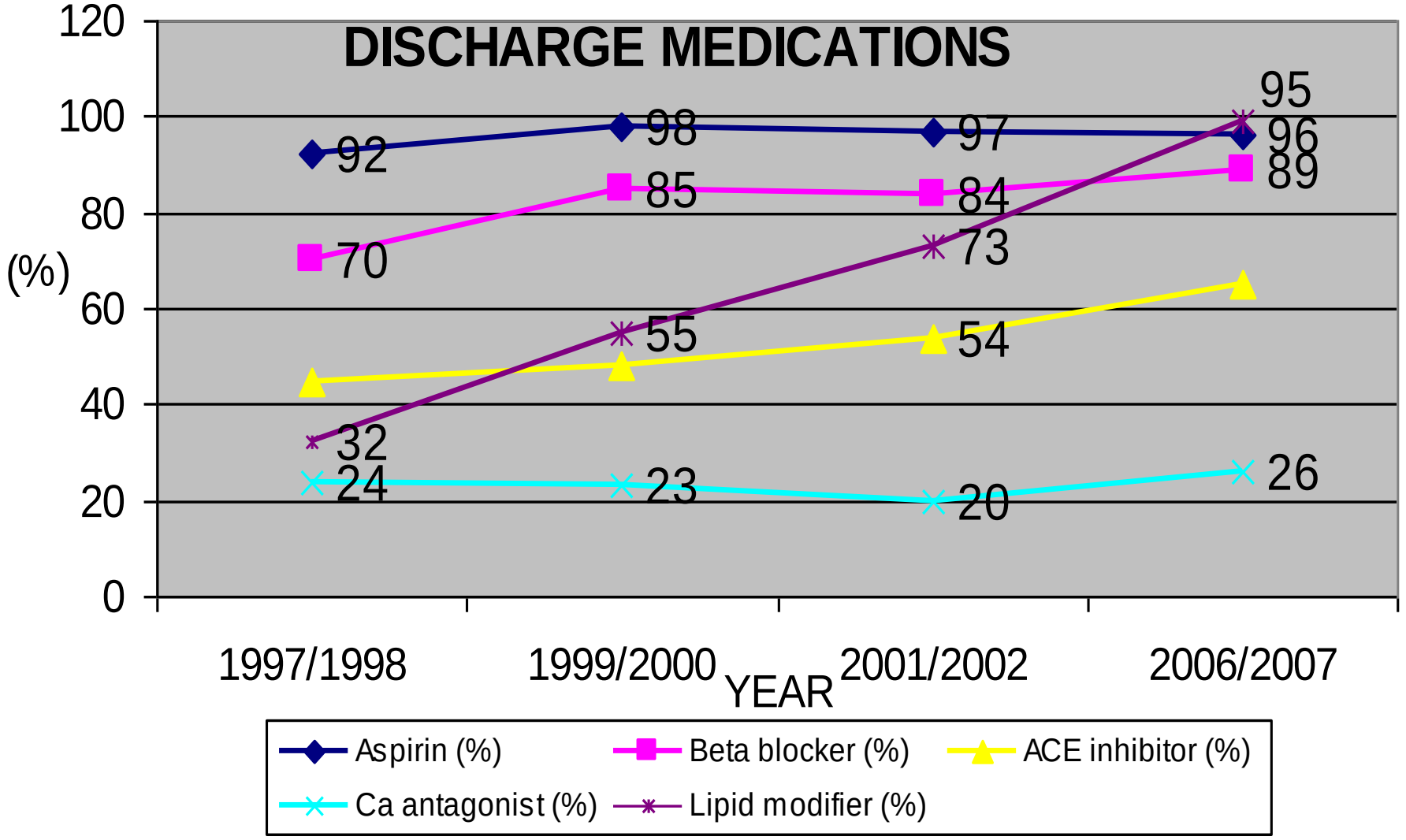
[All 'Definite' ACS Pts (STEMI/Non-STEMI/UAP): 2002 (n=721) & 2007 (n=874)]



ACS Audits: NZ (2007) vs Europe (2004)
Discharge Medications



Christchurch 1997-2006



Rupert. Follow-up by you

- Rehab class – cant attend because of work
- Short of breath lifting but no pain. BP 130/80, p 72
- “Do I still needs these pills Doc?”

The benefit of continued medication

Hazard ratios for mortality in patients discharged on combinations of statins and beta-blockers after adjusting to the GRACE score

	1-year	2-year	3-year	4-year
St without Bb	0.31 (0.09–1.01)	0.62 (0.29–1.34)	0.67 (0.34–1.34)	0.57 (0.29–1.12)
Both St and Bb	0.55 (0.30–1.00)	0.66 (0.41–1.08)	0.78 (0.50–1.20)	0.72 (0.48–1.09)
Neither St nor Bb	1.56 (0.88–2.76)	2.09 (1.31–3.34)	2.28 (1.48–3.53)	2.19 (1.46–3.29)

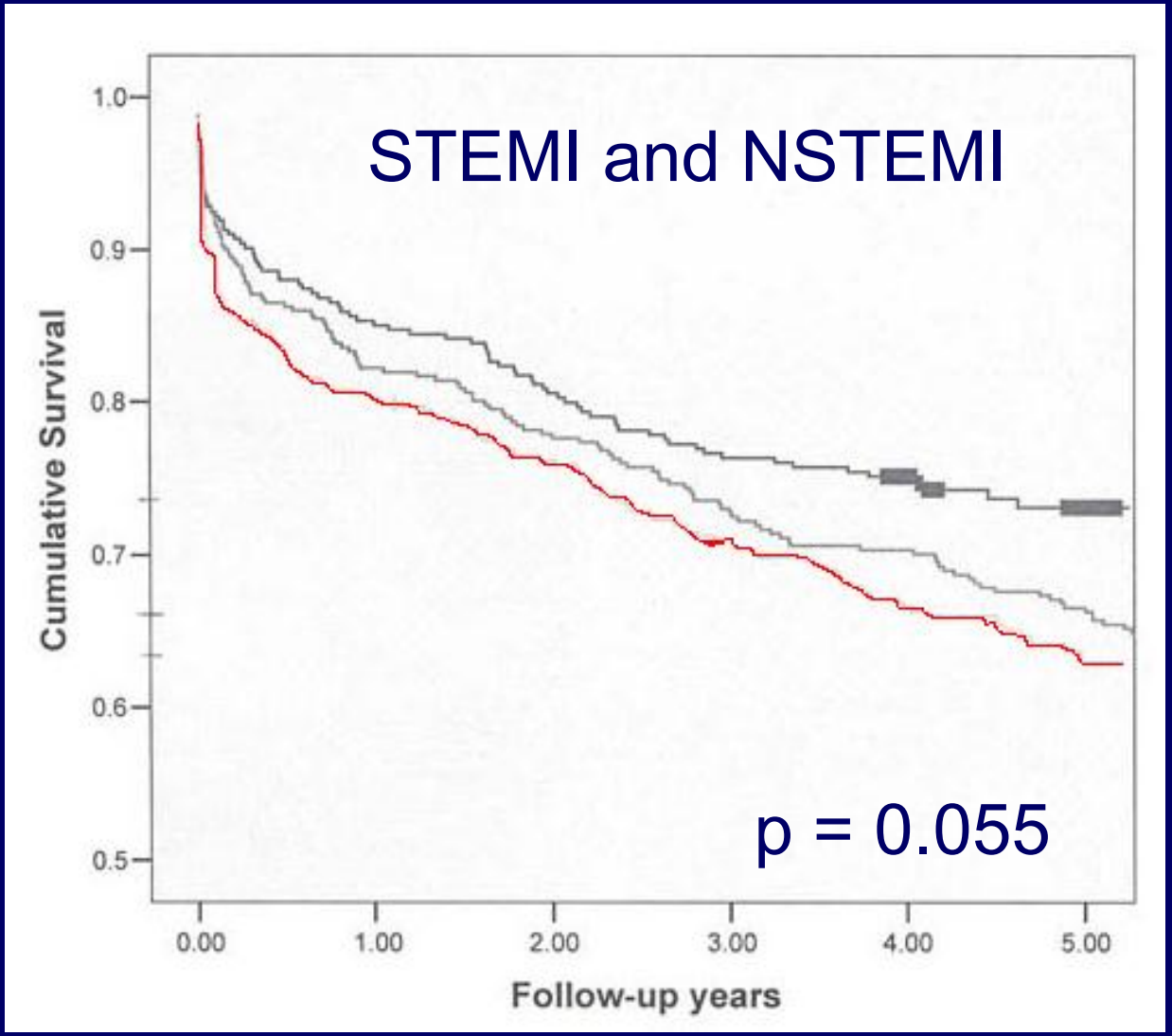
Bb = Beta-blocker, St = Statins.

All patients were on aspirin. Sub-grouping was done regardless of the use of ACE-inhibitors/angiotensin receptor blockers.

Reference group is the patients on Beta-blockers without statins.

Wong et al Int J Cardiol 2009

Survival after AMI - Christchurch



Admitted Oct 1-Dec 31
Case note review
PMS

74%, 2001/02, n=334

66%, 1997/98, n=371

64%, 1999/00, n=371

Anna Mulholland, Summer Student

Rupert. Follow-up at 4 weeks

- Rehab class – wont attend because of work
- No pain. “Do I still needs these pills Doc?”
- Follow-up lipids
- TCHOL 5.0, LDL 3.0, TG 3.1, HDL 0.8

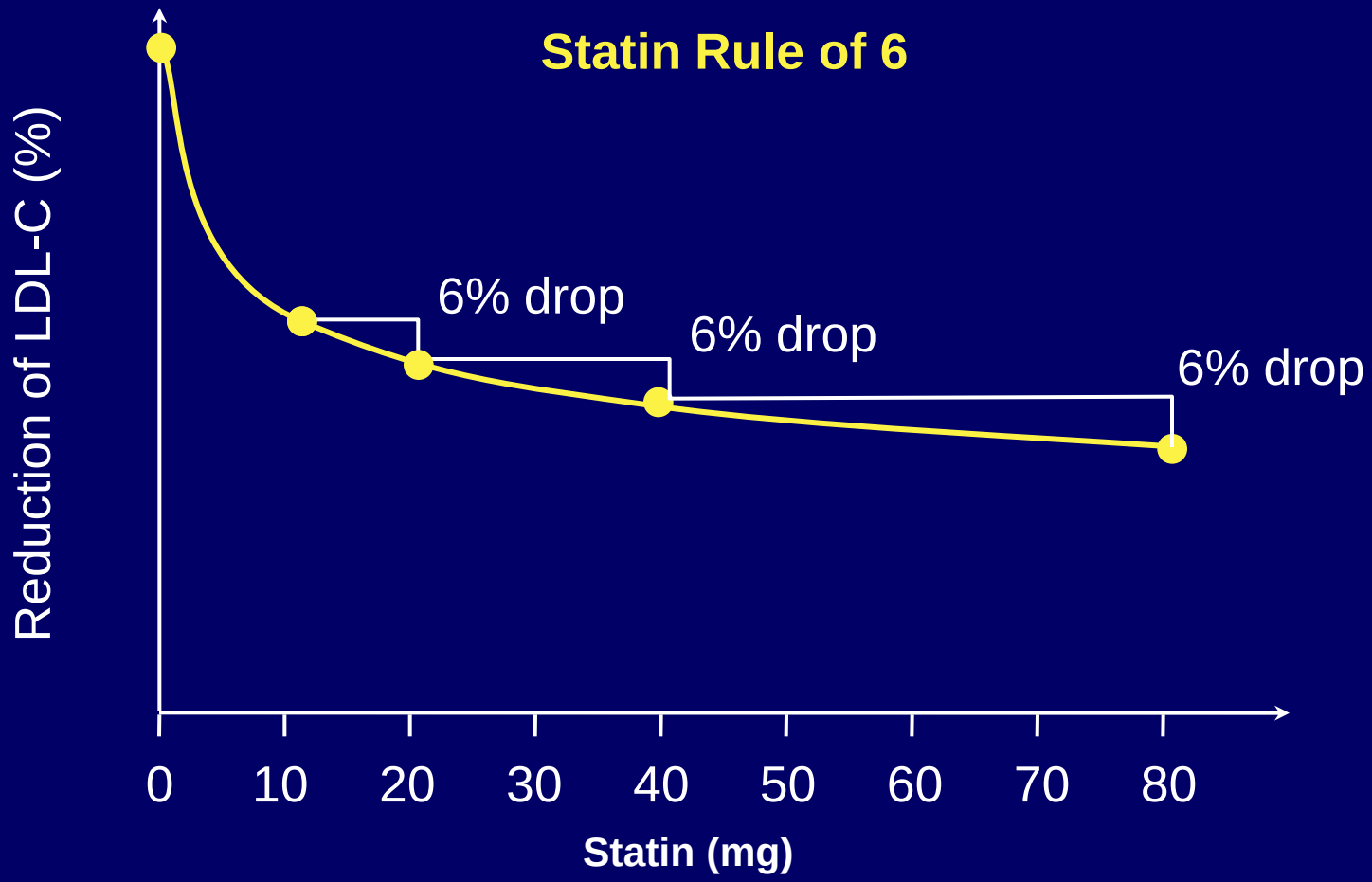
Lipid targets

Table 10	Optimal levels (targets) for people with known cardiovascular disease, or cardiovascular risk >15% or diabetes		
	Known cardiovascular disease or cardiovascular risk >15%	Diabetes	Diabetes and overt nephropathy, microalbuminuria or other renal disease
Lipids			
Total cholesterol	<4.0 mmol/L		
LDL cholesterol	<2.0 mmol/L		
HDL cholesterol	≥1.0 mmol/L		
TC:HDL ratio	<4.0		
Triglycerides	<1.7 mmol/L		
Blood pressure			
BP	<130/80 mm Hg	<130/80 mm Hg	<125/75 mm Hg

Rupert. Follow-up at 4 weeks

- Rehab class – cant attend because of work
- No pain. “Do I still needs these pills Doc?”
- Follow-up lipids
- TCHOL 5.0, LDL 3.0, TG 3.1, HDL 0.8
- Target is LDL <2.0mmol/L
- Double atorvastatin

Doubling a Statin Dose Yields Only a 6% Incremental Drop in LDL-C



Adapted from Knopp RH et al *N Engl J Med* 1999;341:498–509; Stein E *Am J Cardiol* 2002;89(suppl):50C–57C.

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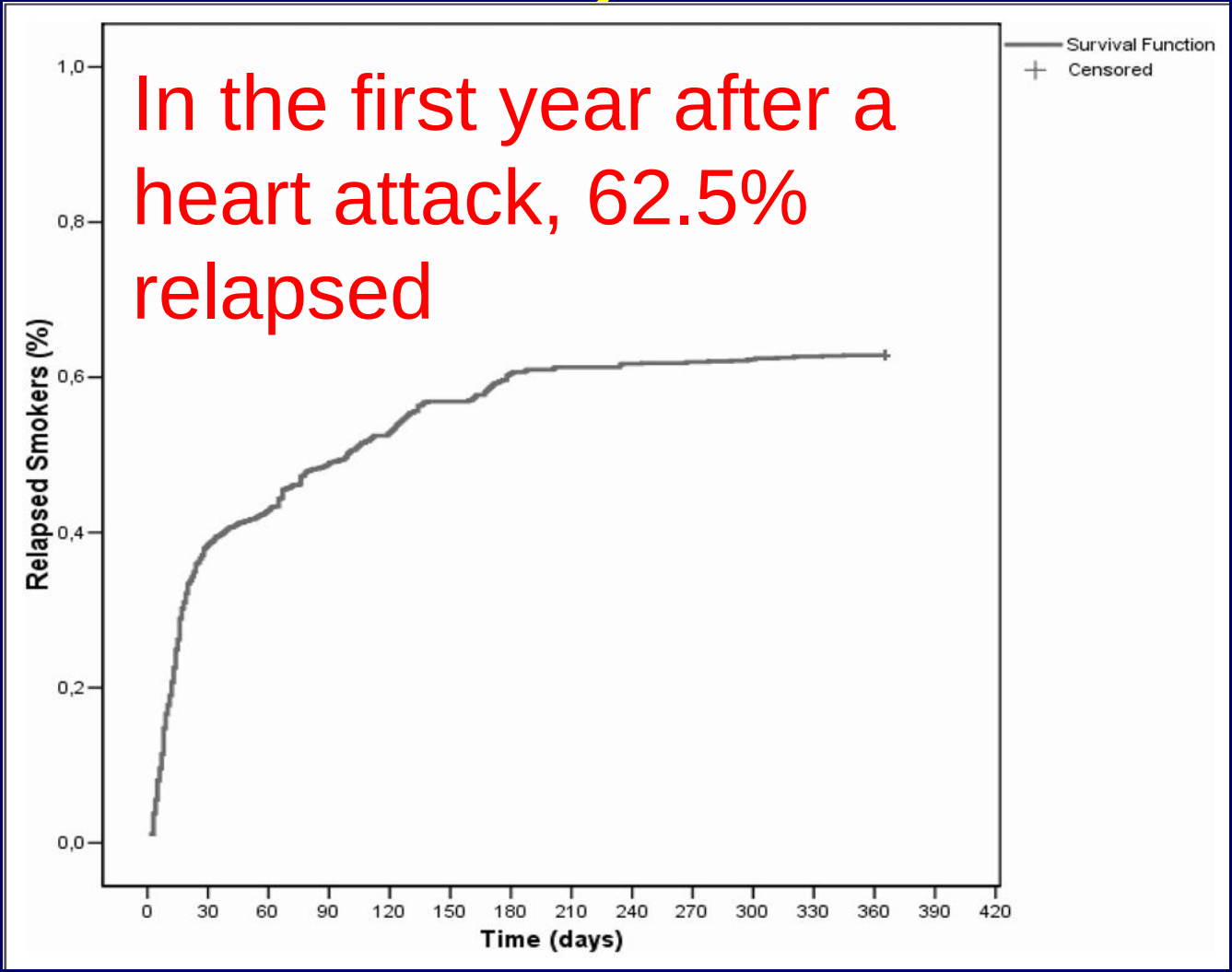
- TCHOL 5.0, LDL 3.0, TG 3.1, HDL 0.8
- Target is LDL <2.0mmol/L
- Double atorvastatin
- Started smoking as putting on weight
- Does that matter?

Failure to stay smoke free



Effect of Smoking Relapse on Outcome After Acute Coronary Syndromes Colivicchi et al Am J Cardiol 2011 in press

Failure to stay smoke free

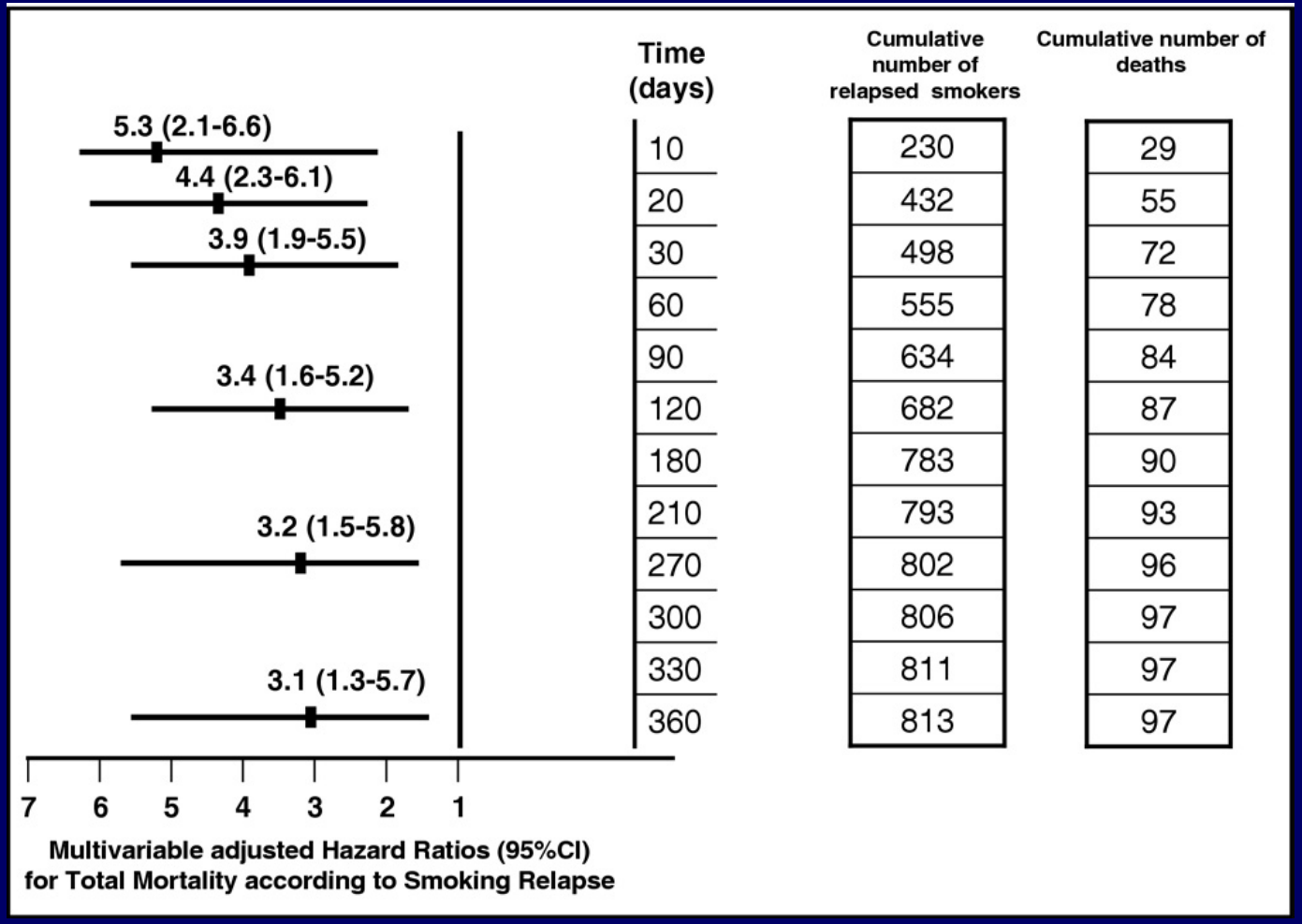


Figure 2. Multivariate adjusted HRs over time (horizontal bars represent 95% CIs) for association between smoking relapse and all-cause mortality Colivicchi et al Am J Cardiol 2011 in press

Rupert. Follow-up at 4 weeks

- Rehab class – cant attend because of work
- No pain. “Do I still needs these pills Doc?”
- Follow-up lipids
- TCHOL 5.0, LDL 3.0, TG 3.1, HDL 0.8
- Target is LDL <2.0mmol/L
- Double atorvastatin
- Started smoking as putting on weight
- Does that matter? Yes
- Still on cilazapril 1mg. Any comment?

Rupert. Follow-up at 4 weeks

- Rehab class – cant attend because of work
- No pain. “Do I still needs these pills Doc?”
- Follow-up lipids
- TCHOL 5.0, LDL 3.0, TG 3.1, HDL 0.8
- Target is LDL <2.0mmol/L
- Double atorvastatin
- Started smoking as putting on weight
- Does that matter? Yes
- Still on cilazapril 1mg.
- History and examination: he has orthopnoea and basal crackles.

Rupert. Follow-up at 4 weeks

- Rehab class – cant attend because of work
- No pain. “Do I still needs these pills Doc?”
- Follow-up lipids
- TCHOL 5.0, LDL 3.0, TG 3.1, HDL 0.8
- Target is LDL <2.0mmol/L
- Double atorvastatin
- Started smoking as putting on weight
- Does that matter? Yes
- Still on cilazapril 1mg.
- History and examination. Has orthopnoea, and basal crackles.
- Add Frusemide 40mg, increase Cilazapril to 2.5mg, check renal function and elecs next week

Rupert: Summary at 4 weeks

- Delayed presentation with anterior STEMI
- No recurrent angina but has evolved important heart failure despite acute stenting
- Impacts on employment
- Importance of prevention

Case 1:46M. Follow-up at 8 weeks

- Still short of breath
- Occasional bibasal crackle
- Na+ 133, K+ 3.7, Creat 0.10, Cr Clearance 80
- What next

Case 1:46M. Follow-up at 8 weeks

- Still short of breath
- Occasional bibasal crackle
- Na+ 133, K+ 3.7, Creat 0.10, Cr Clearance 80
- What next
- Spirometry OK
- BNP 400 (normal <40, borderline 40-80)

Case 1:46M. Follow-up at 8 weeks

- Still short of breath
- Occasional bibasal crackle
- Na+ 133, K+ 3.7, Creat 0.10, Cr Clearance 80
- What next
- BNP 400 (normal <40, borderline 40-80)
- Add spironolactone 25mg, consider increasing Frusemide

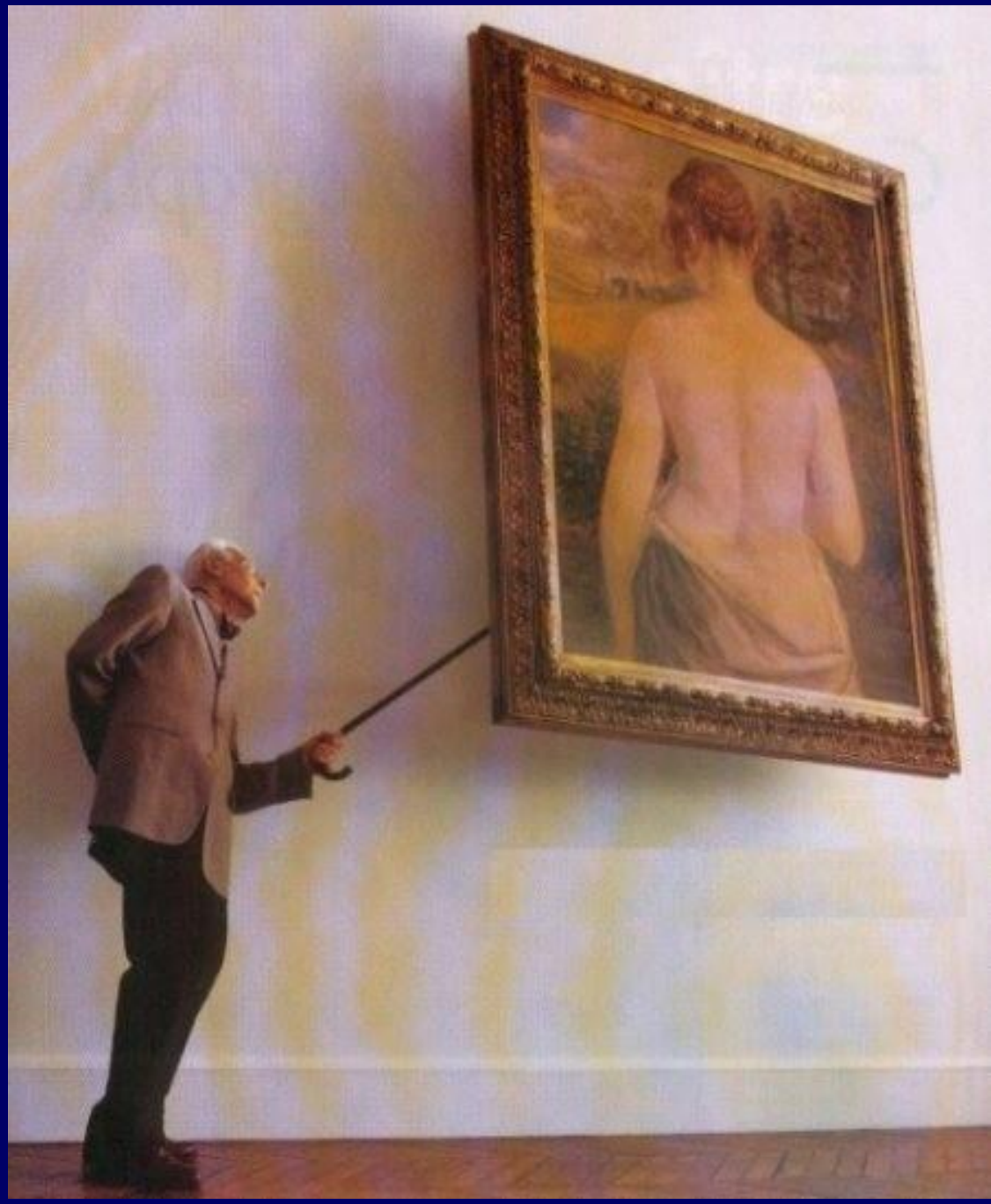
Case 1:46M. Follow-up at 8 weeks

- Still short of breath
- Occasional bibasal crackle
- Na+ 133, K+ 3.7, Creat 0.10, Cr Clearance 80
- What next
- BNP 400 (normal <40, borderline 40-80)
- Add spironolactone 25mg
- CARDIOLOGY REVIEW at 10 weeks:
- Repeat ECHO EF 30%, LV volumes normal. No change in pills

Life after a heart attack

- Prevention better than palliation
- Rehabilitation programs - group/home based
- Check angina status, advice re activities, work
- Body weight/dietary advice (the Green Prescription)
- Smoking cessation support
- Medications at discharge
  - Aspirin and Clopidogrel
  - Statin/diet Target LDL <2.0 mmol/L
  - Beta blocker
  - ACE inhibitor
  - Other meds for angina, heart failure, diabetes

Still much to learn

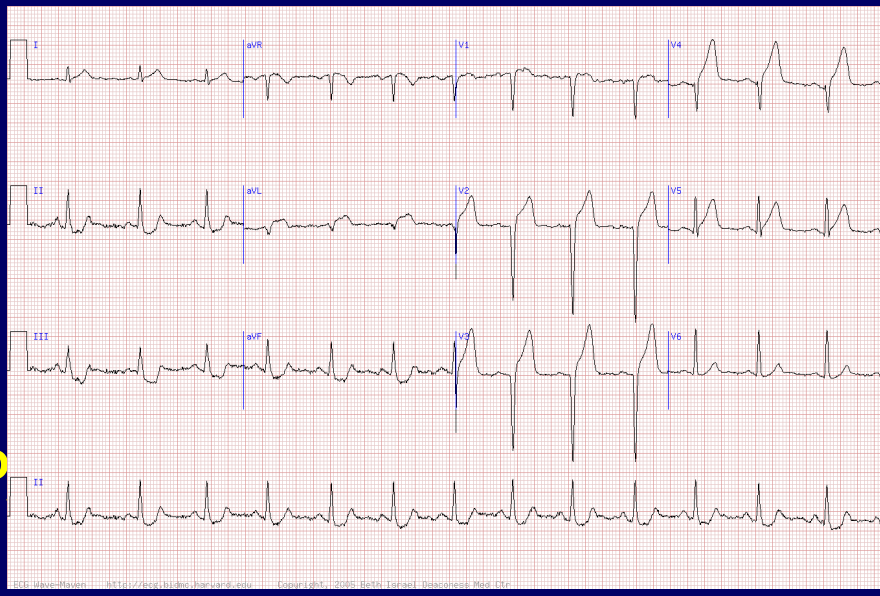


Revascularisation for ACS

- **Table 2. *Hazard Ratios and 95% Confidence Interval for***
- ***Mortality Comparing Patients With versus Patients Without In-Hospital Revascularisation.***
- **Unadjusted**
- **0.29 (0.20–0.42)**
- **Adjusted for baseline prognostic factors (a)**
- **0.37 (0.25–0.55)**
- **Adjusted for baseline prognostic factors and statins**
- **0.39 (0.27–0.58)**
- **Adjusted for baseline prognostic factors and all EBMs**
- **RR 0.38 (0.25–0.56)**
- **All EBMs included statins, beta-blockers, aspirin and ACE-inhibitors/A-II antagonists.**
- **(a) Baseline prognostic factors included age, history of ischaemic heart disease, clinical congestive heart failure, pulse on admission, initial serum creatinine and maximum troponin level; as previously found in this data set [1].**

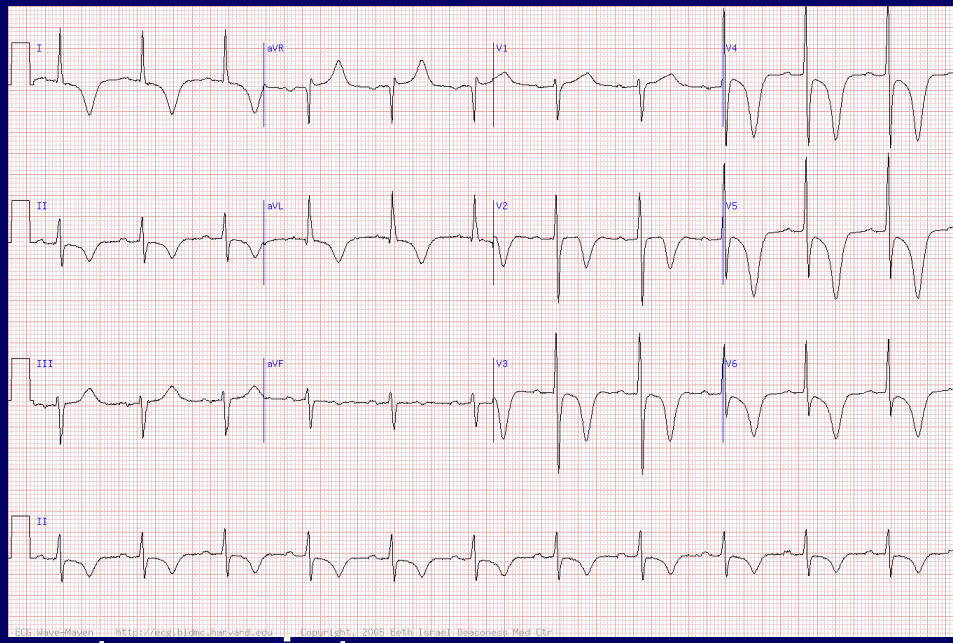
Wong et al Heart, Lung and Circulation 2009;18:262–265

Treatment – ST elevation



- Aspirin 150 to 300mg
- Clopidogrel
- Reperfusion therapy –
- Thrombolytic – SK, TPA, RP
- Or Direct PTCA/Stent (PCI)
- Adjunctive heparin, hirudin, IIbIIIa inhibitors
- Beta-blocker iv or oral
- IV nitrates and/or calcium antagonist
- ACE inhibitor
- Statin
- In-hospital angiogram if not done at admission

Treatment – no ST elevation



- Aspirin 150 to 300mg
- Clopidogrel 300 or 600mg
- Oxygen
- Heparin, ivUFH, sc LMWH
- Beta-blocker iv or oral
- IV nitrates and /or calcium antagonist
- ACE-inhibitor
- If persisting pain, and ECG changes or raised troponin, consider iv GP IIbIIIa antagonist such as tirofiban
- In-hospital angiography if NSTEMI

Treatment of AMI in Christchurch

Oct1 – Dec31, Cardiology	1997/98	1999/00	2001/02
N	371	371	334
Median age (yrs)	71	73	71
Women (%)	31	39	37
Median stay (d)	6	6	5
In-hosp angiogram (%)	20	34	60
In-hosp PCI (%)	11	26	43
In-hosp CABG (%)	2	5	5
In-hosp revasc (%)	13	31	48
Discharge meds -			
Beta-blocker (%)	71	85	84
Statin (%)	32	54	73
ACE-inhibitor	45	48	54

Mulholland et al CSANZ 2007

Routine vs Selective Invasive Strategies in Patients With Acute Coronary Syndromes

A Collaborative Meta-analysis of Randomized Trials

Shamir R. Mehta, MD, MSc
Christopher P. Cannon, MD
Keith A. A. Fox, MBCHB
Lars Wallentin, MD

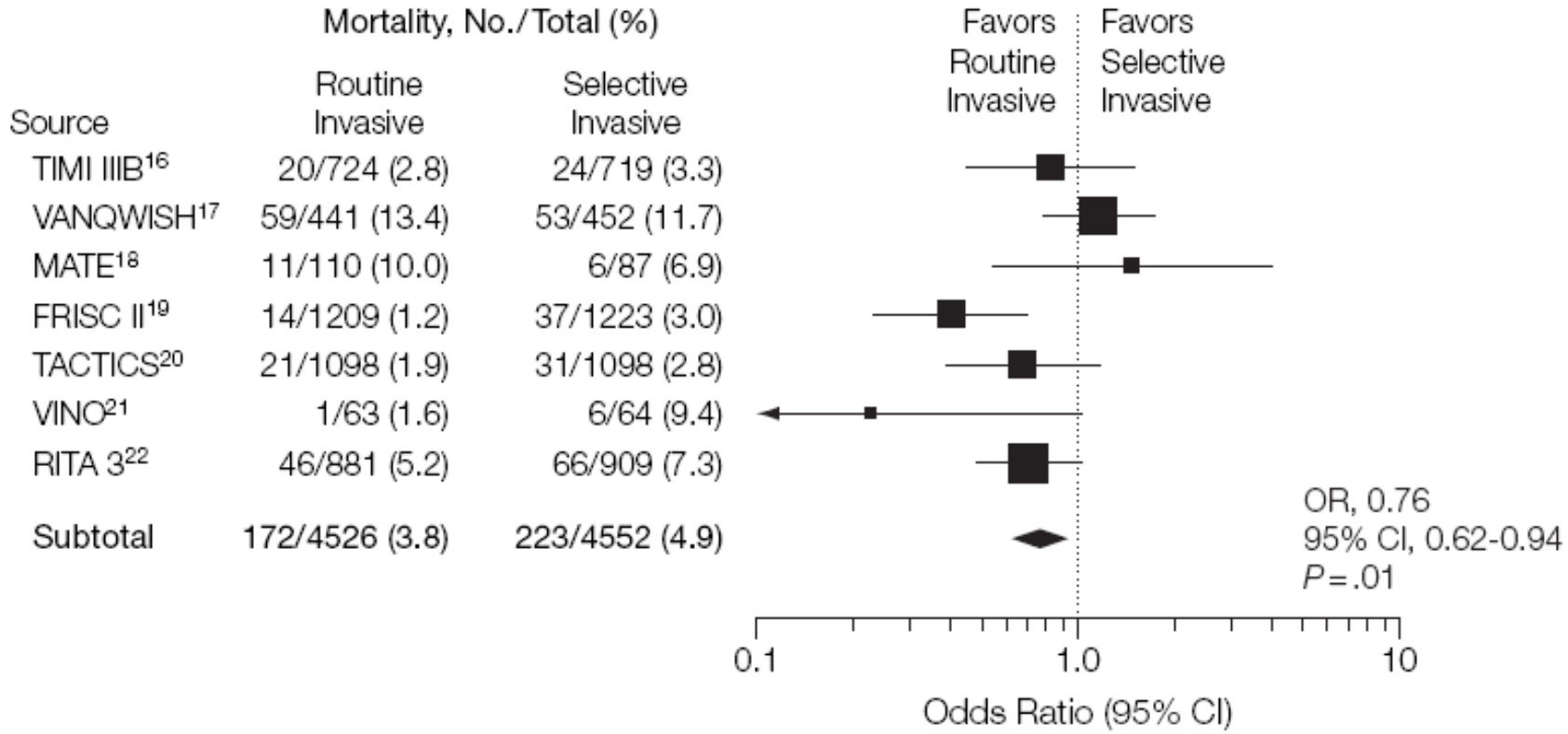
Context Patients with unstable angina or non–ST-segment elevation myocardial infarction (NSTEMI) can be cared for with a routine invasive strategy involving coronary angiography and revascularization or more conservatively with a selective invasive strategy in which only those with recurrent or inducible ischemia are referred for acute intervention.

In patients with unstable angina and NSTEMI, a routine early invasive strategy is superior to a selective invasive strategy in reducing major cardiovascular events as well as severe angina and rehospitalization. However, the main benefit of a routine invasive strategy was in preventing events over the longer term with evidence of early hazard.

JAMA. 2005;293:2908-2917

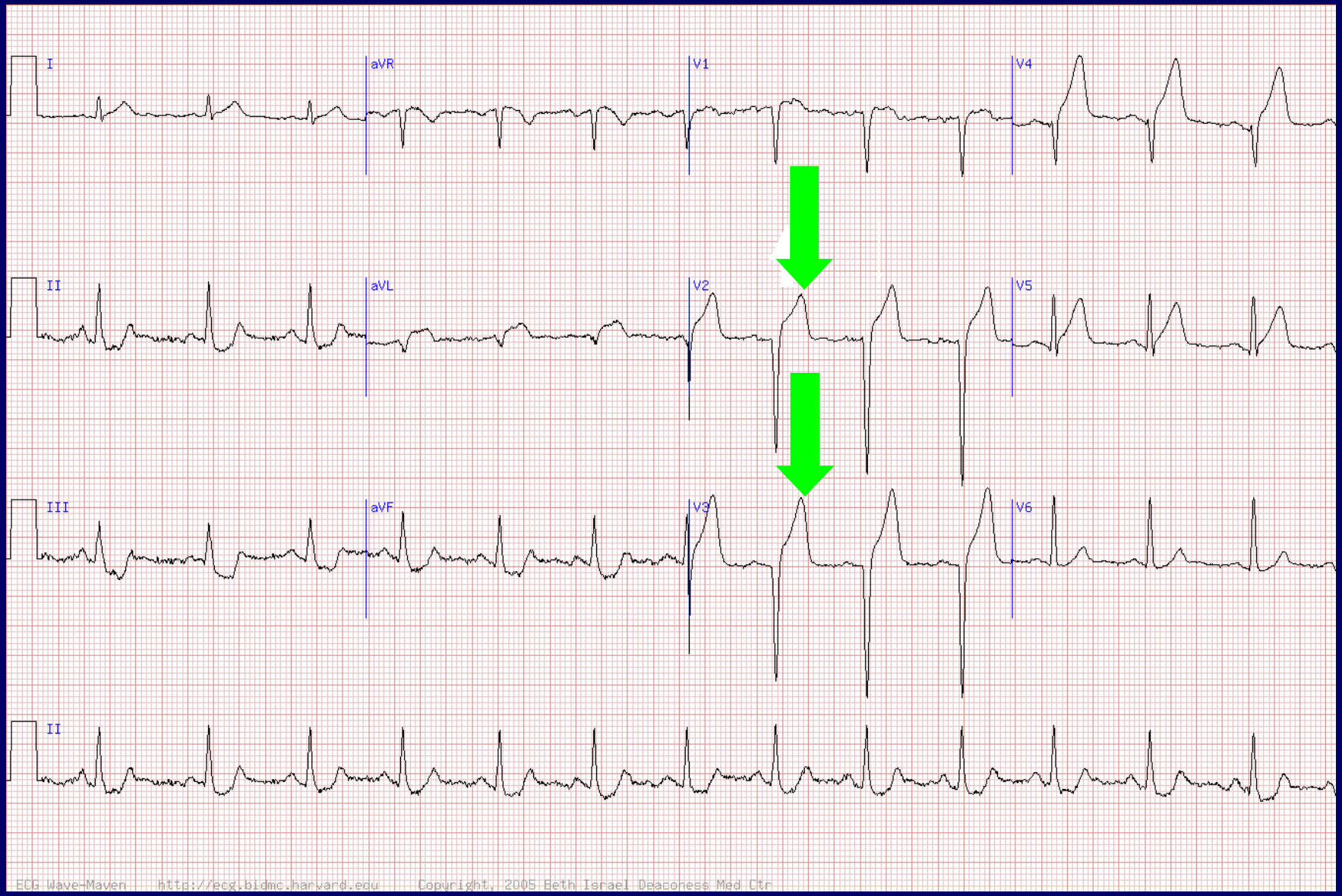
In-hospital revascularisation

Figure 2. Main Outcomes From Hospital Discharge to End of Follow-up in Trials of Routine vs Selective Invasive Management of Acute Coronary Syndromes

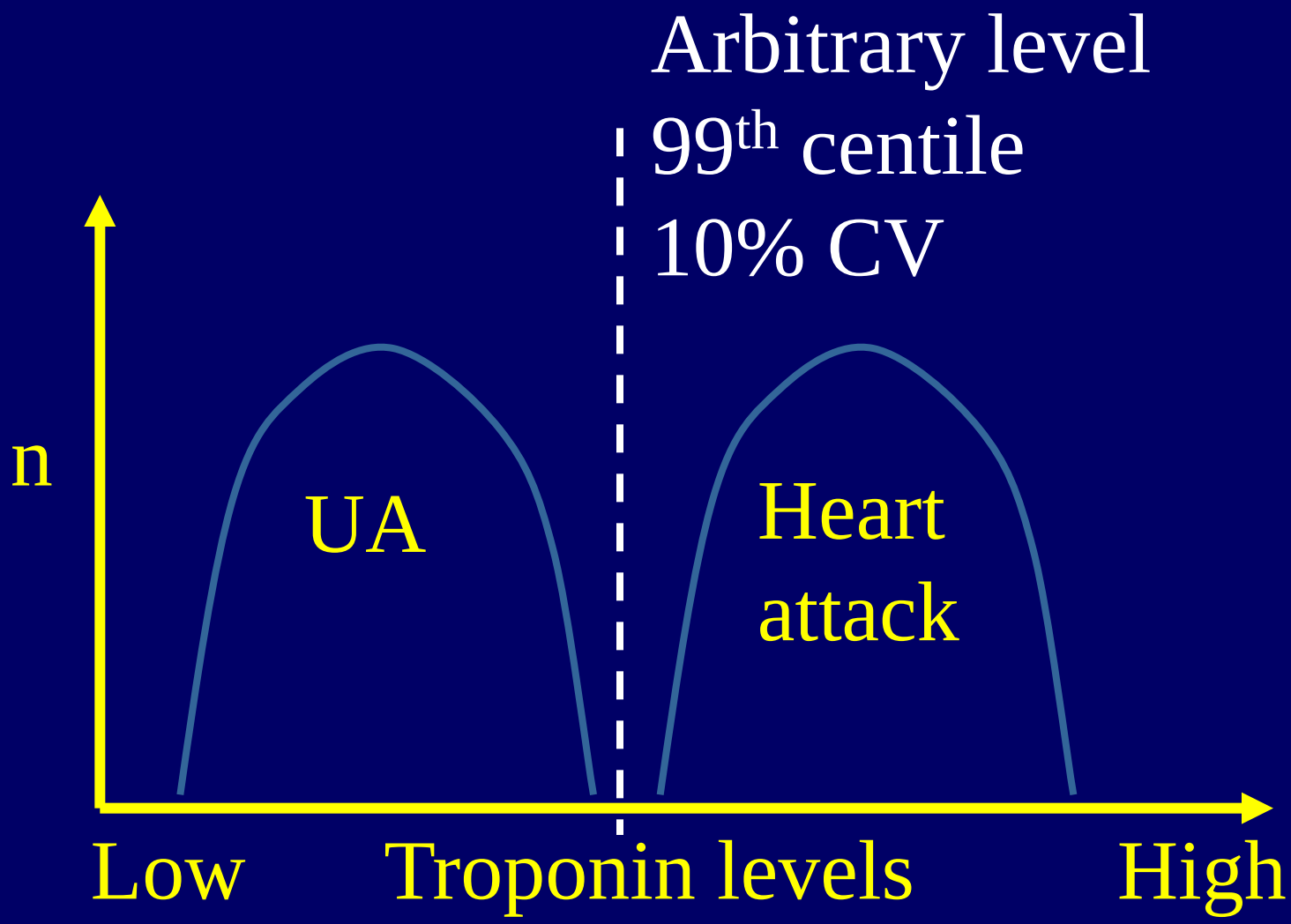


JAMA. 2005;293:2908-2917

Anterior ST elevation MI

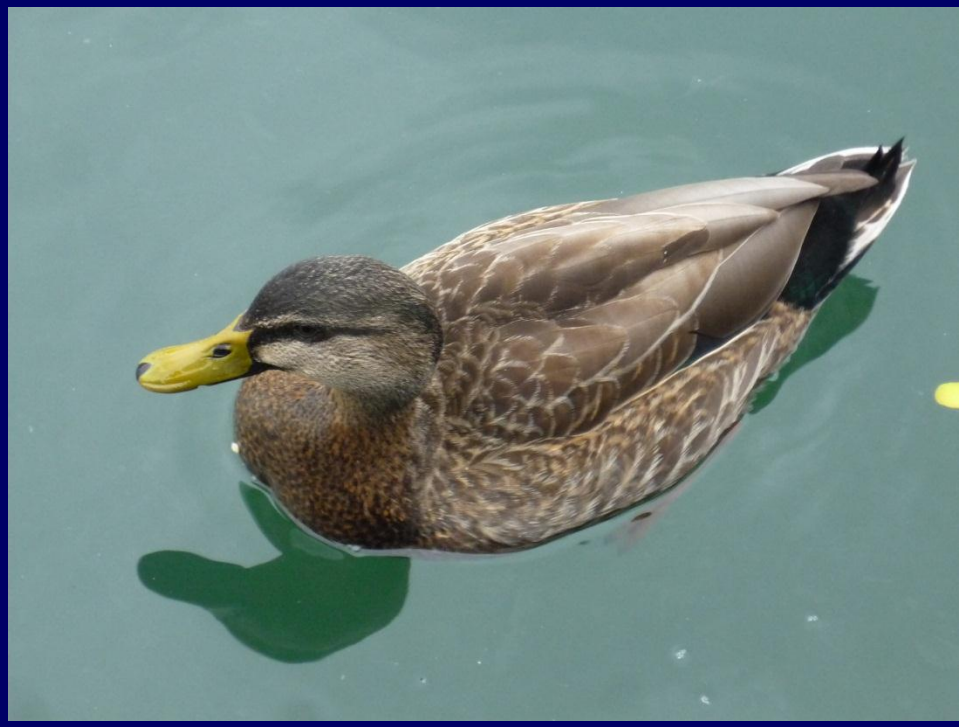


Diagnosis: The other dream



But is rise and fall in serial troponins always due to plaque rupture?

- If it looks like a duck
- And quacks like a duck
- And swims like a duck



- Then it might be a Type 1 duck due to plaque rupture or erosion

Rise and fall in serial troponins

- Or it could be a Type 2 duck

Type 2	**MI secondary to ischemia due to an imbalance of O_2 supply and demand, as from coronary spasm or embolism, anemia, arrhythmias, hypertension, or hypotension**

Rise and fall in serial troponins

- Or it might not even be duck

The term myocardial infarction does not include … myocardial necrosis due to miscellaneous causes eg renal failure, heart failure, cardioversion, EP ablation, sepsis, myocarditis, cardiac toxins, or infiltrative diseases Thygesen et al 2007