This Year in Review
Seminal Papers in Cardiology

Dr Raymond Massay

Cardiology 2015

- Clinical:
  - Novel risk factors increasingly recognized as important – stress, psychogenic, environmental, periodontal disease, pregnancy associated hypertension and diabetes
- Diagnostic:
  - High sensitivity Troponins I and T
- Medical Management:
  - New drugs for heart failure and cholesterol lowering

Cardiology 2015

- Surgical Management:
  - Left ventricular assist devices
- Intervention:
  - Radial approach
  - Bio-absorbable stents
  - TAVR
  - Mitral valve clips
  - Aortic stents
- Medical Imaging:
  - Increasing importance of cardiac MRI

Selected Topics

- Ticagrelor in acute NSTEMI
- Angiotensin – Neprilysin inhibition to treat heart failure
- Stress
  - Environmental
  - Psychogenic – Case presentation of Takotsubo cardiomyopathy
- Something Old
  - Statins and the skin

European Heart Journal
doi:10.1093/eurheartj/ehv320

ESC GUIDELINES

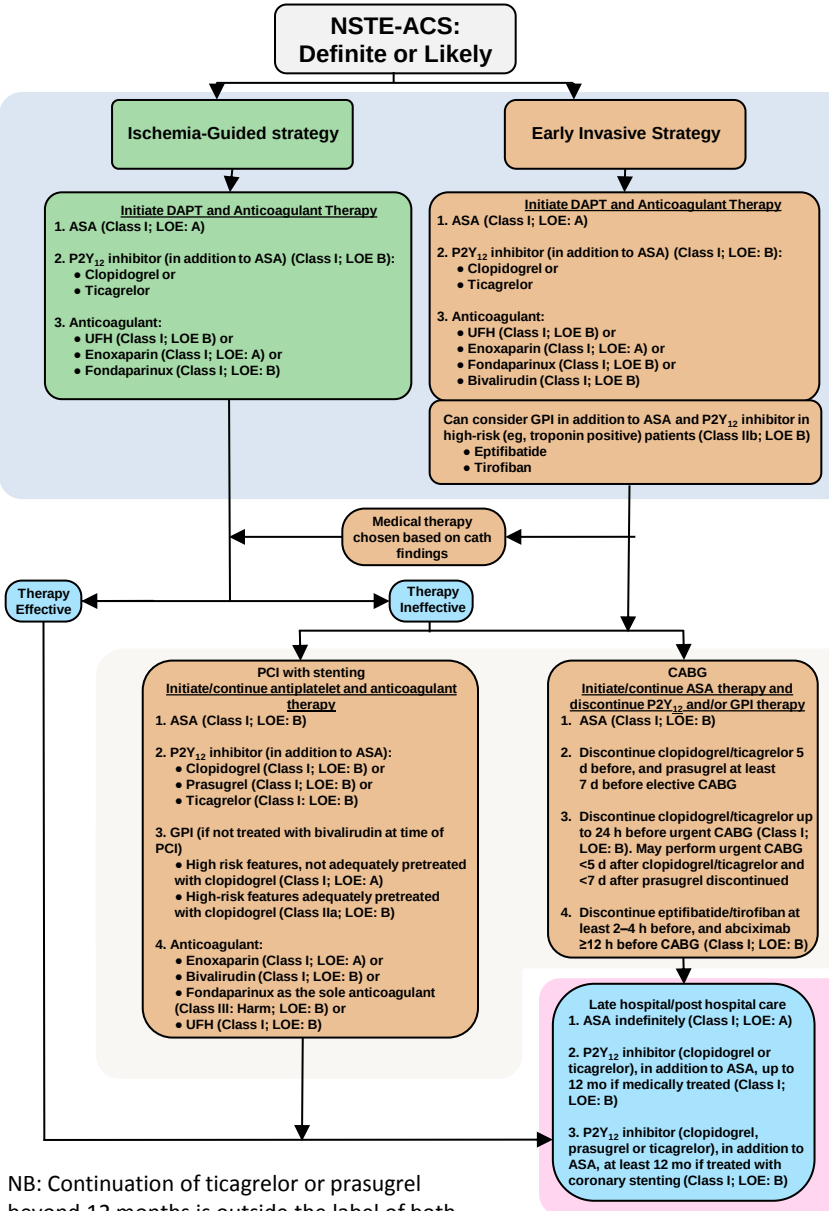
2015 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation

Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC)

Recommendations for platelet inhibition in non-ST-elevation acute coronary syndromes

Recommendations	Class[a]	Level[b]	Ref.[c]
Oral antiplatelet therapy			
Aspirin is recommended for all patients without contraindications at an initial oral loading dose[d] of 150–300 mg (in aspirin-naive patients) and a maintenance dose of 75–100 mg/day long-term regardless of treatment strategy.	I	A	129–132
A $P2Y_{12}$ inhibitor is recommended, in addition to aspirin, for 12 months unless there are contraindications such as excessive risk of bleeds.	I	A	137, 148, 153
• Ticagrelor (180 mg loading dose, 90 mg twice daily) is recommended, in the absence of contraindications,[e] for all patients at moderate-to-high risk of ischaemic events (e.g. elevated cardiac troponins), regardless of initial treatment strategy and including those pretreated with clopidogrel (which should be discontinued when ticagrelor is started).	I	B	153
• Prasugrel (60 mg loading dose, 10 mg daily dose) is recommended in patients who are proceeding to PCI if no contraindication.[e]	I	B	148, 164
• Clopidogrel (300–600 mg loading dose, 75 mg daily dose) is recommended for patients who cannot receive ticagrelor or prasugrel or who require oral anticoagulation.	I	B	137
$P2Y_{12}$ inhibitor administration for a shorter duration of 3–6 months after DES implantation may be considered in patients deemed at high bleeding risk.	IIb	A	187–189, 192

2014 AHA/ACC guidelines for the management of patients with NSTE-ACS Recommended treatment algorithm



Pathway stage	$P2Y_{12}$ recommendation
• Early management (**angiography or initial medical management**)	• **Initiate ticagrelor** or clopidogrel, **preferably ticagrelor** • (NB: prasugrel is not a recommended option at this stage)
• **Invasive management** (PCI)	• **Initiate/continue ticagrelor** or clopidogrel, or initiate prasugrel (only after coronary anatomy has been defined)
• **Late/post-hospital care**	• **Medically managed: Ticagrelor** or clopidogrel for ***up to* 12 months**, preferably ticagrelor • **Stent: Ticagrelor,** prasugrel or clopidogrel **for *at least* 12 months**, preferably ticagrelor or prasugrel

NB: Continuation of ticagrelor or prasugrel beyond 12 months is outside the label of both drugs

Top-line recommendations in NSTE-ACS

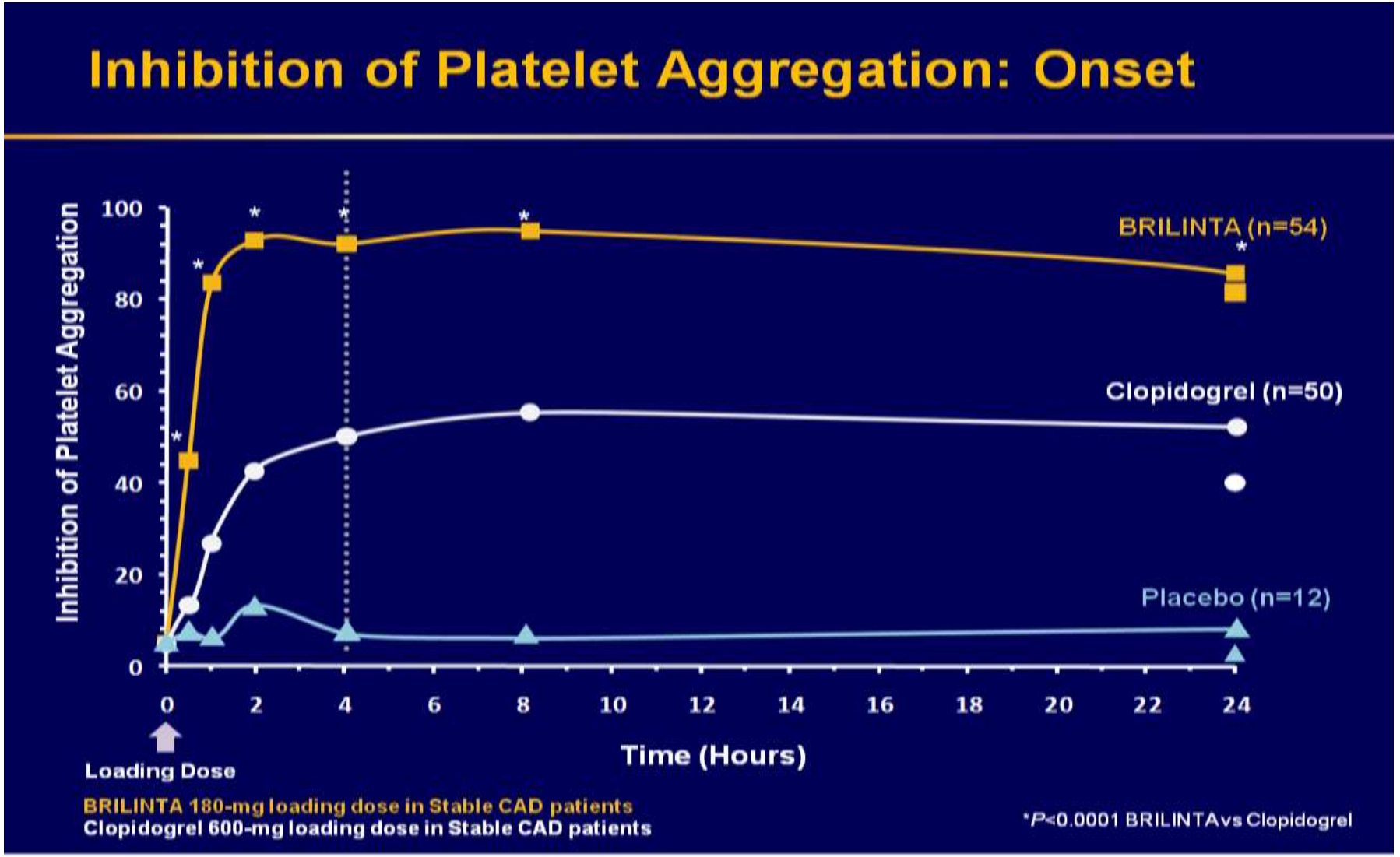
Early management strategy (initial ischaemia-guided or early invasive strategy) before definition of coronary anatomy

Recommendation	Class	Level	Evidence
$P2Y_{12}$ inhibitor (**either clopidogrel or ticagrelor**) in addition to aspirin, for up to 12 months in patients treated initially with either an early invasive or ischaemia-guided strategy	I	B	CURE[1], CURRENT-OASIS 7[2], PLATO[3], PLATO non-invasive substudy[4]
It is reasonable to choose **ticagrelor in preference to clopidogrel** for patients treated with an early invasive or ischaemia-guided strategy	IIa	B	PLATO[3], PLATO non-invasive substudy[4]

Contraindications and other label requirements still apply

1. Yusuf S et al. N Engl J Med 2001;345:494–502
2. Mehta SR et al. N Engl J Med 2010;363:930–942
3. Wallentin L et al. N Engl J Med 2009;361:1045–1057
4. James SK et al. BMJ 2011;342:d3527

Onset, and level of IPA, Ticagrelor vs Clopidogrel



Important Comparators

	TICAGRELOR	CLOPIDOGREL
Onset of action	Fast (orally active)	Slow (pro drug)
Therapeutic Path	No drug-drug interactions	Drug-drug interactions
Level of inhibition	Predictably high (no enzyme system involved)	Variable, based on individual Genotype
Individual Response	All responders	Some non-responders
PPI co-therapy?	Yes (no interaction)	Some interaction

PLATO Trial (18,624 patients)

Endpoint	Ticagrelor group n (%)	Clopidogrel group n (%)	Hazard Ratio for TICAGRELOR group (95% CI)	P Value†
Death from vascular causes, MI, or stroke	**864/9333 (9.8)**	**1014/9291 (11.7)**	0.84 (0.77–0.92)	<0.001‡
Death from any cause, MI, or stroke	**901/9333 (10.2)**	**1065/9291 (12.3)**	0.84 (0.77–0.92)	<0.001‡
MI	**504/9333 (5.8)**	**593/9291 (6.9)**	0.84 (0.75–0.95)	0.005‡
Death from vascular causes	**353/9333 (4.0)**	**442/9291 (5.1)**	0.79 (0.69–0.91)	0.001‡
Stroke	**125/9333 (1.5)**	**106/9291 (1.3)**	1.17 (0.91–1.52)	0.22
Ischemic	**96/9333 (1.1)**	**91/9291 (1.1)**	--	0.74
Hemorrhagic	**23/9333 (0.2)**	**13/9291 (0.1)**	--	0.10
Unknown	**10/9333 (0.1)**	**2/9291 (0.02)**	--	0.04

Learning Objectives

Upon completion of this activity, participants should be able to:

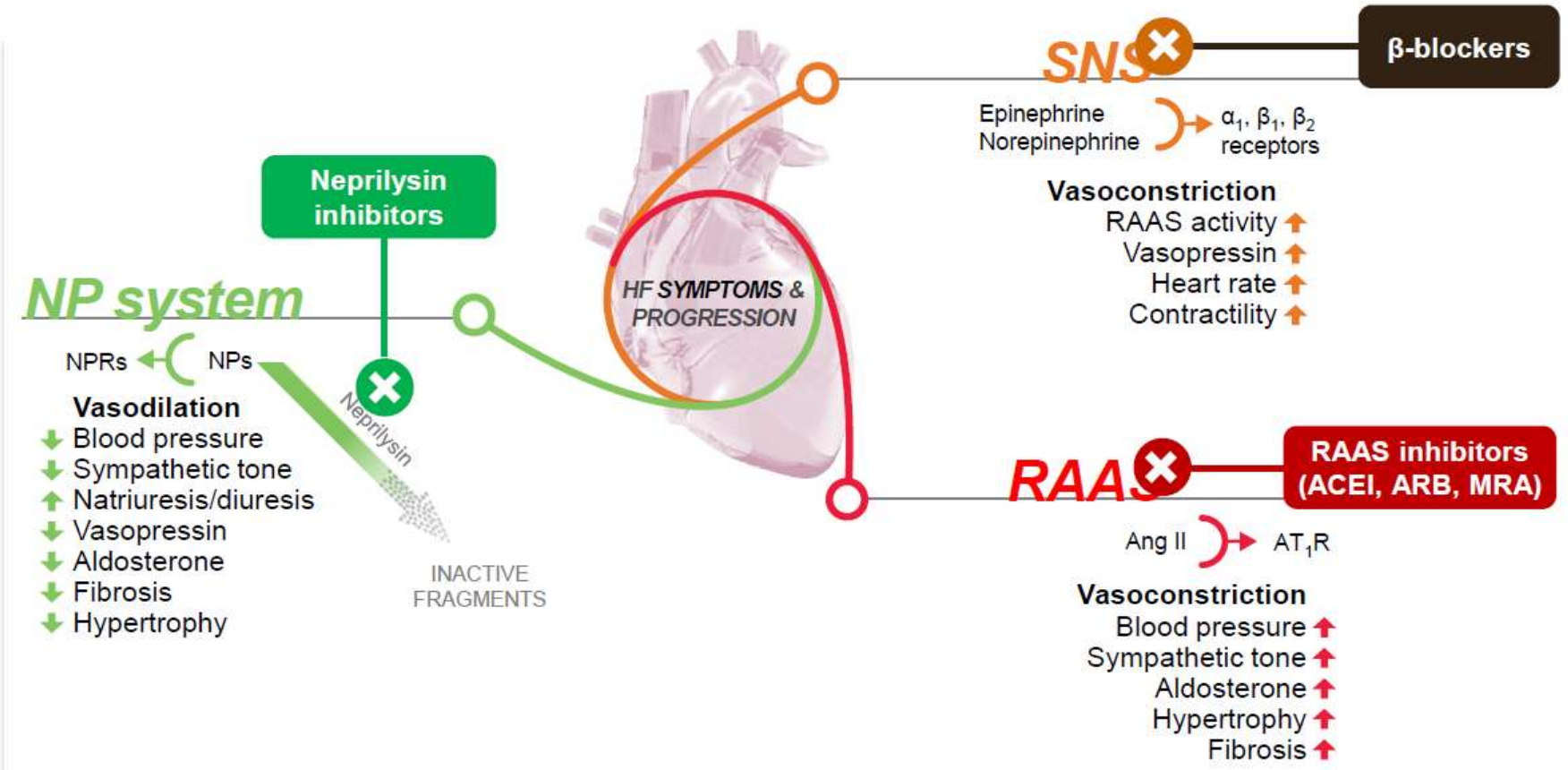
- Recognize factors contributing to clinical inertia in HF as well as limitations in systems and structure for management of HF
- Debate the need for a concerted multidisciplinary effort to overhaul HF management
- Describe the role of the renin angiotensin system, neprilysin inhibition, and natriuretic peptides in HF pathophysiology
- Analyze emerging scientific evidence for the next generation of dual-function therapies in HF management

Program Agenda

18:15	**Registration (Link)**
18:30	**Introduction and welcome** Mariell L. Jessup, MD
18:35	**A call to action in heart failure: what have we achieved and where are we going?** Piotr Ponikowski, MD, PhD
18:45	**Dual action molecules in heart failure: from bench to bedside** John C. Burnett, Jr., MD
19:00	**A case of heart failure management today: can we do better for our patients?** John J. V. McMurray, MD
19:20	**Q&A session** Mariell L. Jessup, MD

NOVARTIS
PHARMACEUTICALS

Evolution of pharmacologic approaches in HF: *Neprilysin inhibition as a new therapeutic strategy*[1]



- Neprilysin inhibitors: natriuretic and other vasoactive peptides enhancement

ACEI=angiotensin-converting enzyme inhibitor; Ang=angiotensin; ARB=angiotensin receptor blocker; AT1R = angiotensin II type 1 receptor; HF=heart failure; MRA=mineralocorticoid receptor antagonist; NP=natriuretic peptide; NPRs=natriuretic peptide receptors; RAAS=renin-angiotensin-aldosterone system; SNS=sympathetic nervous system

1. McMurray et al. Eur J Heart Fail. 2013;15:1062–73;
Figure references: Levin et al. N Engl J Med 1998;339:321–8; Nathisuwan & Talbert Pharmacotherapy 2002;22:27–42; Kemp & Conte. Cardiovascular Pathology 2012;365–71; Schrier & Abraham N Engl J Med 2009;341:577–85

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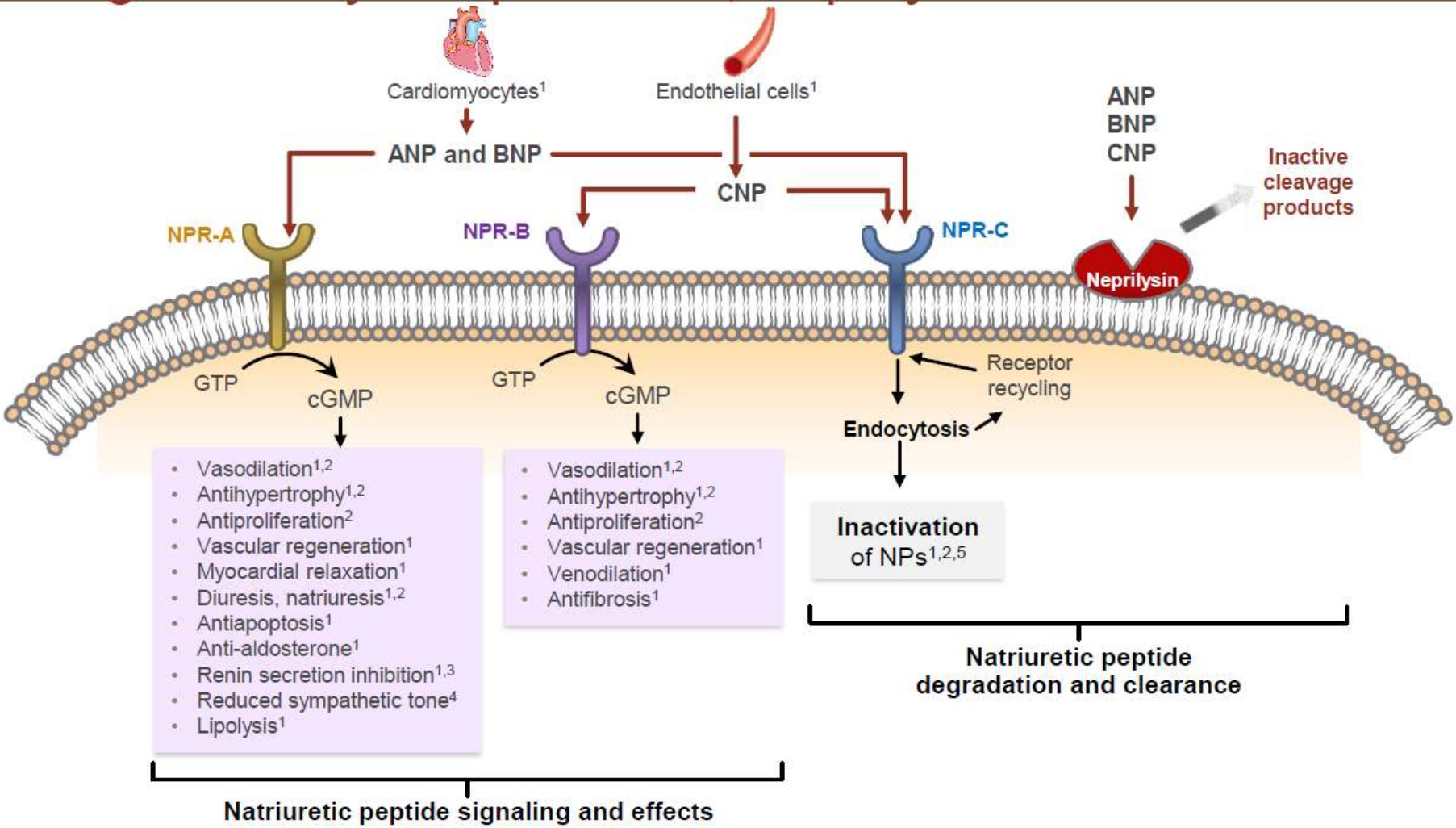
Natriuretic peptides are cleared via NPR-C and degraded by the protease, neprilysin



ANP=atrial natriuretic peptide; BNP=B-type natriuretic peptide; cGMP=cyclic guanosine monophosphate; CNP=C-type natriuretic peptide; GTP=guanosine triphosphate; NP=natriuretic peptide; NPR=natriuretic peptide receptor

1. Mangiafico et al. Eur Heart J 2013;34:886–93; 2. Gardner et al. Hypertension 2007;49:419–26; 3. Pandey. J Am Soc Hypertens 2008;2:210–26; 4. Levin et al. N Engl J Med 1998;339;321–8; 5. Von Lueder et al. Pharmacol Ther 2014 [Epub ahead of print]

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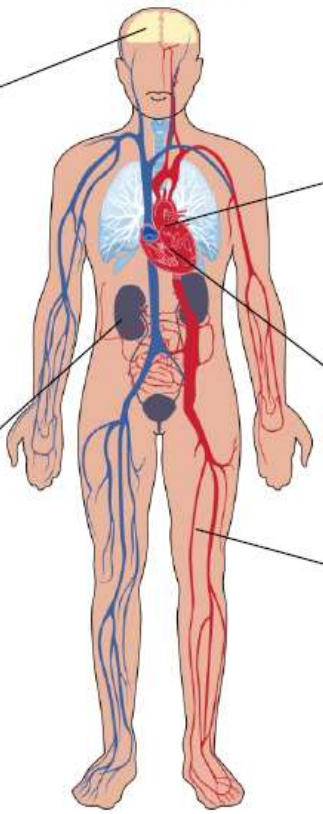
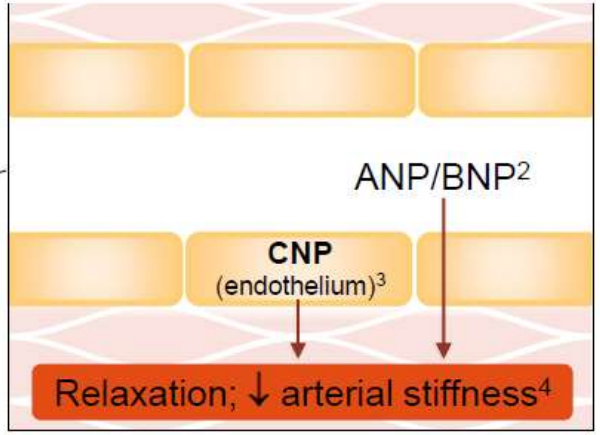
Natriuretic peptides have potential beneficial actions in HF

Release of ANP and BNP from heart and CNP in vasculature[1,2]

↓ Sympathetic outflow[2]
↓ Vasopressin[2]
↓ Salt appetite and water intake[2]





↓ Hypertrophy[2,5–7]
↓ Fibroblast proliferation[4,8,9]

↑ Na^+/H_2O loss[2]
↓ Aldosterone[2]
↓ Renin[2]

Vasodilation[2,3,4]
↓ Systemic vascular resistance[4]
↓ Pulmonary artery pressure[4]
↓ Pulmonary capillary wedge pressure[4]
↓ Right atrial pressure[4]

1. Mangiafico et al. Eur Heart J 2013;34:886–93; 2. Levin et al. N Engl J Med 1998;339:321–8; 3. Lumsden et al. Curr Pharm Des 2010;16:4080–8; 4. Langenickel & Dole. Drug Discovery Today: Ther Strateg 2012;9:e131–9; 5. Gardner et al. Hypertension 2007;49:419–26; 6. Tokudome et al. Circulation 2008;117:2329–39; 7. Horio et al. Hypertension 2000;35:19–24; 8. D'Souza et al. Pharmacol Ther 2004;101:113–29; 9. Cao & Gardner Hypertension 1995;25:227–34

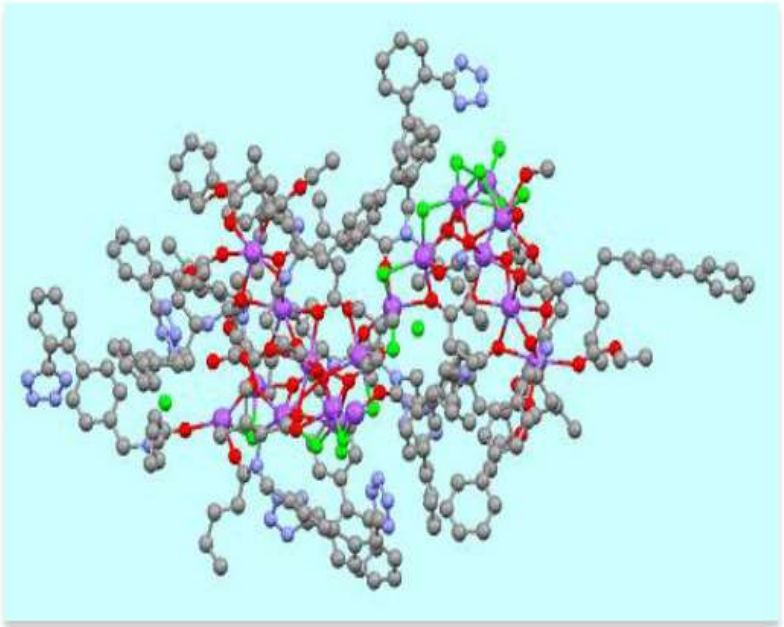
ANP=atrial natriuretic peptide;
BNP=B-type natriuretic peptide;
CNP=C-type natriuretic peptide; HF=heart failure

NOVARTIS

LCZ696 is a first-in-class angiotensin receptor neprilysin inhibitor (ARNI)

- LCZ696 is a novel drug which delivers simultaneous neprilysin inhibition and AT_1 receptor blockade[1–3]
- LCZ696 is a salt complex that comprises the two active components:[2,3]
  - sacubitril (AHU377) – a pro-drug; further metabolized to the neprilysin inhibitor LBQ657, and
  - valsartan – an AT_1 receptor blocker

  in a 1:1 molar ratio



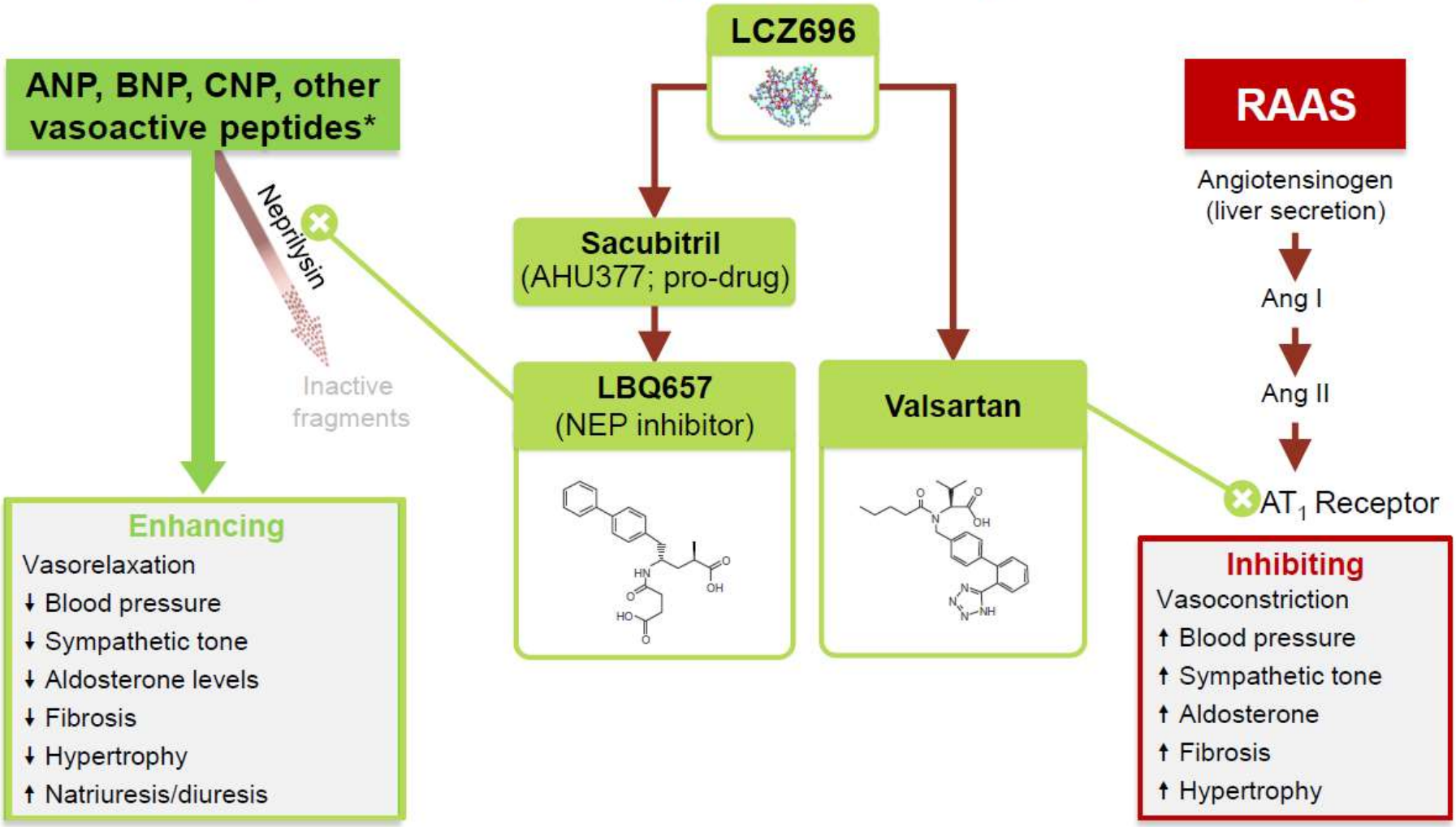
3D LCZ696 structure[2]

ARNI=angiotensin receptor neprilysin inhibitor; AT1=angiotensin II type 1

1. Bloch & Basile. J Clin Hypertens 2010;12:809–12;
2. Gu et al. J Clin Pharmacol 2010;50:401–14;
3. Langenickel & Dole. Drug Discov Today: Ther Strateg 2012;9:e131–5

NOVARTIS

LCZ696 simultaneously inhibits neprilysin (via LBQ657) and blocks AT_1 receptors (via valsartan)



*Neprilysin substrates listed in order of relative affinity for neprilysin: ANP, CNP, Ang II, Ang I, adrenomedullin, substance P, bradykinin, endothelin-1, BNP

Ang=angiotensin; ANP=atrial natriuretic peptide; AT1=angiotensin II type 1; BNP=B-type natriuretic peptide; CNP=C-type natriuretic peptide; NEP=neprilysin; RAAS=renin-angiotensin-aldosterone system

Levin et al. N Engl J Med 1998;339:321–8;
Nathisuwan & Talbert. Pharmacotherapy 2002;22:27–42;
Schrier & Abraham N Engl J Med 2009;341:577–85;
Langenickel & Dole. Drug Discov Today: Ther Strateg 2012;9:e131–9;
Feng et al. Tetrahedron Letters 2012;53:275–8

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The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 11, 2014 VOL. 371 NO. 11

Angiotensin–Neprilysin Inhibition versus Enalapril in Heart Failure

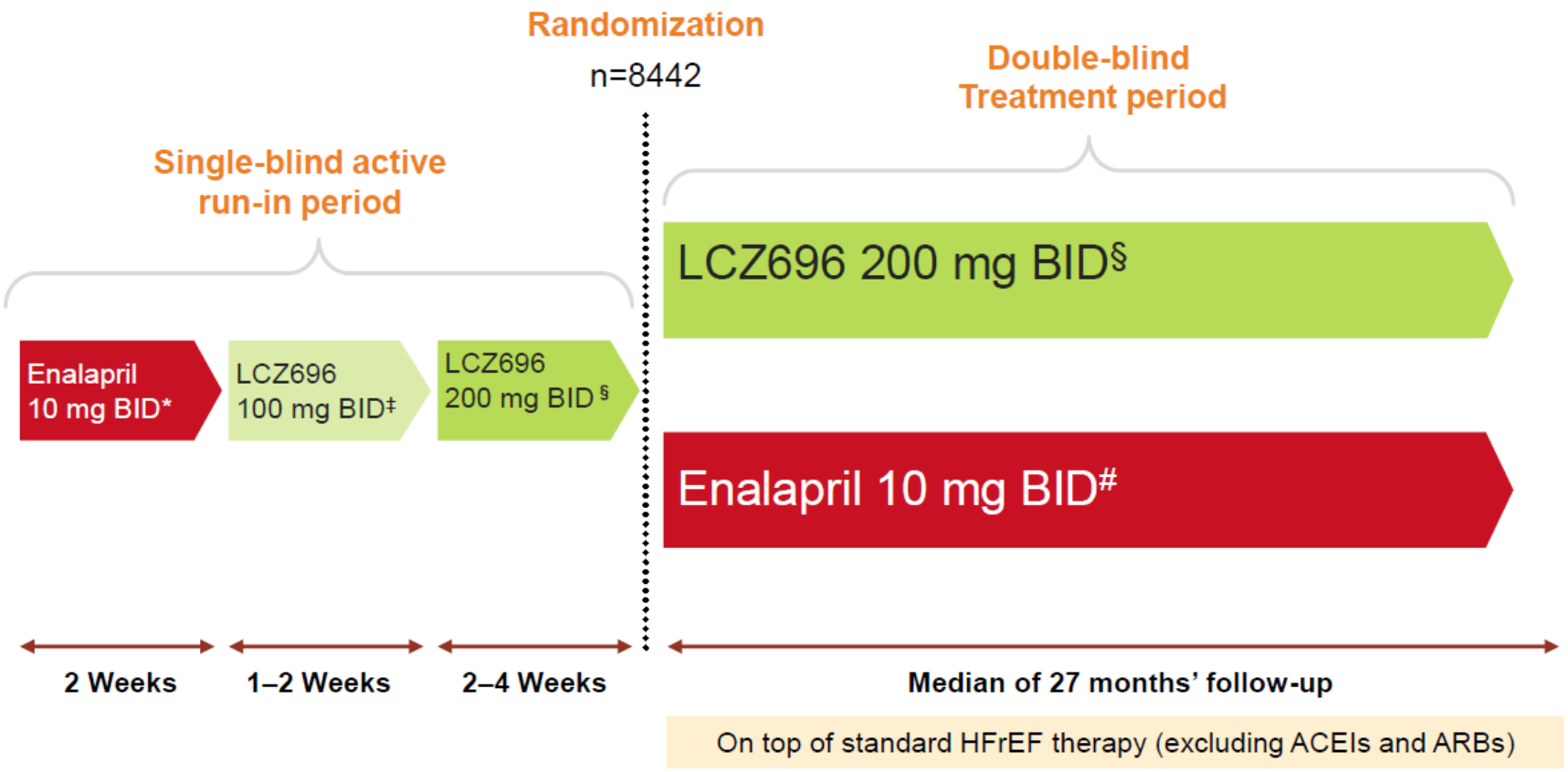
John J.V. McMurray, M.D., Milton Packer, M.D., Akshay S. Desai, M.D., M.P.H., Jianjian Gong, Ph.D., Martin P. Lefkowitz, M.D., Adel R. Rizkala, Pharm.D., Jean L. Rouleau, M.D., Victor C. Shi, M.D., Scott D. Solomon, M.D., Karl Swedberg, M.D., Ph.D., and Michael R. Zile, M.D., for the PARADIGM-HF Investigators and Committees*

PARADIGM-HF Study

Prospective comparison of ARNI with ACEI to Determine Impact on Global Mortality and morbidity in Heart Failure

A multicenter, randomized, double-blind, parallel-group, active-controlled study to evaluate the efficacy and safety of LCZ696 compared with enalapril on morbidity and mortality in patients with chronic HF and reduced ejection fraction

PARADIGM-HF: study design

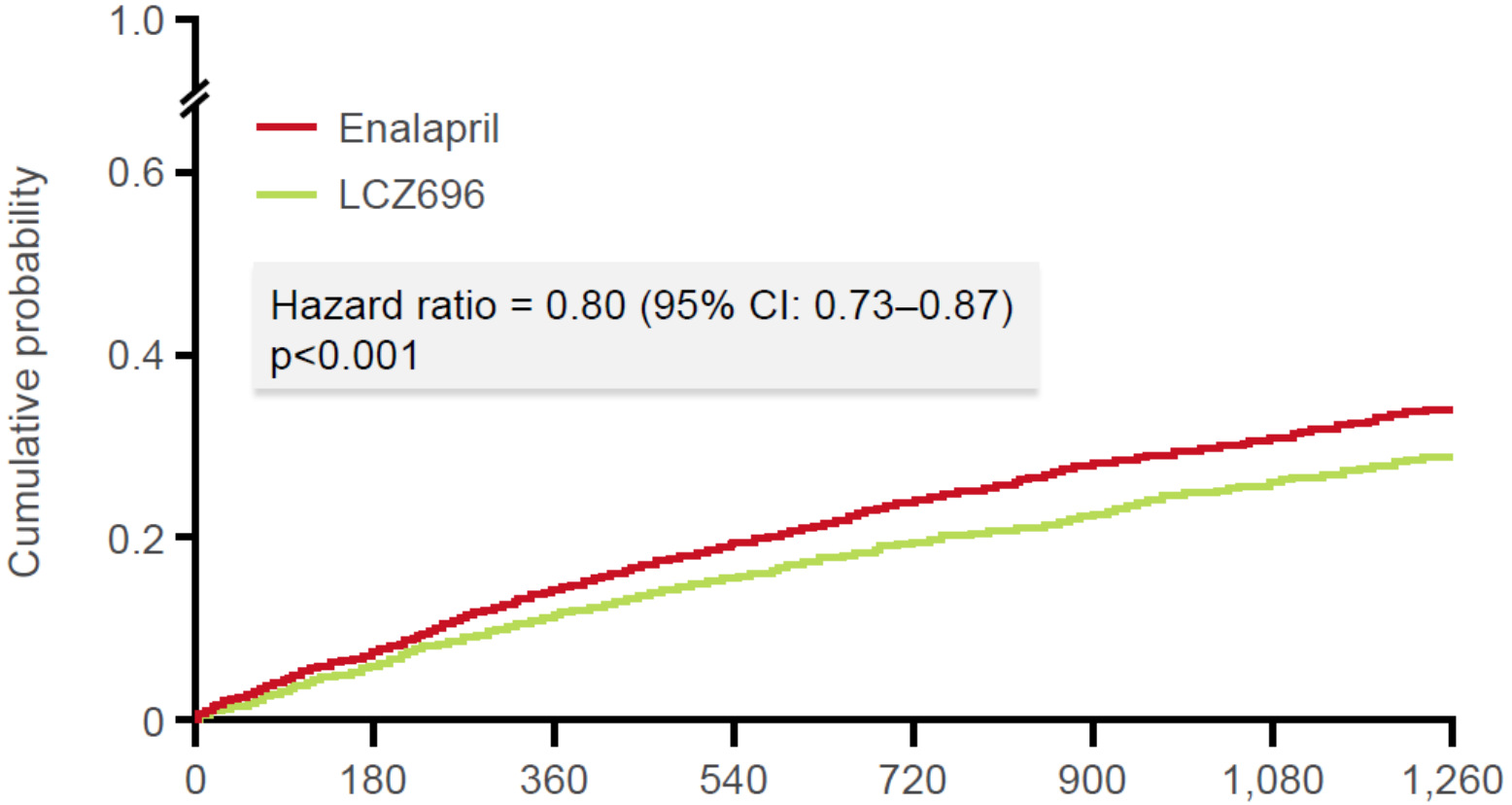


*Enalapril 5 mg BID (10 mg TDD) for 1–2 weeks followed by enalapril 10 mg BID (20 mg TDD) as an optional starting run-in dose for those patients who are treated with ARBs or with a low dose of ACEI; ‡200 mg TDD; §400 mg TDD; #20 mg TDD. ACEI=angiotensin-converting enzyme inhibitor; ARB=angiotensin receptor blocker; BID=twice daily; HFrEF=heart failure with reduced ejection fraction; PARADIGM-HF=Prospective comparison of ARNI with ACEI to Determine Impact on Global Mortality and morbidity in Heart Failure; TDD=total daily dose

McMurray et al. Eur J Heart Fail 2013;15:1062–73
McMurray et al. Eur J Heart Fail 2014;16:817–25
McMurray et al. N Engl J Med 2014;371:993–1004

Primary endpoint:

Death from CV causes or first hospitalization for HF

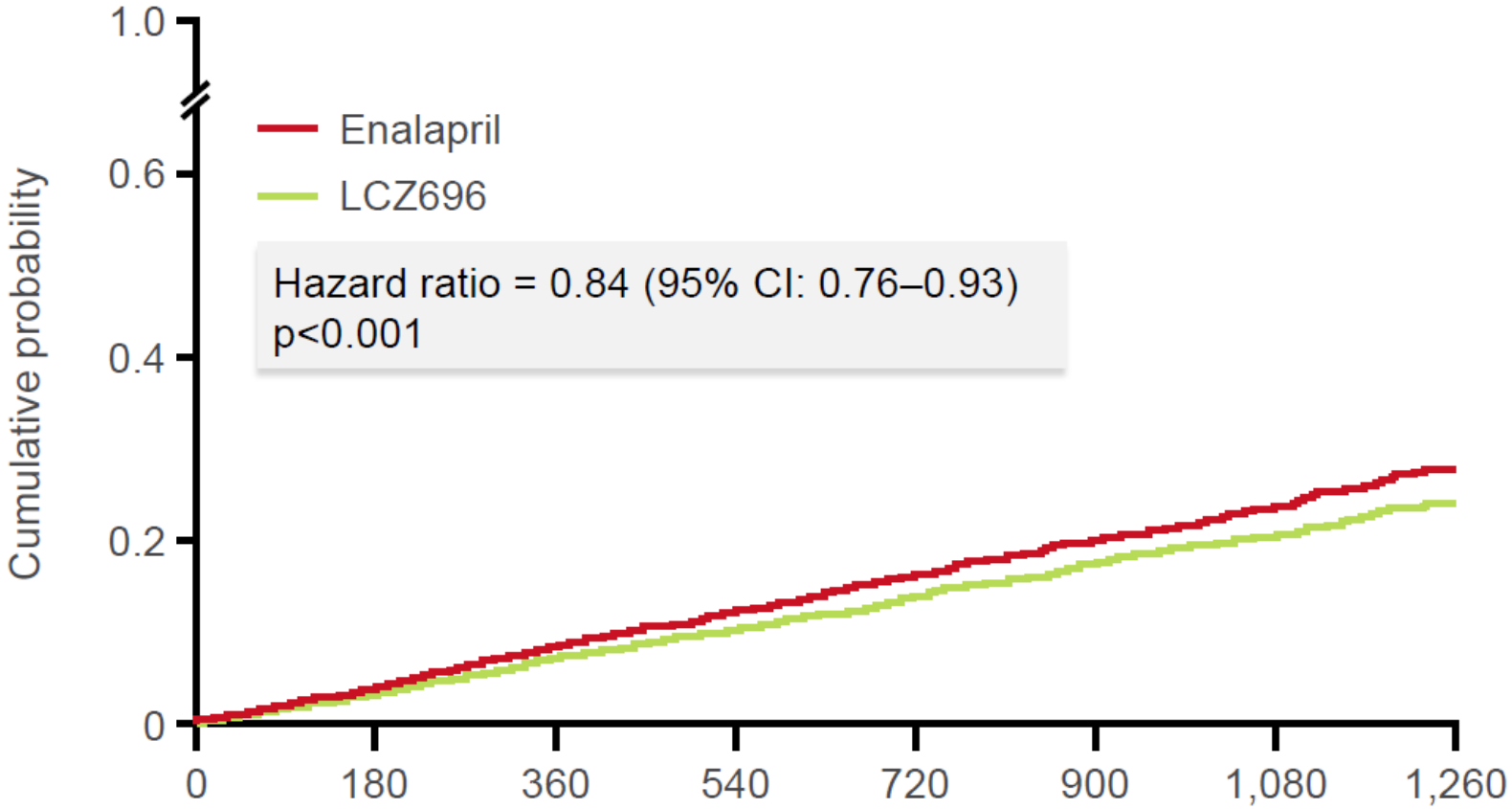


No at risk								
LCZ696	4,187	3,922	3,663	3,018	2,257	1,544	896	249
Enalapril	4,212	3,883	3,579	2,922	2,123	1,488	853	236

CI=confidence interval; CV=cardiovascular; HF=heart failure

McMurray et al. N Engl J Med 2014;371:993–1004

Death from any cause



No at risk								
LCZ696	4,187	4,056	3,891	3,282	2,478	1,716	1,005	280
Enalapril	4,212	4,051	3,860	3,231	2,410	1,726	994	279

CI=confidence interval

McMurray et al. N Engl J Med 2014;371:993–1004

Primary outcome

Outcome, n %	LCZ696 (n=4,187)	Enalapril (n=4,212)	Hazard ratio* (95% CI)	p value‡
Primary composite outcome				
Death from CV causes or first hospitalization for worsening of HF	914 (21.8)	1,117 (26.5)	0.80 (0.73–0.87)	<0.001
Death from CV causes	558 (13.3)	693 (16.5)	0.80 (0.71–0.89)	<0.001
First hospitalization for worsening of HF	537 (12.8)	658 (15.6)	0.79 (0.71–0.89)	<0.001

- The difference in favor of LCZ696 was seen early in the trial and at each interim analysis
- Over the duration of the trial, the numbers of patients who would need to have been treated (NNT) to prevent:
  - one primary event was 21 patients, and
  - one death from CV causes was 32 patients

*Calculated with the use of stratified cox proportional-hazard models; ‡Two-sided p-values calculated by means of a stratified log-rank test without adjustment for multiple comparisons. CI=confidence interval; CV=cardiovascular; HF=heart failure; NNT=number needed to treat

McMurray et al. N Engl J Med 2014;371:993–1004

King Edward I (1272)

Banned burning of sea-coal in London

"Whosoever shall be found guilty of burning coal shall suffer the loss of his head"

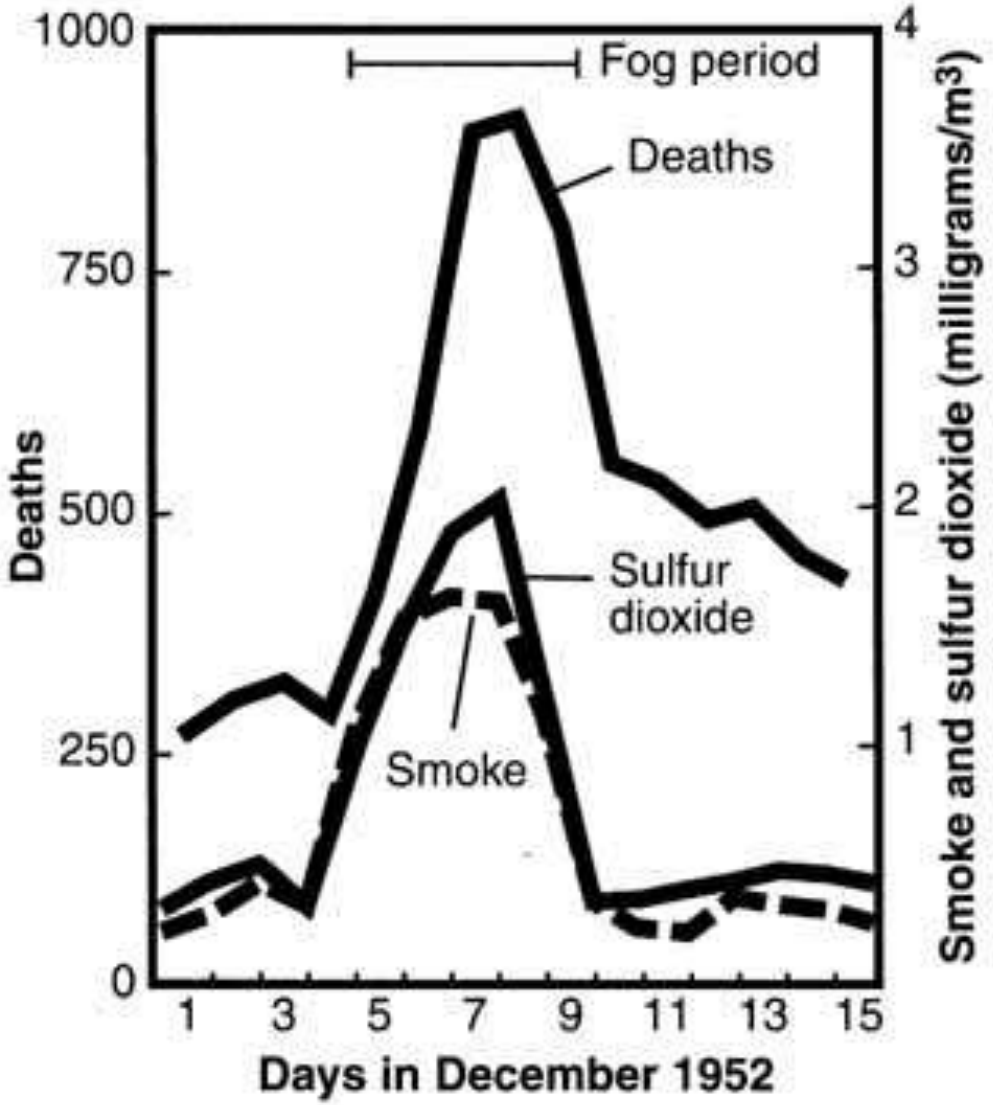
"...[London's] Inhabitants breathe nothing but an impure and thick Mist, accompanied with a fuliginous and filthy vapor,... corrupting the Lungs and disordering the entire habit of their Bodies;..."

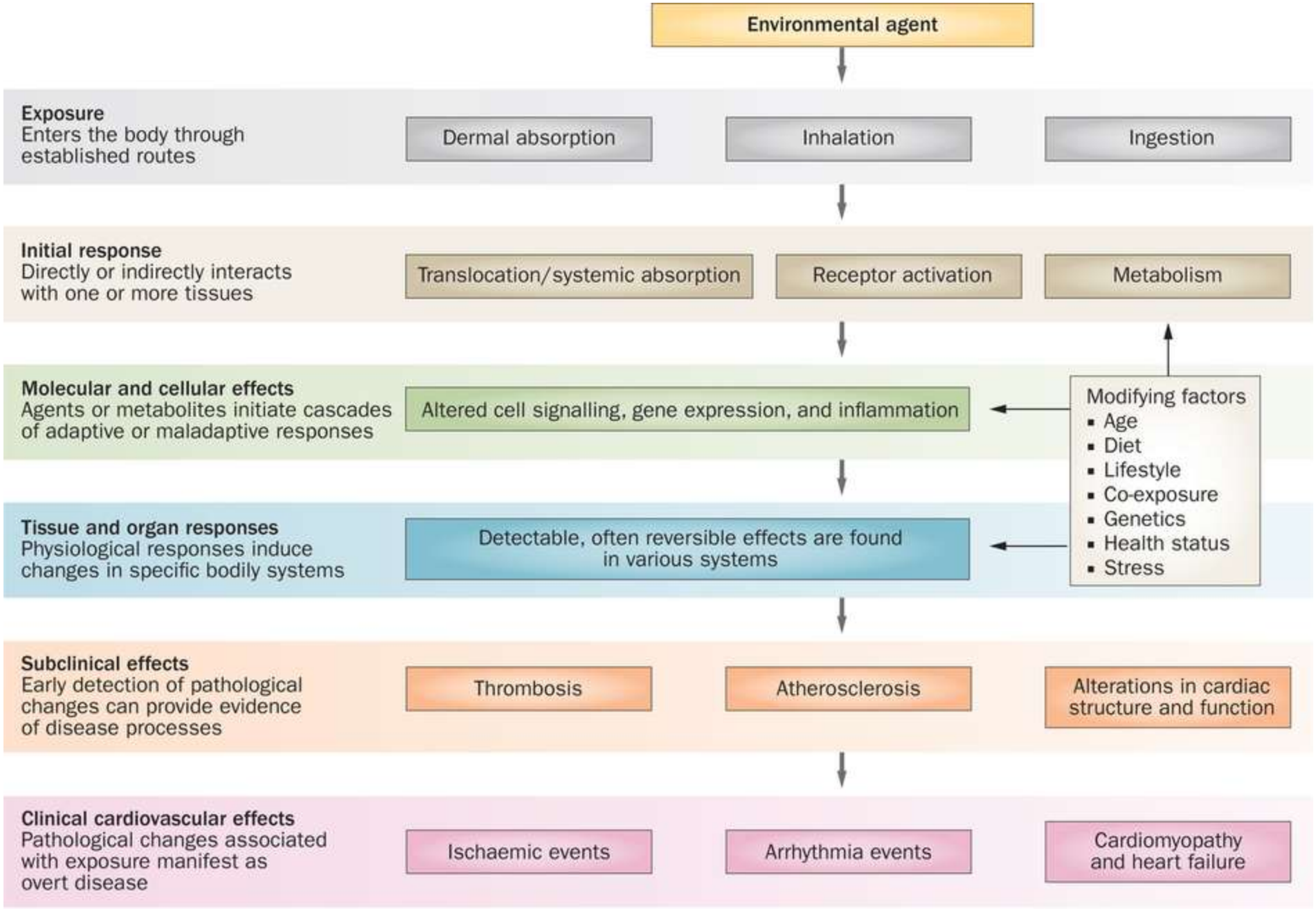
John Evelyn, *Fumifugium*, 1661

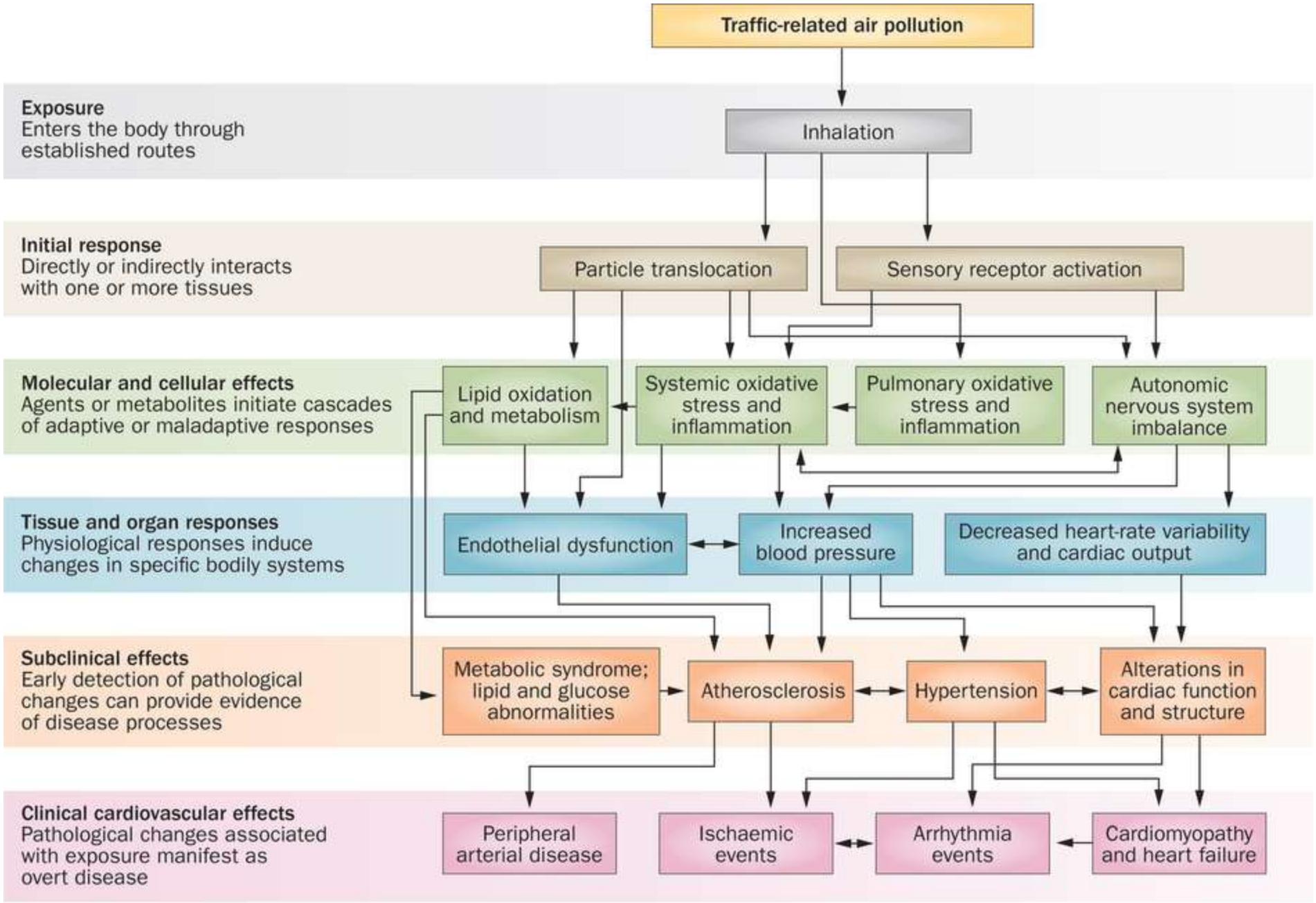
The houses of Parliament, Stormy Sky
Claude Monet, 1904

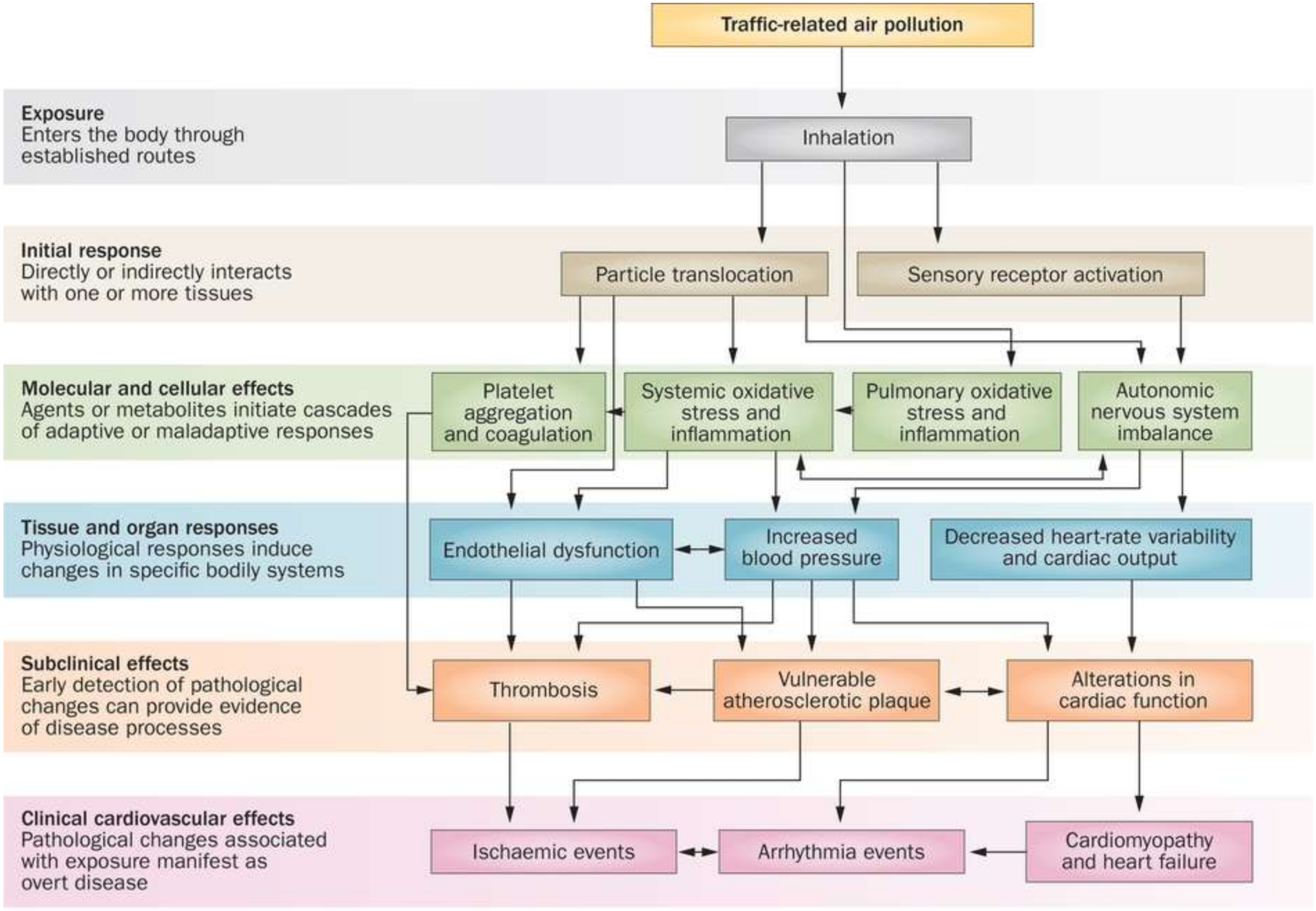
Slide from Robert Brook

London Smog 1952




Environmental agent
Exposure
Enters the body through established routes
Dermal absorption
Inhalation
Ingestion
Initial response
Directly or indirectly interacts with one or more tissues
Translocation/systemic absorption
Receptor activation
Metabolism
Molecular and cellular effects
Agents or metabolites initiate cascades of adaptive or maladaptive responses
Altered cell signalling, gene expression, and inflammation
Modifying factors
Age
Diet
Lifestyle
Co-exposure
Genetics
Health status
Stress
Tissue and organ responses
Physiological responses induce changes in specific bodily systems
Detectable, often reversible effects are found in various systems
Subclinical effects
Early detection of pathological changes can provide evidence of disease processes
Thrombosis
Atherosclerosis
Alterations in cardiac structure and function
Clinical cardiovascular effects
Pathological changes associated with exposure manifest as overt disease
Ischaemic events
Arrhythmia events
Cardiomyopathy and heart failure


Traffic-related air pollution
Exposure
Enters the body through established routes
Inhalation
Initial response
Directly or indirectly interacts with one or more tissues
Particle translocation
Sensory receptor activation
Molecular and cellular effects
Agents or metabolites initiate cascades of adaptive or maladaptive responses
Lipid oxidation and metabolism
Systemic oxidative stress and inflammation
Pulmonary oxidative stress and inflammation
Autonomic nervous system imbalance
Tissue and organ responses
Physiological responses induce changes in specific bodily systems
Endothelial dysfunction
Increased blood pressure
Decreased heart-rate variability and cardiac output
Subclinical effects
Early detection of pathological changes can provide evidence of disease processes
Metabolic syndrome; lipid and glucose abnormalities
Atherosclerosis
Hypertension
Alterations in cardiac function and structure
Clinical cardiovascular effects
Pathological changes associated with exposure manifest as overt disease
Peripheral arterial disease
Ischaemic events
Arrhythmia events
Cardiomyopathy and heart failure


Traffic-related air pollution
Exposure
Enters the body through established routes
Inhalation
Initial response
Directly or indirectly interacts with one or more tissues
Particle translocation
Sensory receptor activation
Molecular and cellular effects
Agents or metabolites initiate cascades of adaptive or maladaptive responses
Platelet aggregation and coagulation
Systemic oxidative stress and inflammation
Pulmonary oxidative stress and inflammation
Autonomic nervous system imbalance
Tissue and organ responses
Physiological responses induce changes in specific bodily systems
Endothelial dysfunction
Increased blood pressure
Decreased heart-rate variability and cardiac output
Subclinical effects
Early detection of pathological changes can provide evidence of disease processes
Thrombosis
Vulnerable atherosclerotic plaque
Alterations in cardiac function
Clinical cardiovascular effects
Pathological changes associated with exposure manifest as overt disease
Ischaemic events
Arrhythmia events
Cardiomyopathy and heart failure

Global association of air pollution and heart failure: a systematic review and meta-analysis

Anoop S V Shah, Jeremy P Langrish, Harish Nair, David A McAllister, Arnanda L Hunter, Ken Donaldson, David E Newby, Nicholas L Mills

Summary

Interpretation Air pollution has a close temporal association with heart failure hospitalisation and ***heart failure mortality***. Although more studies from developing nations are required, air pollution is a ***pervasive public health issue*** with major cardiovascular and health economic consequences, and it ***should remain a key target for global health policy***.

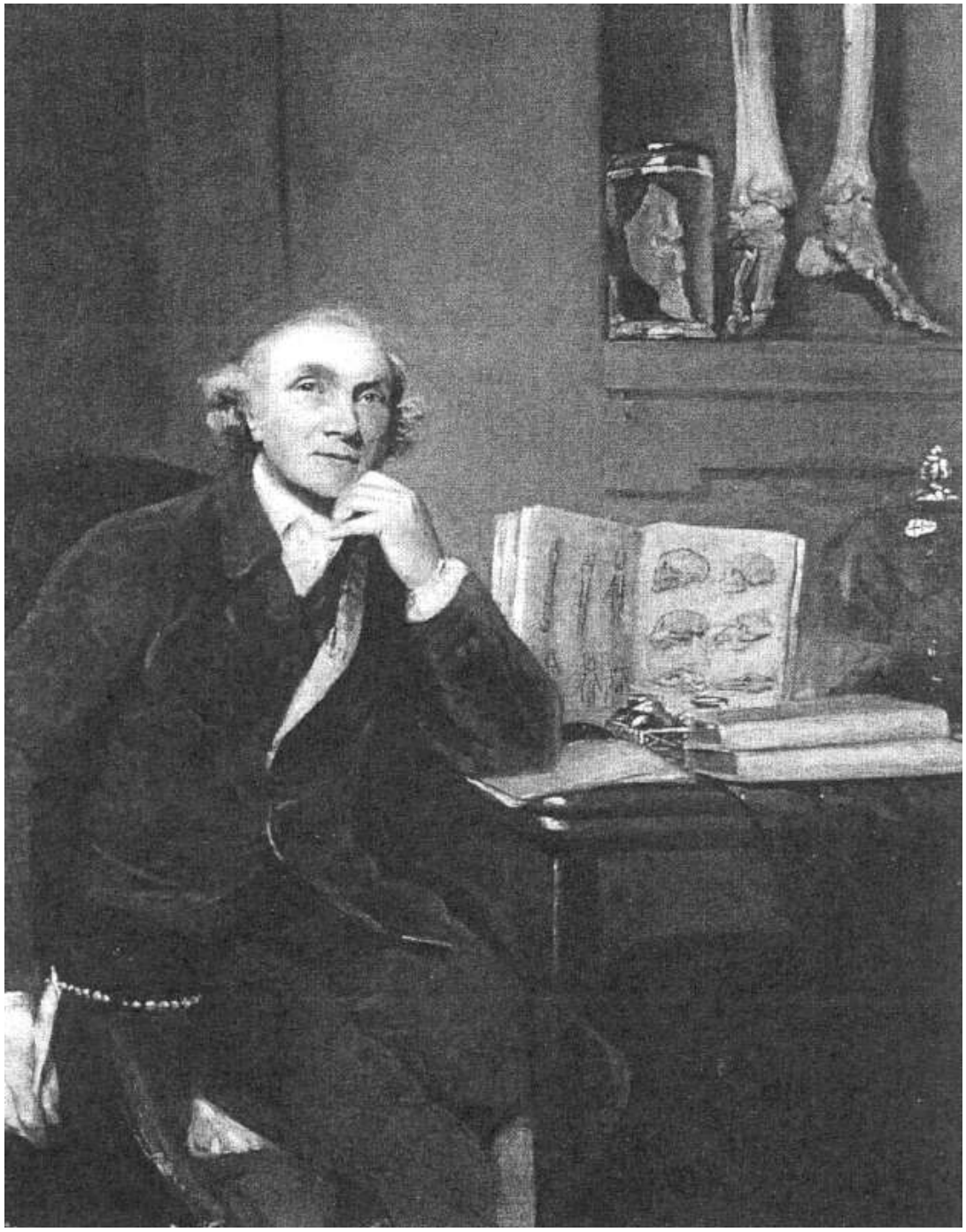
exposure, with more persistent effects for $PM_{2.5}$. In the USA, we estimate that a mean reduction in $PM_{2.5}$ of 3·9 μg/m³ would prevent 7978 heart failure hospitalisations and save a third of a billion US dollars a year.

Interpretation Air pollution has a close temporal association with heart failure hospitalisation and heart failure mortality. Although more studies from developing nations are required, air pollution is a pervasive public health issue with major cardiovascular and health economic consequences, and it should remain a key target for global health policy.

Funding British Heart Foundation.

Air Pollution and Heart Disease

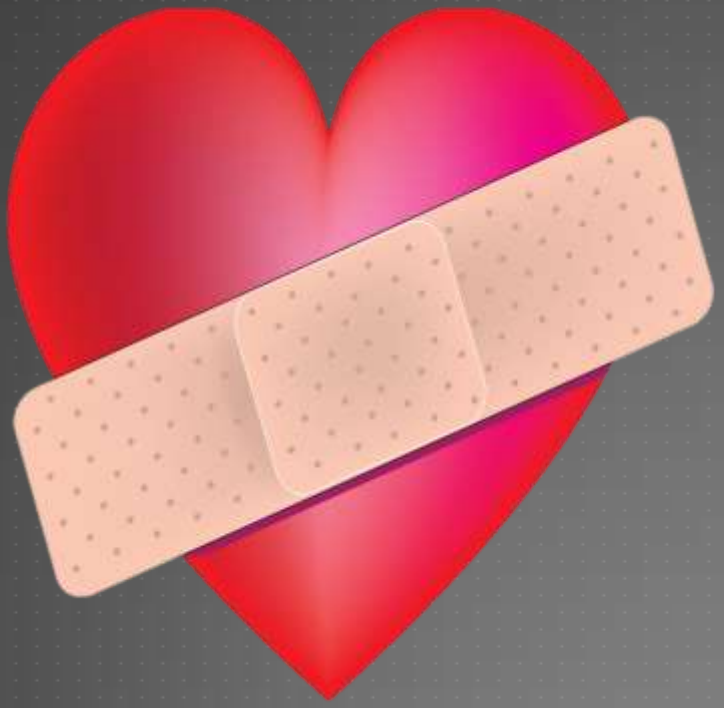
Time for BAMP to make a Statement



“I am now at the mercy of any rogue who cares to anger me.”

John Hunter

Joint Symposium of the Caribbean Cardiac Society and the American College of Cardiology



BROKEN HEART CARDIOMYOPATHY A CASE DISCUSSION

Dr R.J.B. Massay

American College of Cardiology
San Francisco, March 11, 2013

BROKEN HEART CARDIOMYOPATHY

- Female: 37 yr old, Customs Broker
- Never smoked, no alcohol consumption, no physical exercise
- Family History negative for:
  - Sudden cardiac death
  - Premature coronary artery disease
- 5 yr history of asthma; symptom free for 1 yr
- Endometriosis
- MVA: injuries sustained to low back and chest, facial scars (Awaiting Insurance Compensation)

BROKEN HEART CARDIOMYOPATHY

- Episodic chest pain for 2/12
- Retrosternal radiating to left arm
- Of variable duration with spontaneous resolution
- No identifiable precipitating factors
- Aggravated by "moving around"
- Relieved by rest
- No associated autonomic discharge
- "Like an elephant sitting on my chest"

BROKEN HEART CARDIOMYOPATHY

- Self-referred to an emergency room because of a "particularly bad episode" which occurred while bathing "I could not breathe"

BROKEN HEART CARDIOMYOPATHY

- Seen approx 7 hrs post onset of pain
- Normal heart sounds with no murmurs
- Respiratory, GI and CNS all normal
- BP: 148/98 mmHg

D.O.B.:
Meds:
Class:
Dr:
Tech: #1

Age 37

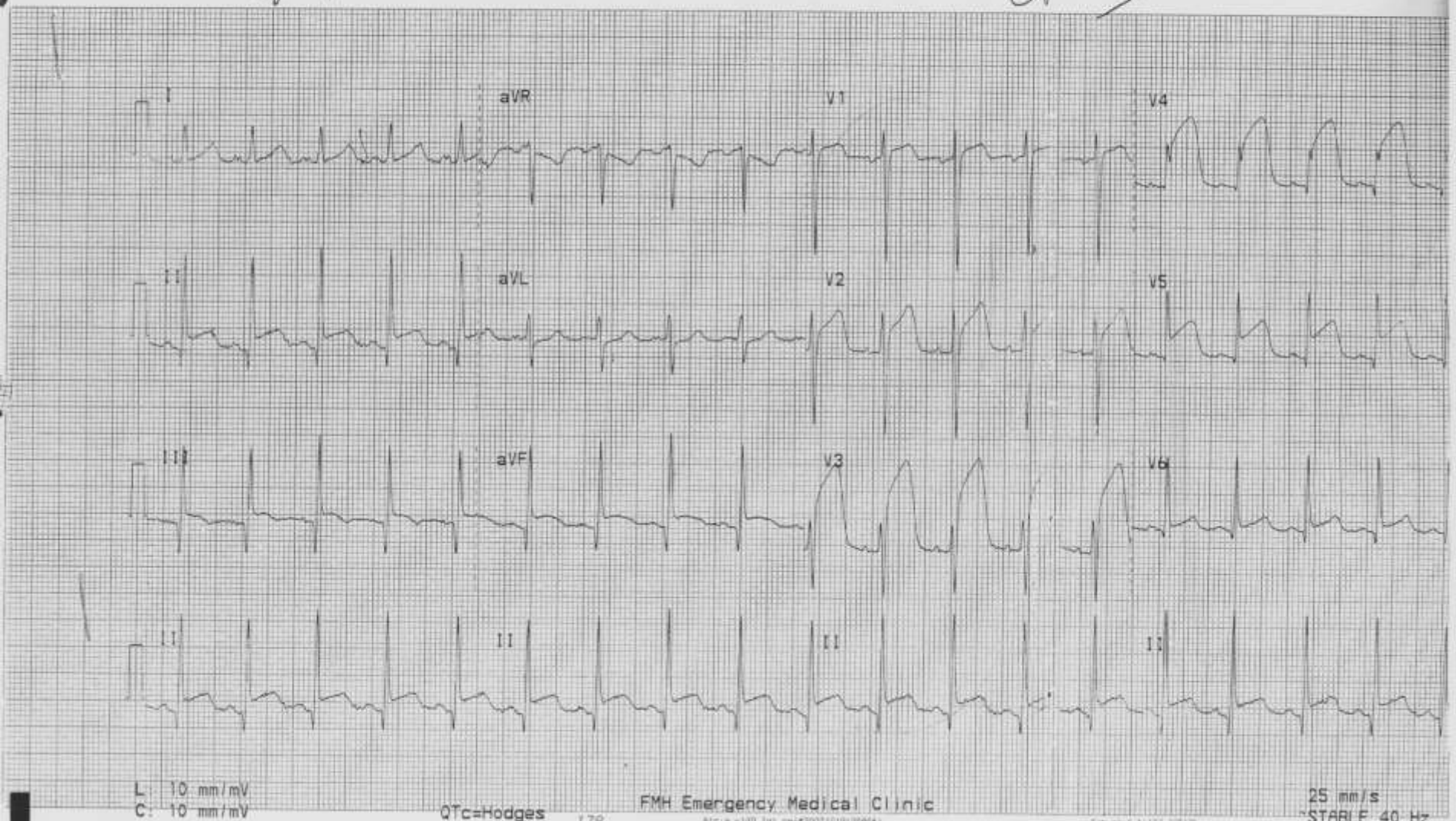
Vent. Rate:	111	bpm
RR Interval:	537	ms
PR Interval:	126	ms
QRS Duration:	80	ms
QT Interval:	328	ms
QTc Interval:	417	ms
QT Dispersion:	58	ms
P-R-T AXIS:	43°	66° 43°

Extensive ST elevation, CONSIDER ACUTE INFARCT

Abnormal ECG

* Unconfirmed Analysis *

extensive antero lateral MI



L: 10 mm/mV
C: 10 mm/mV

QTc=Hodges

FMH Emergency Medical Clinic

25 mm/s
STABLE 40 Hz

BROKEN HEART CARDIOMYOPATHY

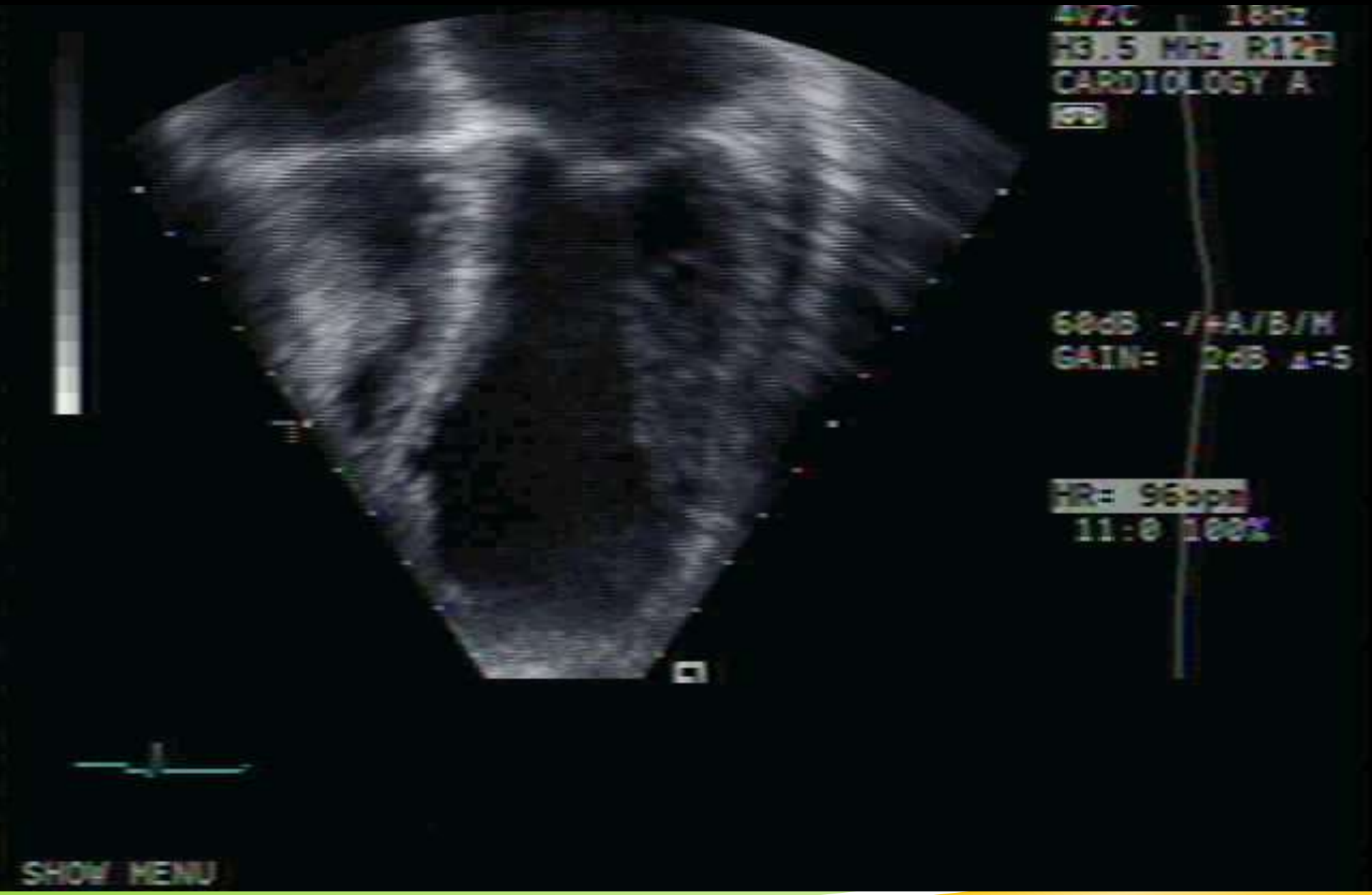
- Dx: ST Segment elevation myocardial infarction
- No thrombolysis
- Rx: Clopidrogel/Aspirin/Enoxaparin/Morphine/ Bisoprolol/Ramipril/Atorvastatin/GTN infusion

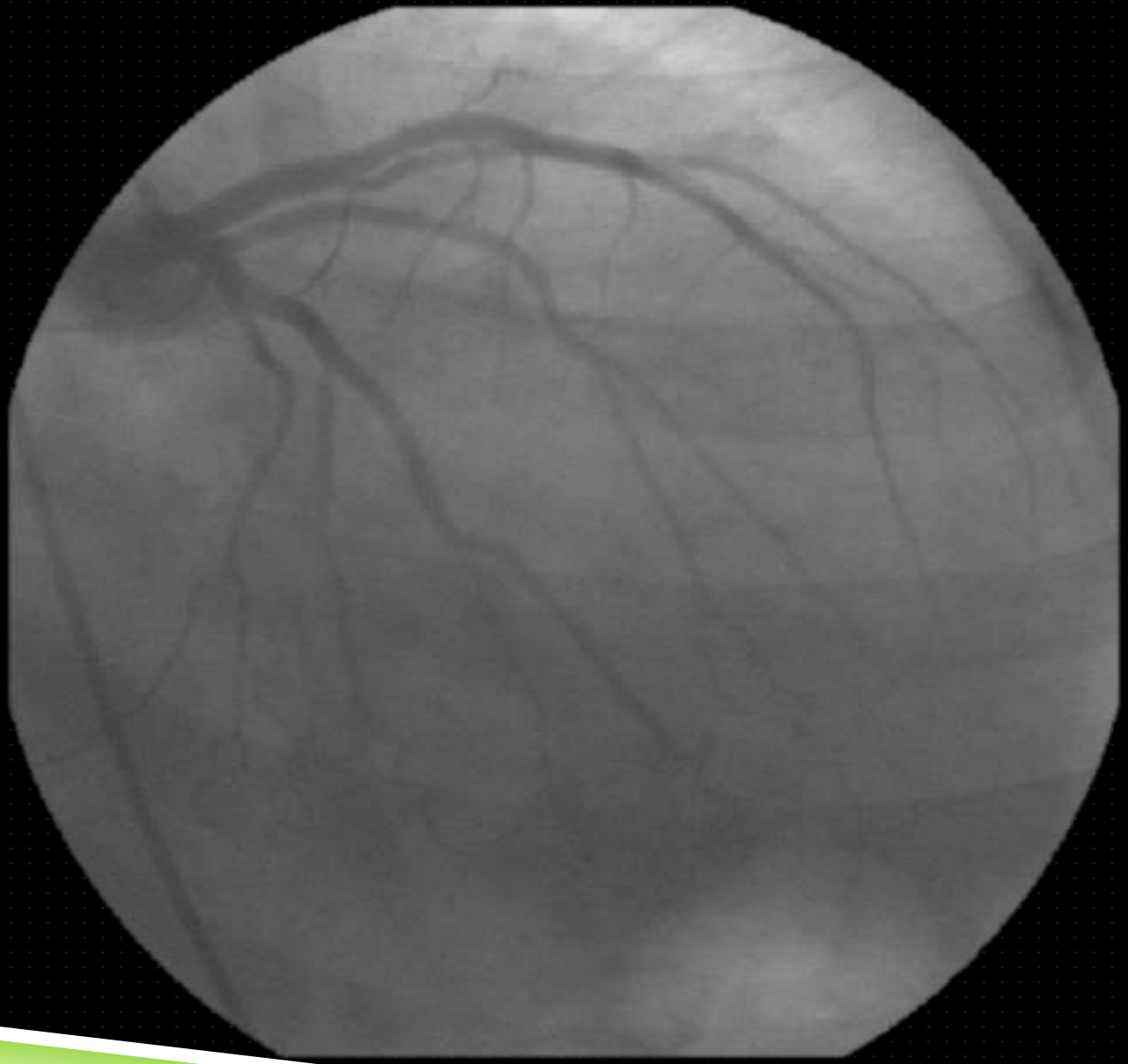
BROKEN HEART CARDIOMYOPATHY

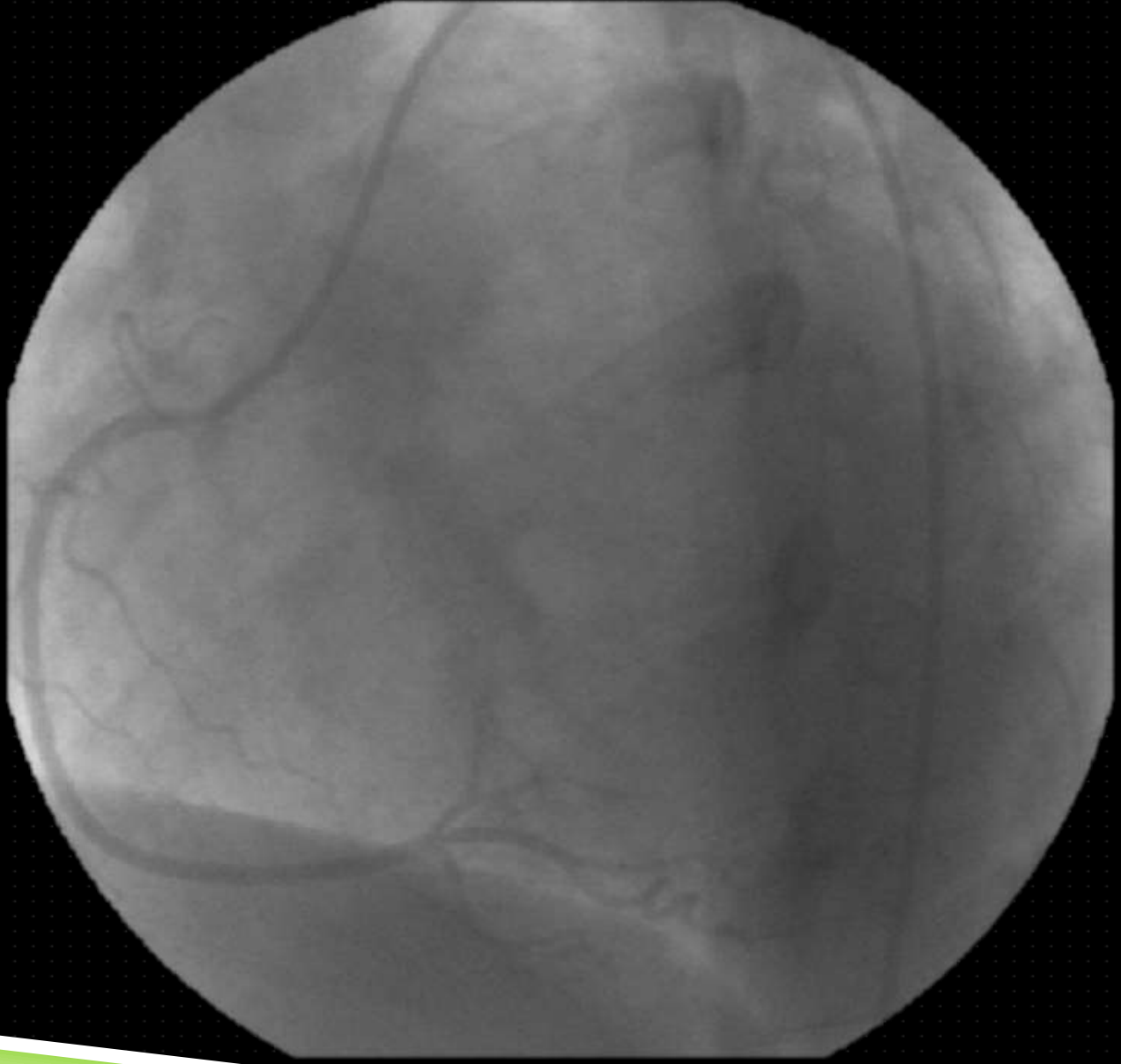
Cardiac enzyme profile

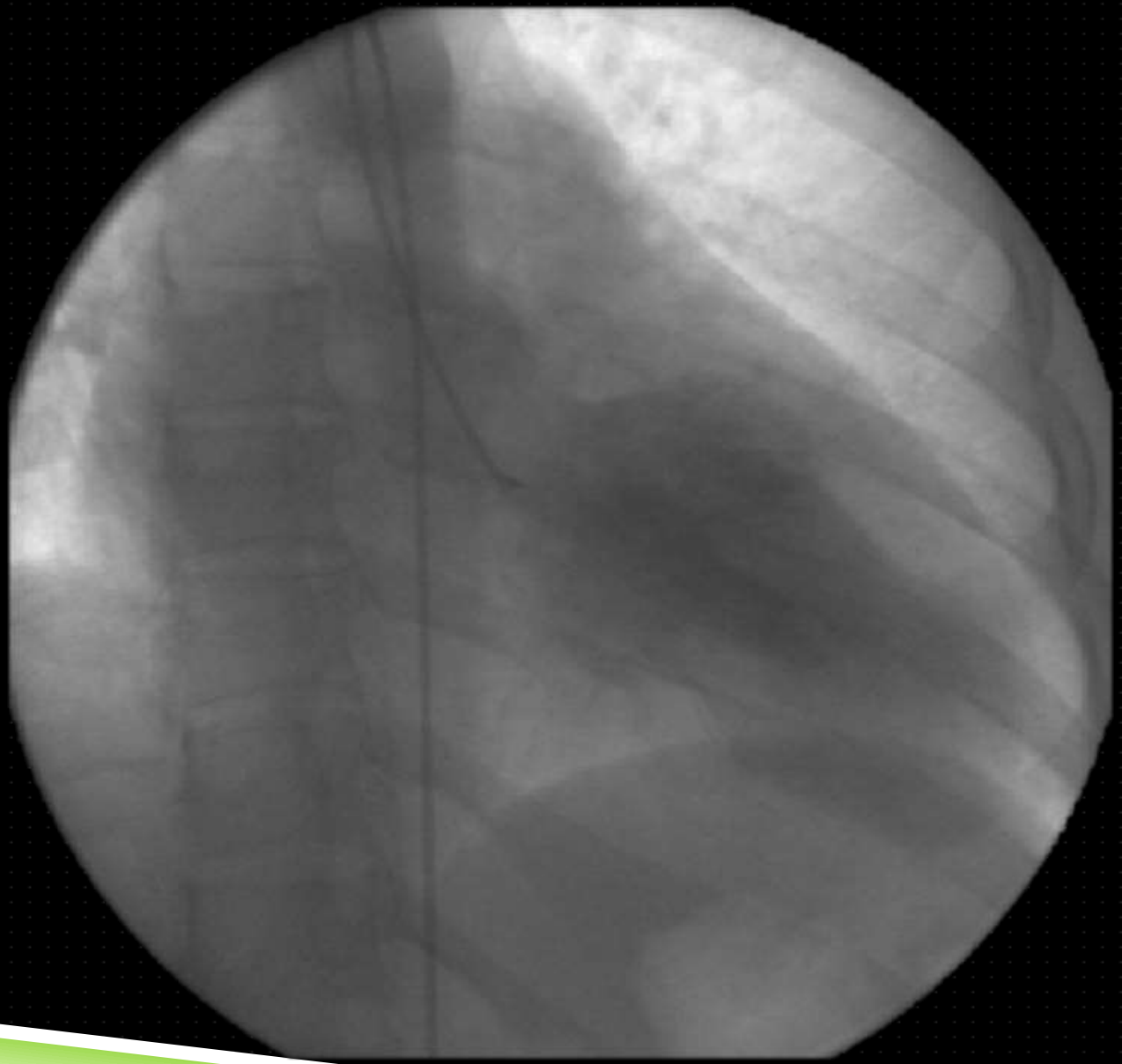
	T_0 + 7 hrs	T_0 + 10 hrs	T_0 + 15 hrs	Normal IU/L
AST	94	99	147	10 - 42
CK	645	717	1296	48 - 376
CK-MB	116	97	186	8 - 82
cTn I	Not Available			

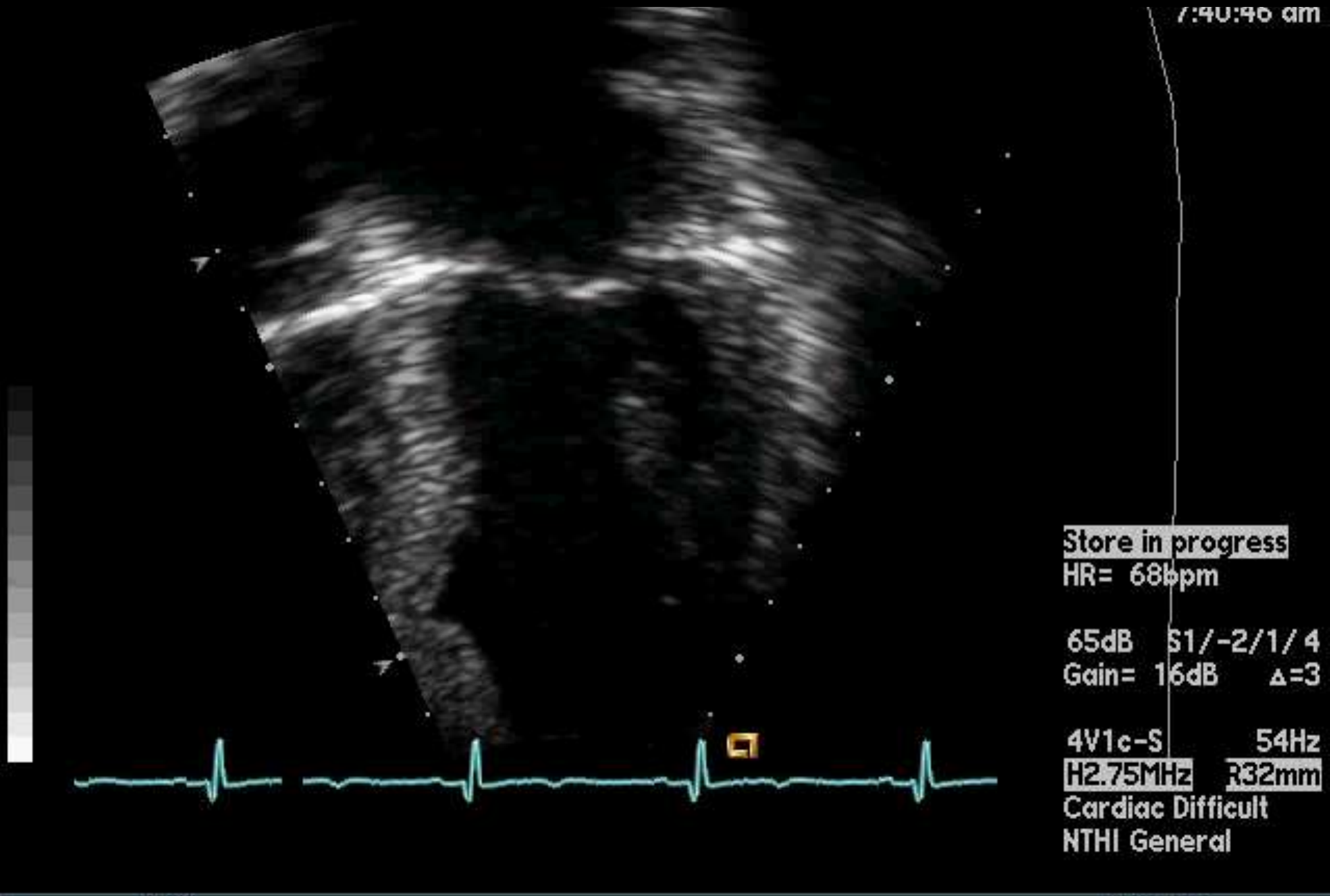
T_0 = Onset of pain


H3.5 MHz R12
CARDIOLOGY A
60dB -/+A/B/M
GAIN: 2dB A=5
HR= 96bpm
11:0
SHOW MENU








7:40:46 am
Store in progress
HR= 68bpm
65dB S1/-2/1/4
Gain= 16dB Δ=3
4V1c-S 54Hz
H2.75MHz R32mm
Cardiac Difficult
NTHI General
Exit
Res Box

SUMMARY

- Acute coronary syndrome with normal coronary arteries and LV apical dyskinesis in a female in a stressful situation = Takotsubo cardiomyopathy
- Premenopausal as compared to oestrogen deficient postmenopausal state
- Recovery delayed (awaiting compensation, so stressor persists)
- Endometriosis

Statins and the Skin

Pityriasis lichenoides chronica Associated with Use of HMG-CoA Reductase Inhibitors

RJB Massay[1], AA Maynard[2]

ABSTRACT

Herein, we present three cases of Pityriasis lichenoides chronica *(PLC) in patients who developed the rash after use of 3-hydroxy-3-methyl-glutaryl-Coenzyme A (HMG-CoA) reductase inhibitors. The patients had complete resolution after standard treatment by dermatologists and withdrawal of the offending agents. In one case, the patient had a previous episode of a similar rash that occurred with HMG-CoA reductase inhibitors use many years previously.* Pityriasis lichenoides chronica *is a condition of unknown aetiology. Several agents have been associated with its presentation. We postulate HMG-CoA reductase inhibition in skin presents a final common pathway for the presentation of PLC in select patients.*

Keywords: Erythematous rash, HMG-CoA reductase inhibitors, *Pityriasis lichenoides chronica*, statins

West Indian Med J 2012; 61 (7): 743

Canadian Journal of Cardiology
Journal canadien de cardiologie

Diagnosis, Prevention, and Management of Statin Adverse Effects and Intolerance: Canadian Working Group Consensus Update

G.B. John Mancini, MD, A. Yashar Tashakkor, MD, Steven Baker, MD, Jean Bergeron, MD, David Fitchett, MD, Jiri Frohlich, MD, Jacques Genest, MD, Milan Gupta, MD, Robert A. Hegele, MD, Dominic S. Ng, MD, Glen J. Pearson, PharmD, Janet Pope, MD

DOI: http://dx.doi.org/10.1016/j.cjca.2013.09.023

References

55. Massay, R.J., Maynard, A.A. **Pityriasis lichenoides chronica associated with use of HMG-CoA reductase inhibitors.** *West Indian Med J.* 2012;61:743–745.

PubMed

List 1: Top Articles, Since 2012 (publication date of the domain article), in the Domain of Article 23620974

Note: when none of the articles in the list is relevant to your topic, this means there haven't been new publications on your topic in this time-frame.

1. ☐ González Rodríguez AJ, Montesinos Villaescusa E, Jordá Cuevas E: **Pityriasis lichenoides chronica associated with herpes simplex virus type 2.** *Case Rep Dermatol Med*; 2012;2012:737428

4. ☐ Zegpi MS, Ruiz FM, Porras NK: **[Pityriasis Lichenoides: Case report and review of the literature].** *Rev Chil Pediatr*; 2015 Mar-Apr;86(2):121-5

5. ☐ Massay RJ, Maynard AA: **Pityriasis lichenoides chronica associated with use of HMG-CoA reductase inhibitors.** *West Indian Med J*; 2012 Oct;61(7):743-5

Your article

3 messages

Roselyn E Epps <rozeepps@gmail.com> Thu, Oct 8, 2015 at 10:32 AM
To: jeffmassay@gmail.com

Dear Dr. Massay,

I would like to request the full article from your publication regarding Pityriasis Lichenoides Chronica and HMG-CoA reductase inhibitors in the West Ind.sMedical Journal, Oct. 2012. If you no longer have a copy, please let me know where one can be found.

Thank you for assistance with this matter.

Roselyn E. Epps, MD
Silver Spring, Maryland, USA

Be a Plenary Speaker at Dermatology 2016 Chicago

1 message

Dermatology 2016 <dermatology@conferenceseries.net>
To: jeffmassay@gmail.com

Thu, Nov 5, 2015 at 8:03 AM

Dear Dr. R J B Massay,

We are pleased to invite you to the **"6th International Conference on Clinical & Experimental Dermatology"** scheduled during May 05-07, 2016. This conference will be held at Chicago, USA.

Dermatology 2016 is a specially designed cluster conference. The main theme of the conference is **"Global Tuning of Innovative Therapies"** which covers a wide range of critically important sessions. Dermatology 2016 would lay a platform for the interaction between experts around the world and aims in accelerating scientific discovery.

This Conference will examine research and development both locally and internationally. It is an honour and privilege to invite you to participate in this Conference as Speaker for Dermatology 2016. **Keynote Speakers Dr. Barry Lycka (Barry Lycka Professional Corp), Canada** and **Dr. Bozena Michniak-Kohn Director of Center for Dermal Research(USA** and experts from around the globe will be expected to share their knowledge. We believe that your contribution to this field is unparalleled and this topic will be of great benefit.

Pityriasis lichenoides chronica associated with Statins

- 3 cases over 10 years
- Different Statins
- Pityriasis lichenoides chronica etiology unknown
- Lipid bilayer in stratum corneum synthesizes cholesterol

Pityriasis Lichenoides Chronica (PLC) Associated with Statins

- Statins can block cholesterol synthesis in the skin increasing transepidermal water loss and resulting in scaling in the skin
- Changes in stratum corneum lipids is the initiating triggering factor for the inflammatory reaction seen in cases of PLC

Selected Topics

- Ticagrelor in acute NSTEMI
- Angiotensin – Neprilysin inhibition to treat heart failure
- Stress
  - Environmental
  - Psychogenic – Case presentation of Takotsubo cardiomyopathy
- Something Old
  - Statins and the skin