

Cardiovascular Disease in **Respiratory Care**

Rethinking the Clinical Implications

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Shreveport, Louisiana

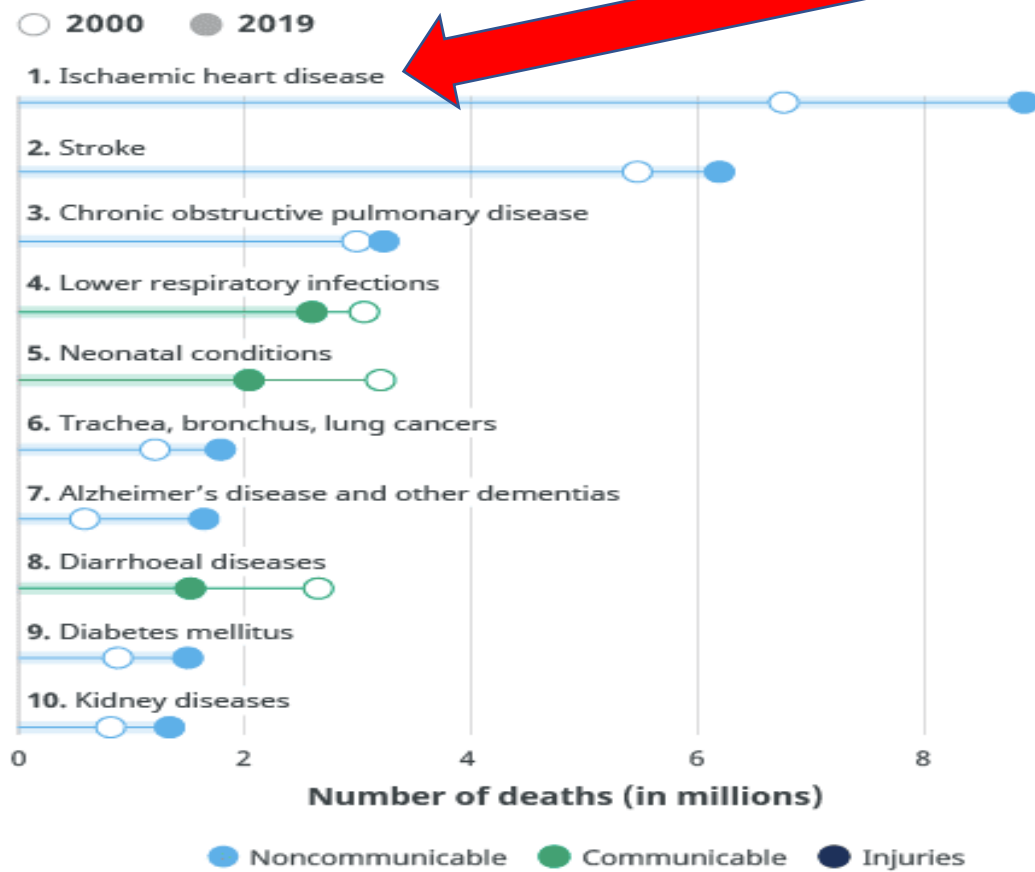
Objectives

- *Describe and classify* the general varieties of prevalent cardiovascular (CV) diseases
- *Discuss* **Respiratory Therapy**-pertinent clinical relevance of patient care in populations with both acute and chronic CV diseases



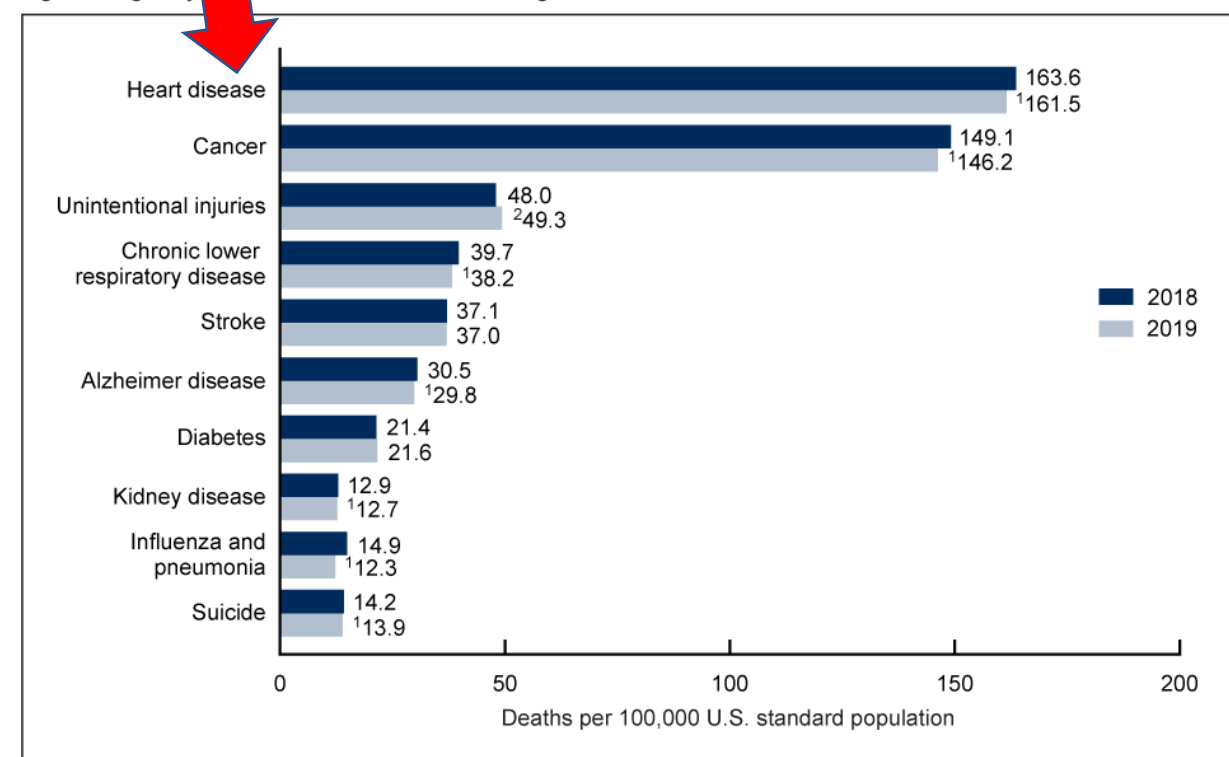


Leading causes of death globally



Source: WHO Global Health Estimates.

Figure 4. Age-adjusted death rates for the 10 leading causes of death in 2019: United States, 2018 and 2019



¹Statistically significant decrease in age-adjusted death rate from 2018 to 2019 ($p < 0.05$).
²Statistically significant increase in age-adjusted death rate from 2018 to 2019 ($p < 0.05$).
 NOTES: A total of 2,854,838 resident deaths were registered in the United States in 2019. The 10 leading causes of death accounted for 73.4% of all deaths in the United States in 2019. Causes of death are ranked according to number of deaths. Rankings for 2018 data are not shown. Data table for Figure 4 includes the number of deaths for leading causes. Access data table for Figure 4 at: <https://www.cdc.gov/nchs/data/databriefs/db395-tables-508.pdf#4>.
 SOURCE: National Center for Health Statistics, National Vital Statistics System, Mortality.



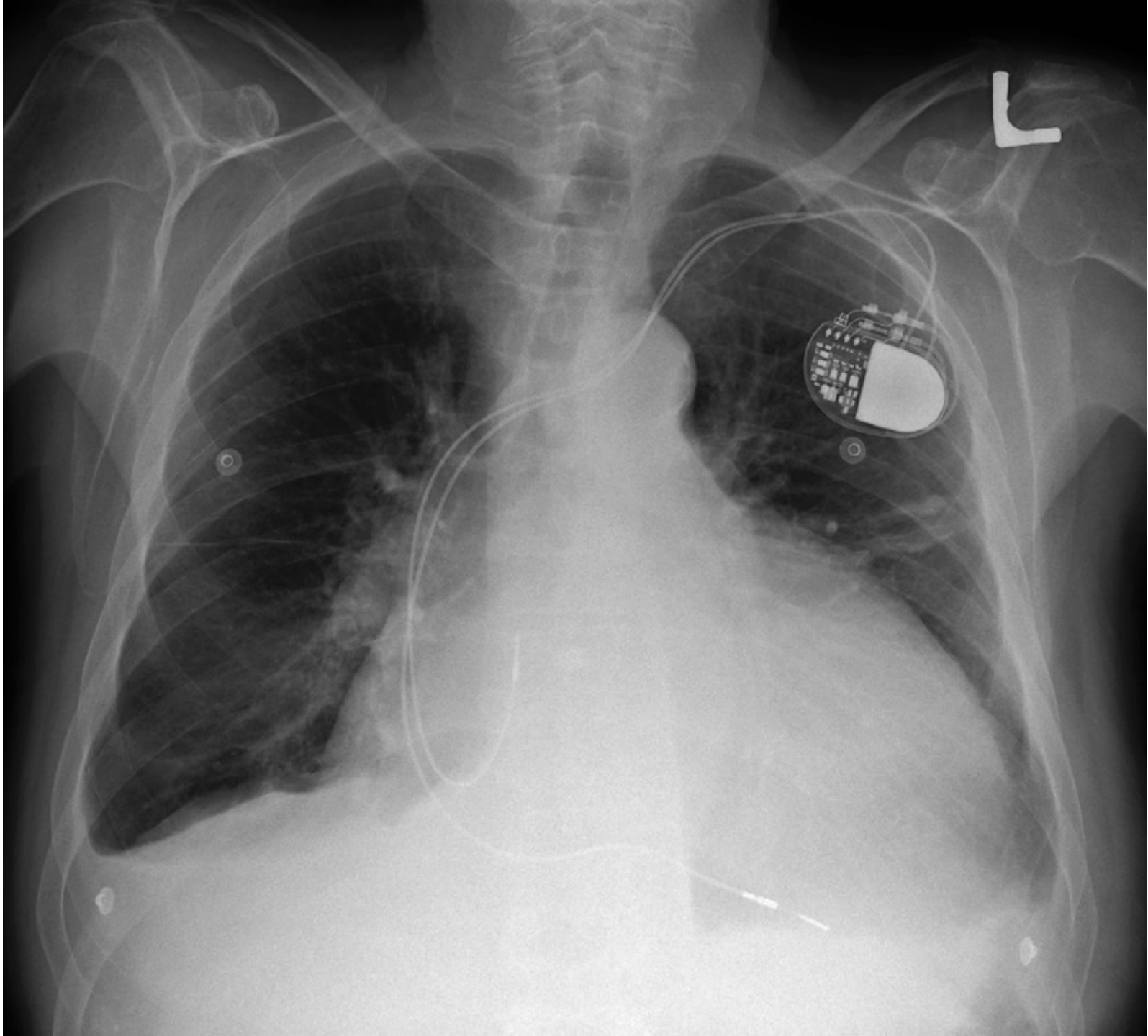
Centers for Disease Control and Prevention

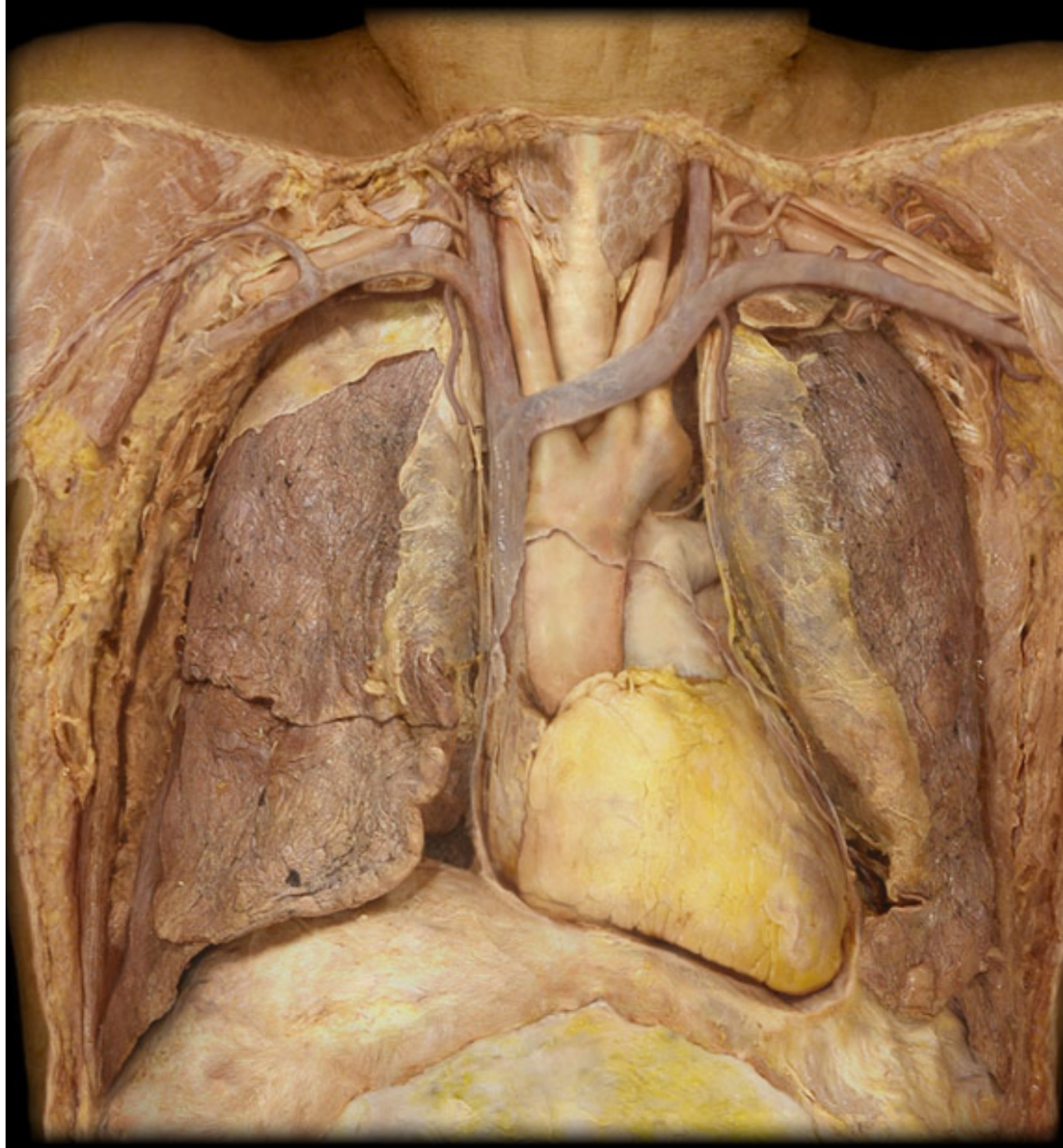
CDC 24/7: Saving Lives, Protecting People™

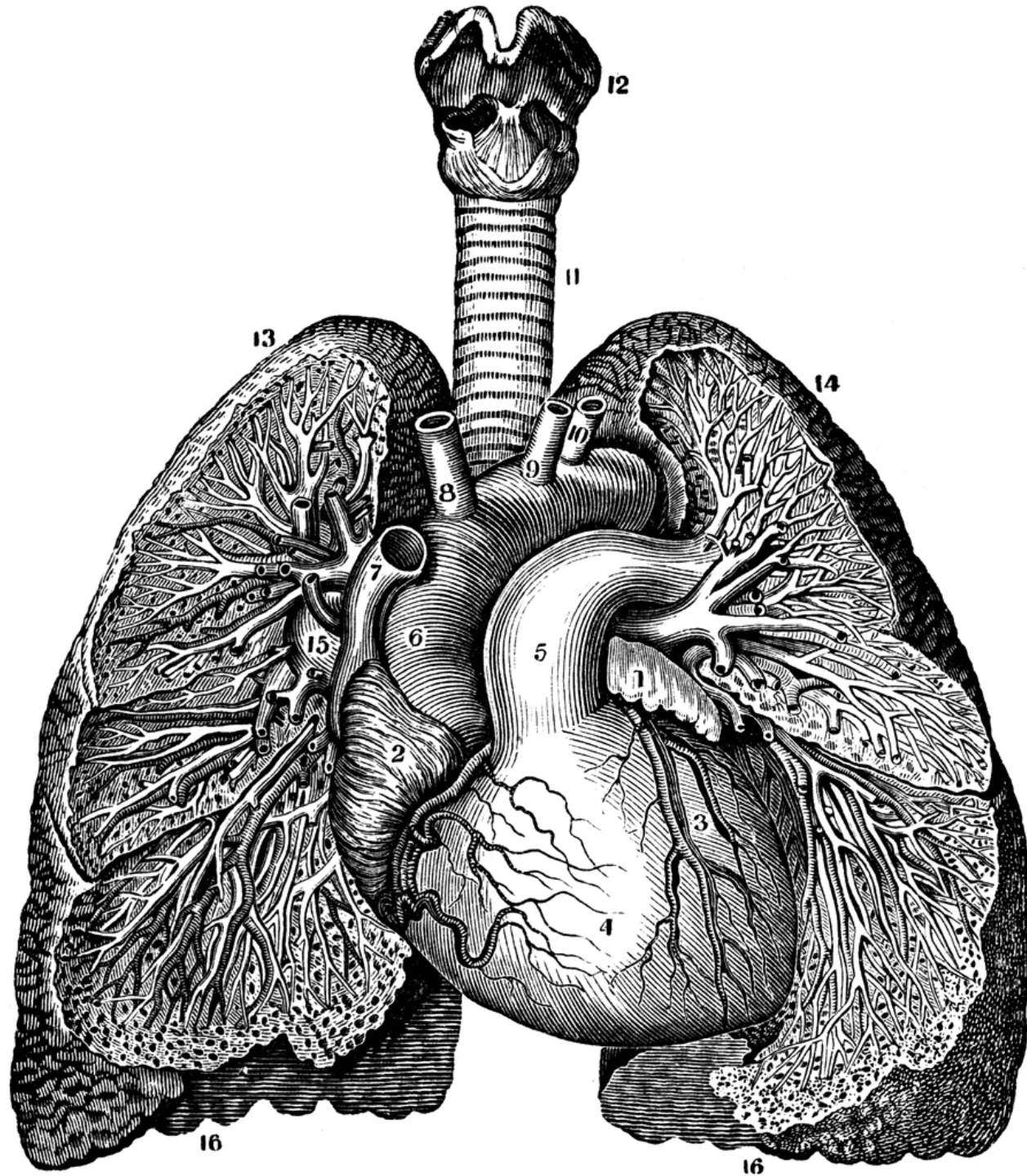
Heart Disease in the United States

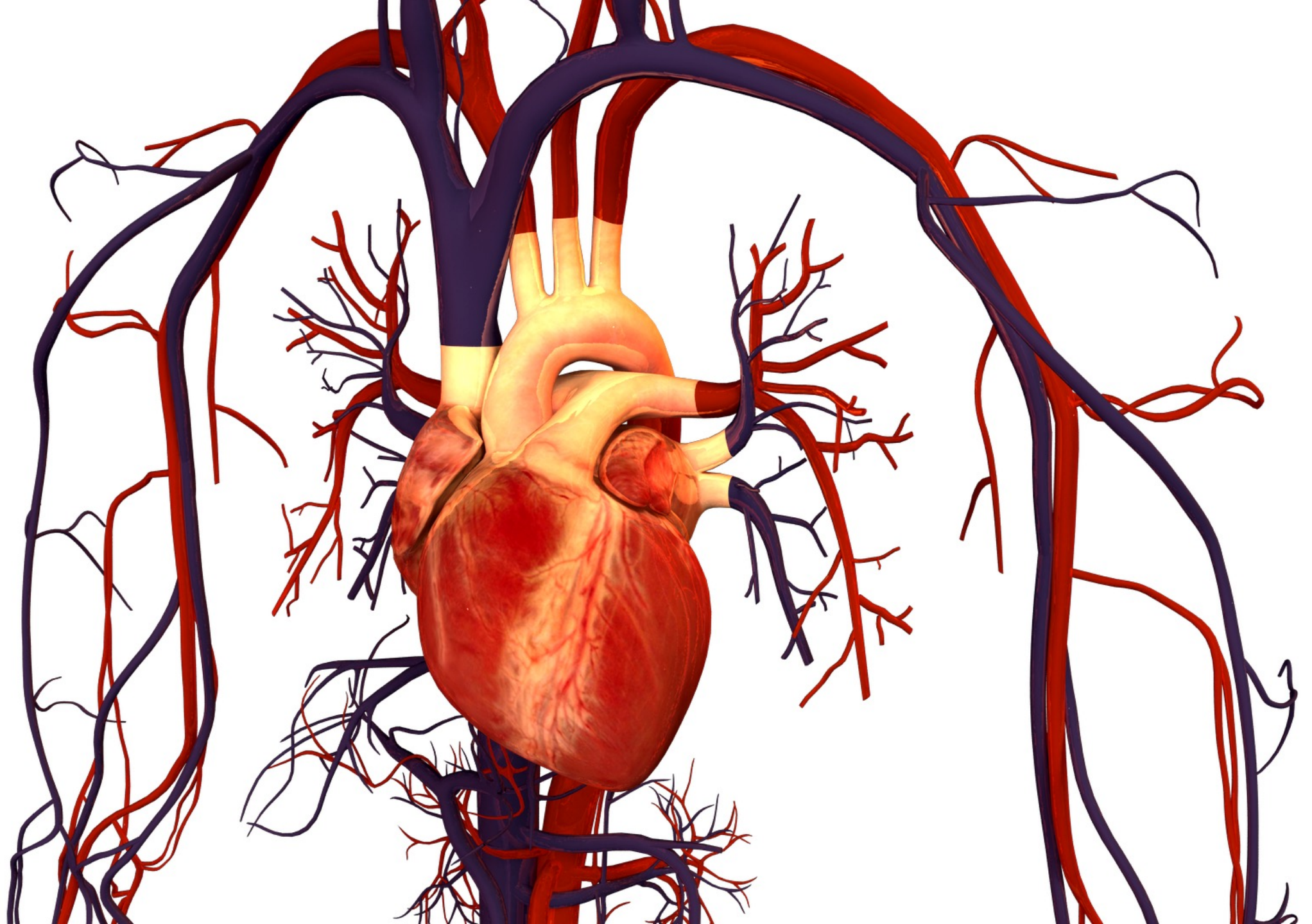
- Heart disease is the **leading cause of death** for men, women, and people of most racial and ethnic groups in the United States.¹
- **One person dies every ~~36~~ seconds** in the United States from cardiovascular disease.¹
34 seconds
- About ~~655,000~~ **Americans** die from heart disease each year—that's **1 in every 4 deaths**.² **697,000**
- Heart disease costs the United States about **\$219 billion** each year from 2014 to 2015.³ This includes the cost of health care services, medicines, and lost productivity due to death.

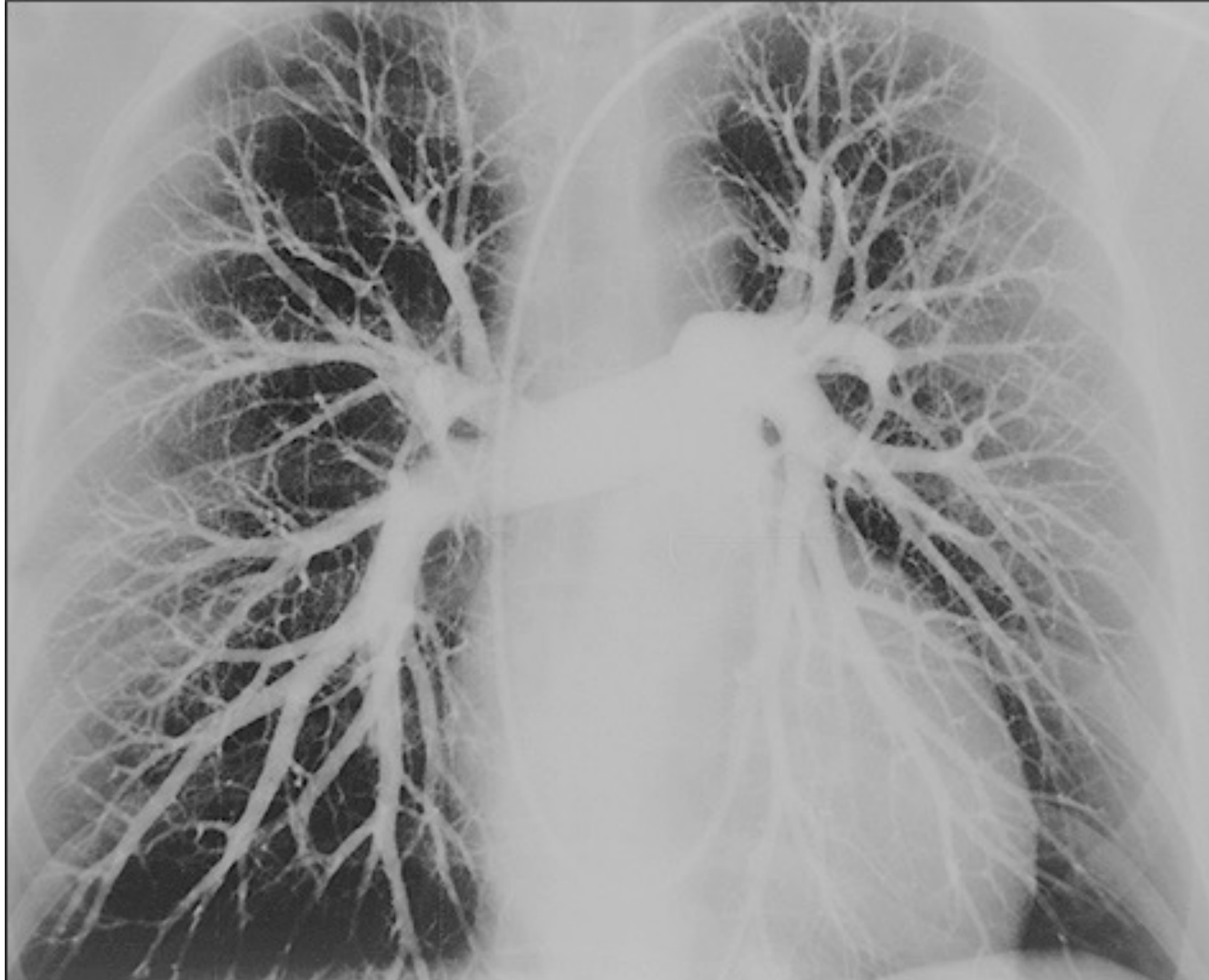















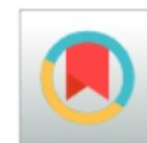
General Types of **Heart Disease**

- Valvular Disease
- Heart Failure
- Coronary Artery Disease (CAD)
- Arrhythmia




Coronary Heart Disease
Cerebrovascular disease
Peripheral artery disease
Aortic Atherosclerosis

Association of Cardiovascular Disease With Respiratory Disease

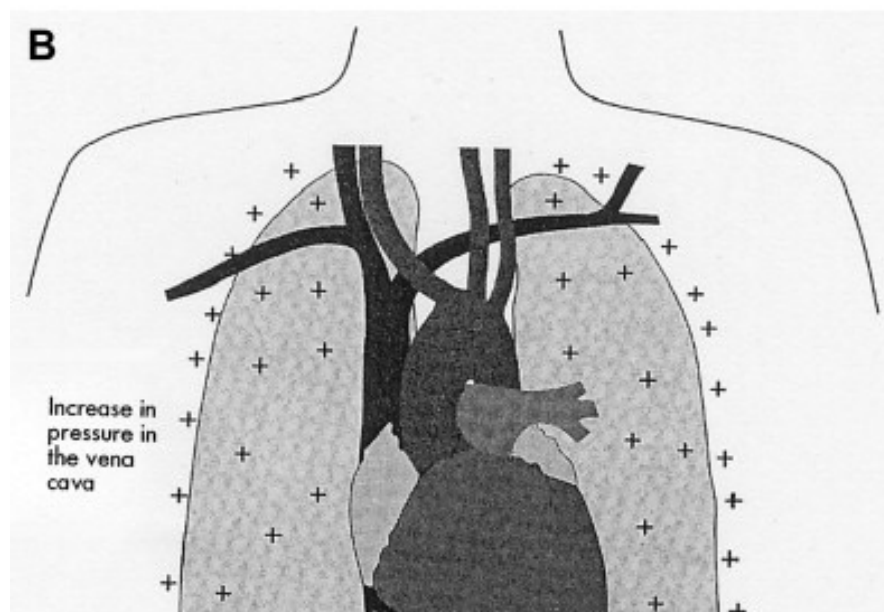
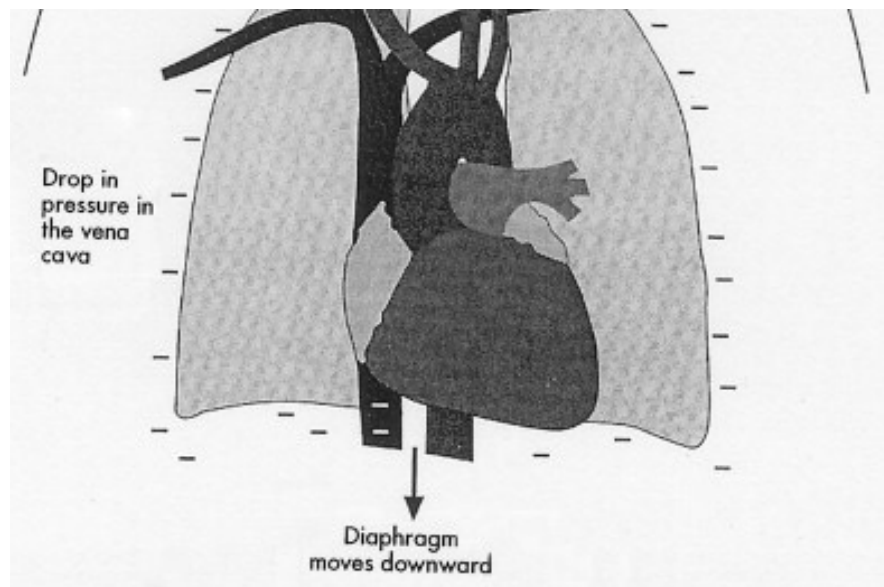


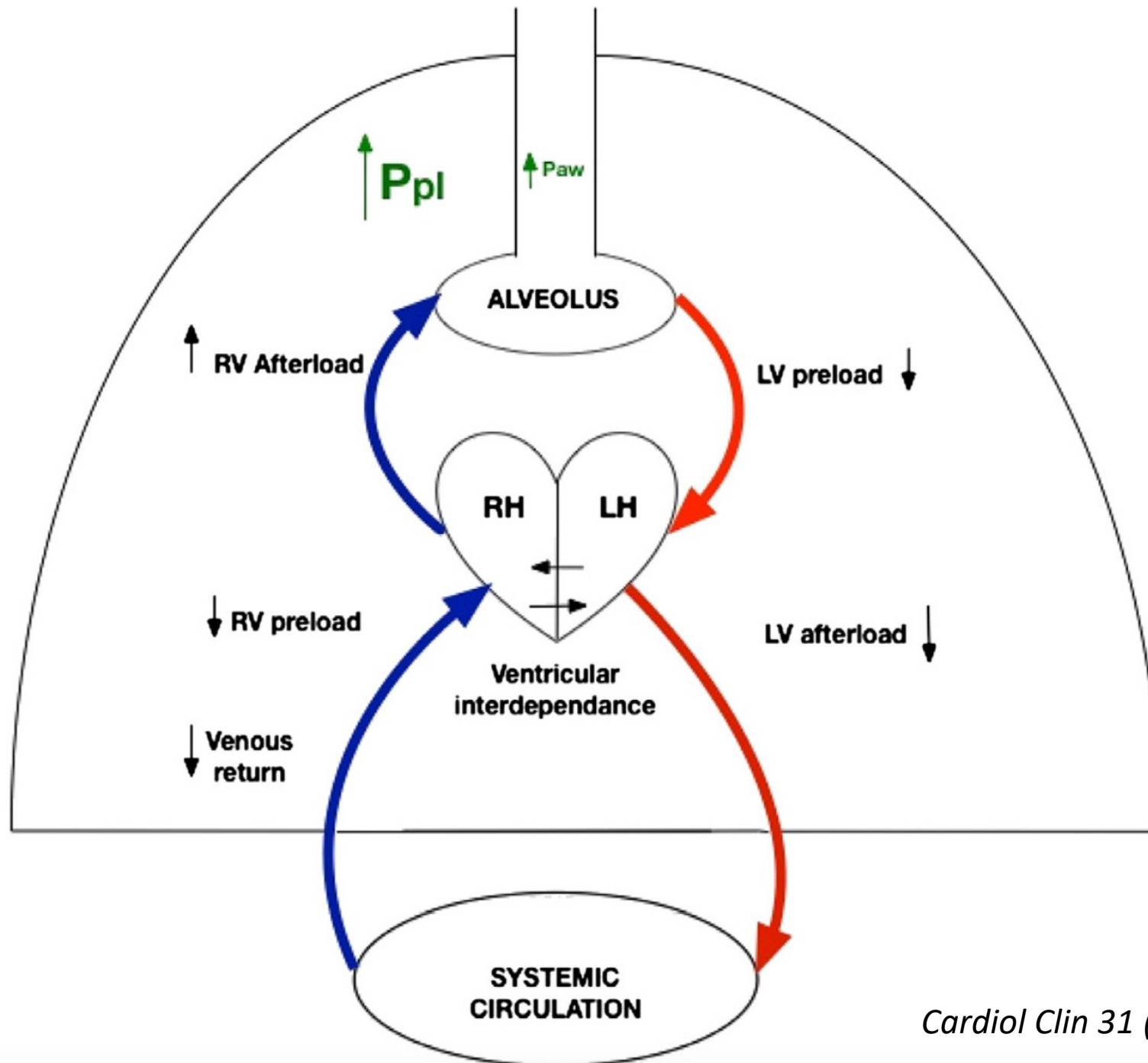
Paul Carter, MBC_HB,^{a,b} Jakub Lagan, LEKARZ,^{c,d} Christien Fortune, MBC_HB,^c Deepak L. Bhatt, MD, MPH,^e
Jørgen Vestbo, DR_{MED}SCI,^{d,f} Robert Niven, MBC_HB,^{d,f} Nazia Chaudhuri, MBC_HB, PHD,^{d,f} Erik B. Schelbert, MD, MS,^{g,h,i}
Rahul Potluri, MBC_HB,^a Christopher A. Miller, MBC_HB, PHD^{c,d,j}

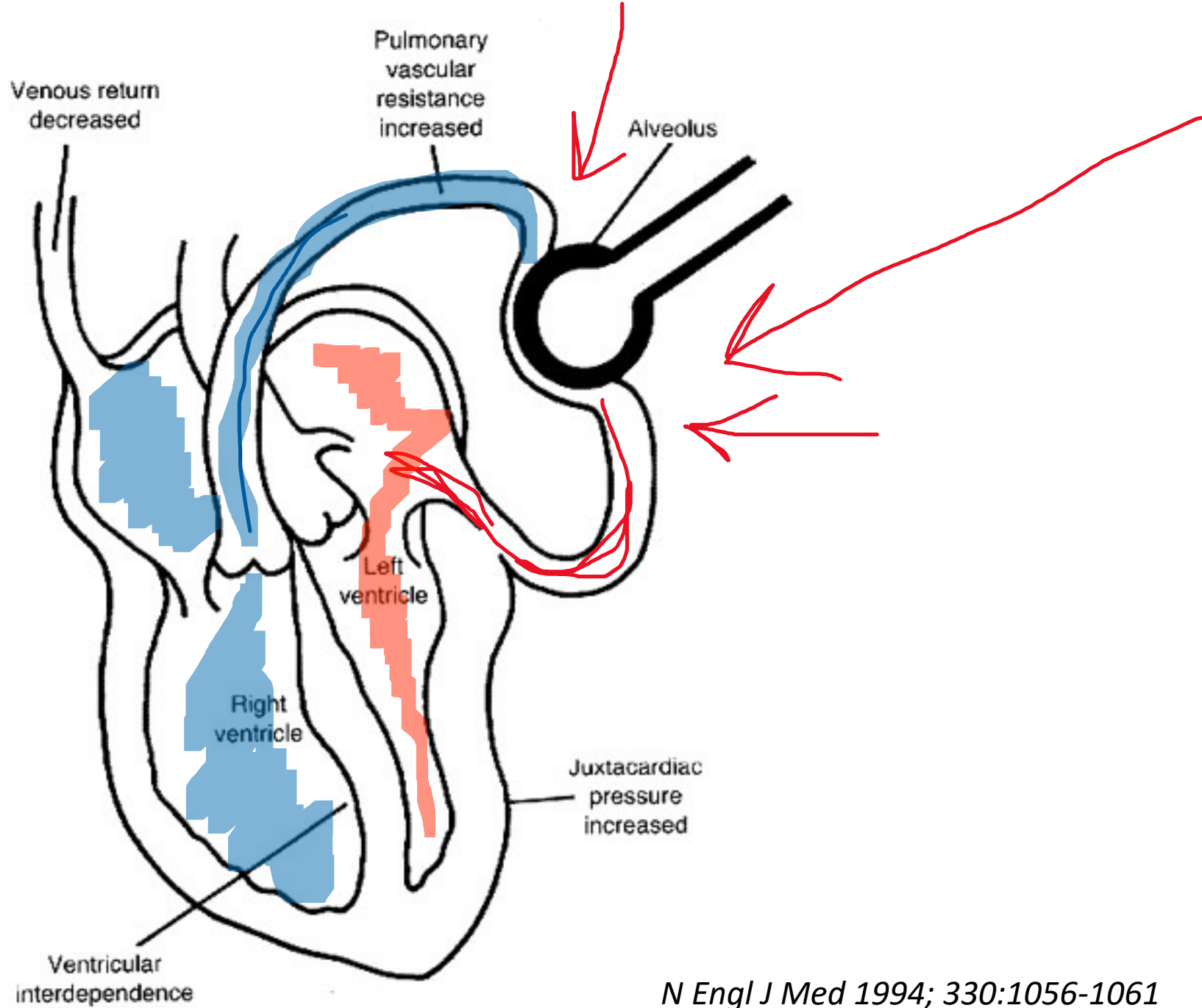
CONCLUSIONS Lung disease is independently associated with cardiovascular diseases, particularly IHD and HF, which contribute significantly to all-cause mortality. However, patients with lung disease are less likely to receive coronary revascularization. (J Am Coll Cardiol 2019;73:2166-77) © 2019 by the American College of Cardiology Foundation.



Patients with combination
cardiovascular and
pulmonary disease are
complex

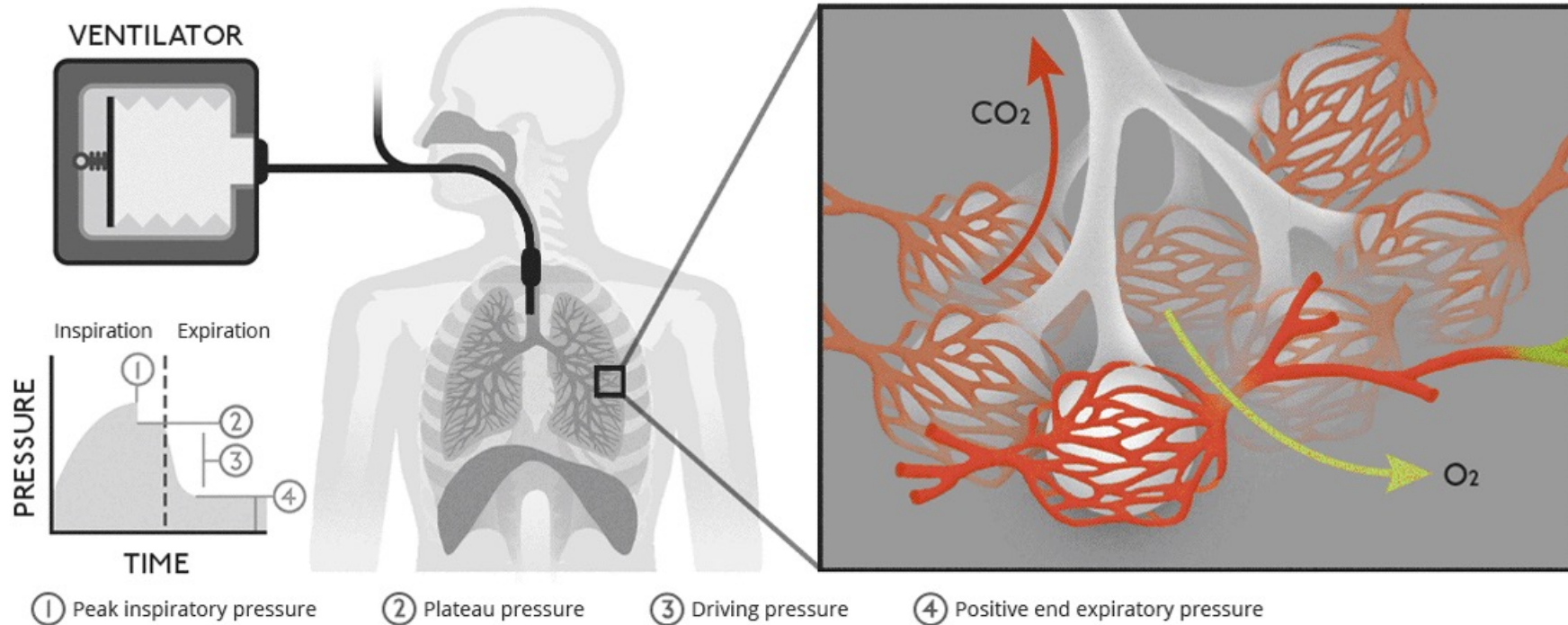








Too much PEEP?



PHYSIOLOGICAL STUDIES OF THE EFFECTS OF INTERMITTENT POSITIVE PRESSURE BREATHING ON CARDIAC OUTPUT IN MAN^{1, 2}

ANDRE COURNAUD, HURLEY L. MOTLEY³, LARS WERKO⁴
AND DICKINSON W. RICHARDS, JR.

*From the Department of Medicine, Columbia University, and the Chest and Medical Services of
the Columbia University Division, Bellevue Hospital, New York, New York*

Received for publication August 18, 1947

The effects of three types of intermittent positive pressure breathing, as differentiated by the shape of the mask pressure curve, have been correlated with the changes in cardiac output observed in 33 experiments on 29 human subjects.

The three types of mask pressure curves were as follows: type I, symmetrical with gradual increasing and decreasing slope, expiratory time approximately the same as inspiratory and the end expiratory pressure above atmospheric; type II, asymmetrical with rapidly increasing pressure during inspiration and rapidly dropping during expiration, long inspiratory and short expiratory time intervals and the end expiratory pressure above atmospheric; and type III, asymmetrical with gradually increasing pressure during inspiration and suddenly dropping early in expiration to atmospheric and expiratory time equal to or exceeding inspiratory.

Cardiac output was decreased more or less in proportion to the increase in mean mask pressure with the first and second type curves. There was no decrease in cardiac output with the third type curve.

The net filling pressure of the right ventricle decreased during the phase of increasing mask pressure and increased during the phase of decreasing mask pressure with all types of curves. The curve of net filling pressure of the right.



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Cardioselective beta-blockers for reversible airway disease

✉ [Shelley R Salpeter](#), [Thomas M Ormiston](#), [Edwin E Salpeter](#), [Richard Wood-Baker](#) [Authors' declarations of interest](#)

Version published: 21 October 2002 [Version history](#)

Cardioselective beta-blockers given in mild to moderate reversible airway disease or COPD do not produce adverse respiratory effects. Given their demonstrated benefit in conditions such as heart failure, cardiac arrhythmias and hypertension, these agents should not be withheld from such patients. Long-term safety still needs to be established.



ERS

EUROPEAN
RESPIRATORY
SOCIETY

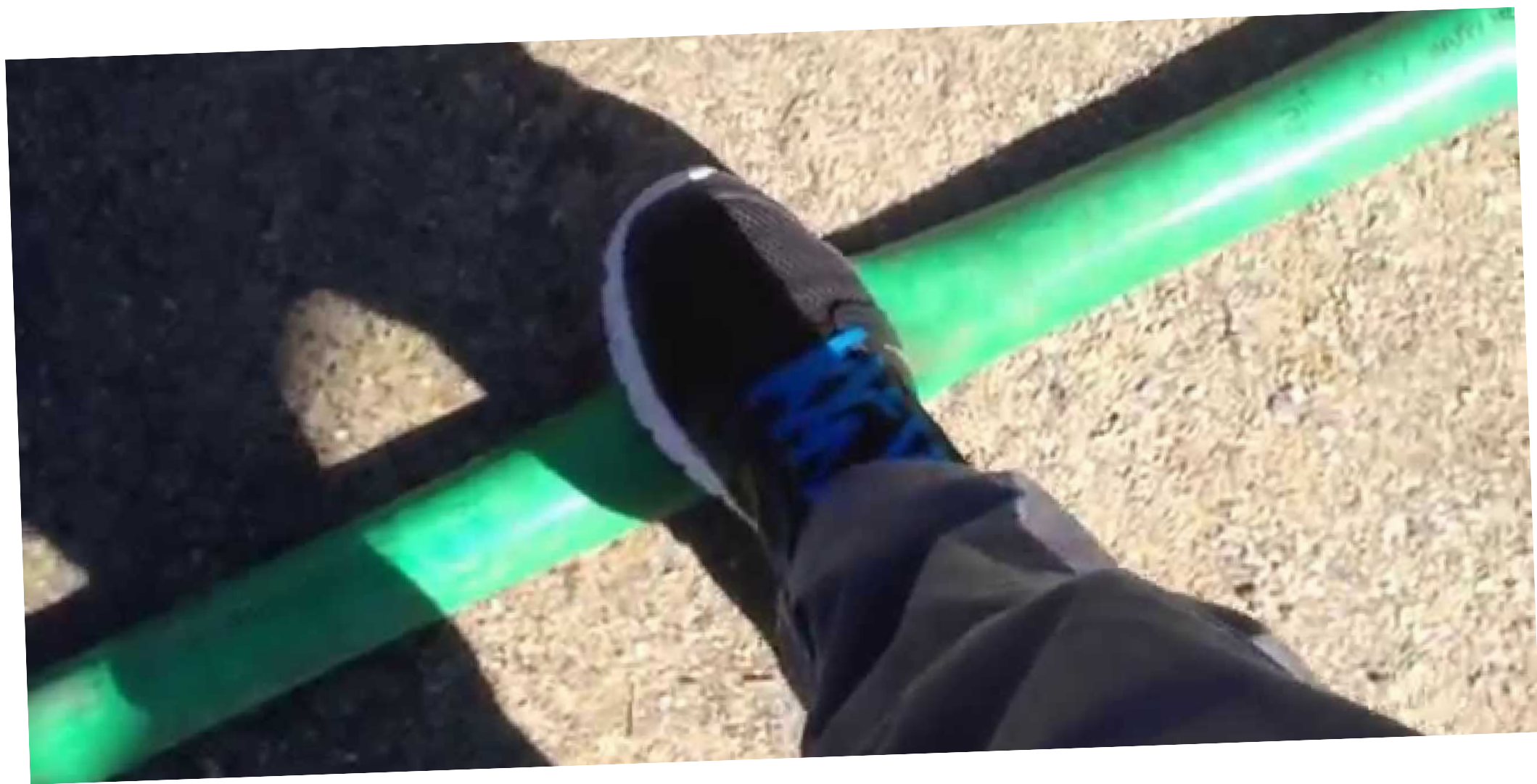
every breath counts

The safety of cardioselective β_1 -blockers in asthma: literature review and search of global pharmacovigilance safety reports

[Miriam Bennett](#),¹ [Catherina L. Chang](#),¹ [Michael Tatley](#),² [Ruth Savage](#),^{2,3,4} and [Robert J. Hancox](#)^{1,5}

“... no published reports of cardioselective β_1 -blockers causing asthma death. Observational data suggest that cardioselective β_1 -blocker use is not associated with increased asthma exacerbation.





Mechanical Ventilation “... independently affects the key determinants of cardiovascular performance: atrial filling or preload; the impedance to ventricular emptying or afterload; heart rate and myocardial contractility.

...Pressures remain greater than atmospheric pressure throughout the respiratory cycle.”



Cardiovascular effects of mechanical ventilation

Lara Shekerdemian, Desmond Bohn

Arch Dis Child. 1999 May;80(5):475-80

“**Positive-pressure ventilation** usually lowers cardiac output, primarily as a result of decreased venous return. Conversely, this form of ventilation can increase cardiac output in patients with impaired myocardial contractility because there is a decrease in left ventricular afterload due to increased intrathoracic pressure.”



The NEW ENGLAND
JOURNAL of MEDICINE

REVIEW ARTICLE CURRENT CONCEPTS

Mechanical Ventilation

Martin J. Tobin



CHEST[®]

Official publication of the American College of Chest Physicians

Cardiovascular Issues in Respiratory Care*

Michael R. Pinsky

Chest 2005;128;592S-597S
DOI 10.1378/chest.128.5_suppl_2.592S

“...positive pressure ventilation increases intrathoracic pressure. Increases in intrathoracic pressure decrease LV afterload... **In patients with hypervolemic heart failure**, this afterload reducing effect can result in **improved LV ejection fraction, increased cardiac output, and reduced myocardial oxygen demand**

Recommended ventilator settings for patients with heart failure with reduced ejection fraction.

Setting	Recommended Initial Ventilator Settings
PEEP	Titrate to adequate oxygenation, work of breathing, and hemodynamics. Recommend preferential use of PEEP for oxygenation if hemodynamically beneficial.
Tidal Volume	8 cc/kg predicted body weight
FiO ₂	Titrate to SpO ₂ 90%–94%. Recommend rapid de-escalation of FiO ₂ after intubation.
Plateau pressure	Maintain below 30 cm H ₂ O. Consider alternative diagnoses if plateau rises above 30.
Respiratory rate	In conjunction with tidal volume, titrate to maintain normal pH (7.35–7.45) and pCO ₂ (35–45 mm Hg)
Inattention to changing needs	Provide the minimal ventilator support to support physiologic stability. MV requires frequent re-evaluation and titration.

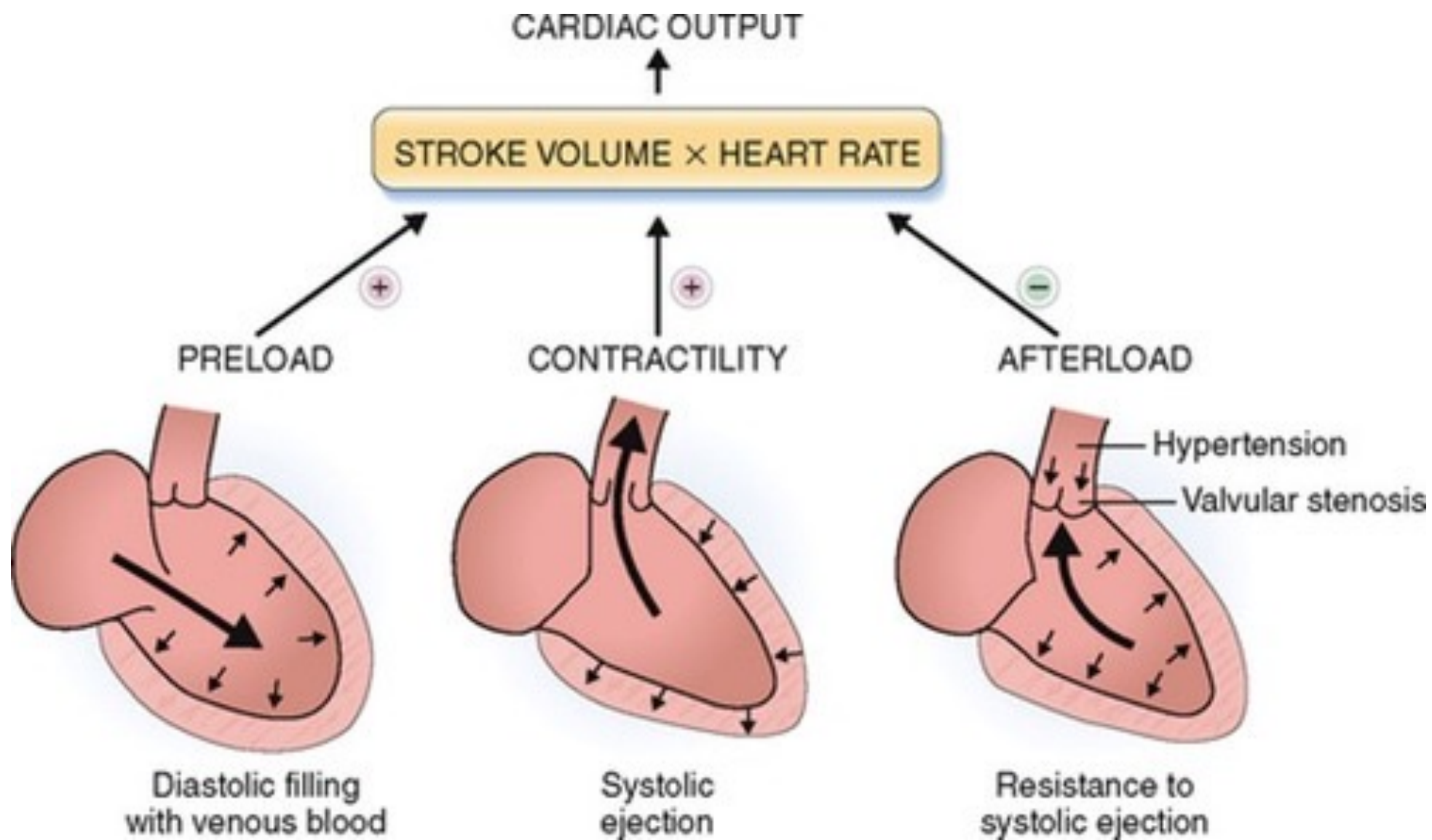
Think differently...

Wait... What exactly is heart failure?

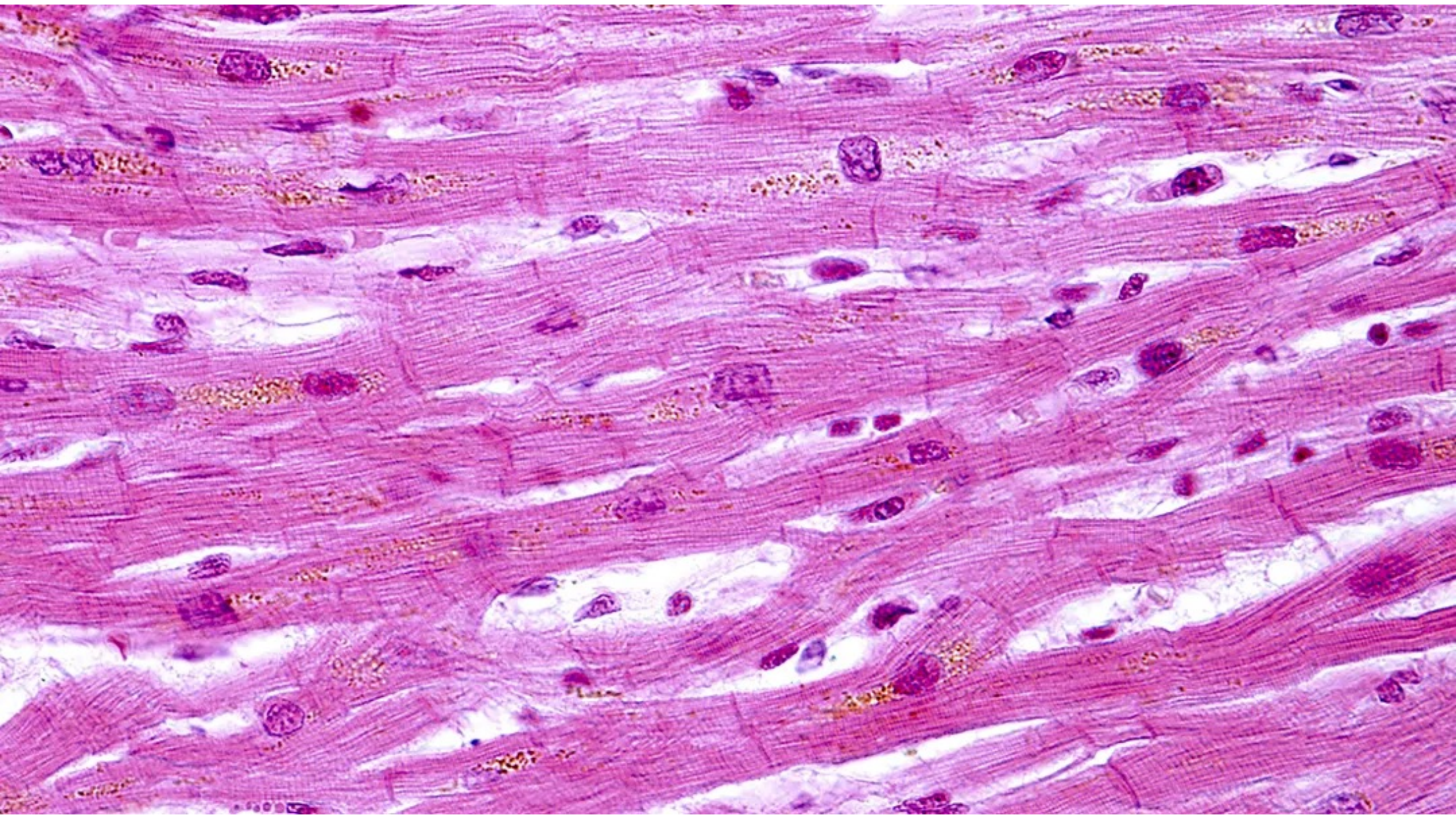
Universal Definition of Heart Failure Proposed

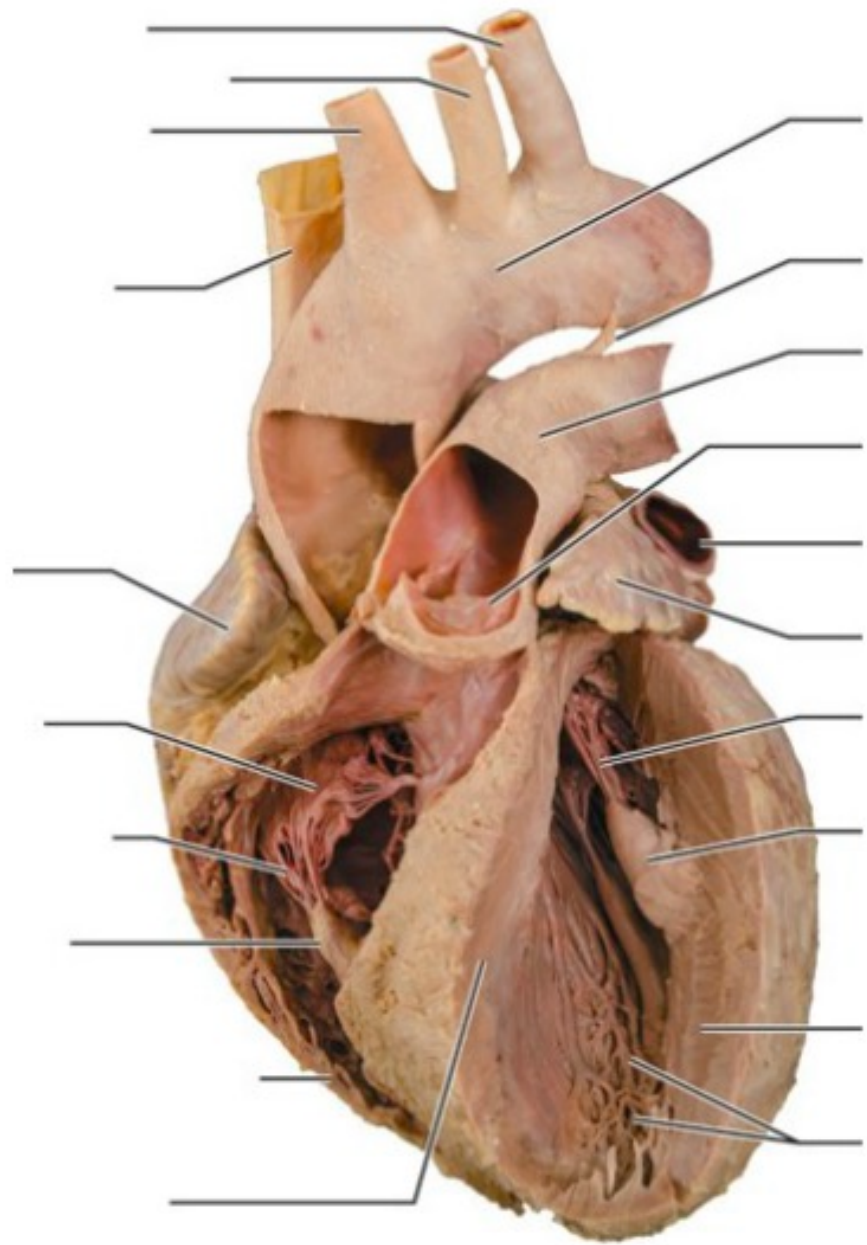
— U.S., European, and Japanese societies seek to standardize classifications

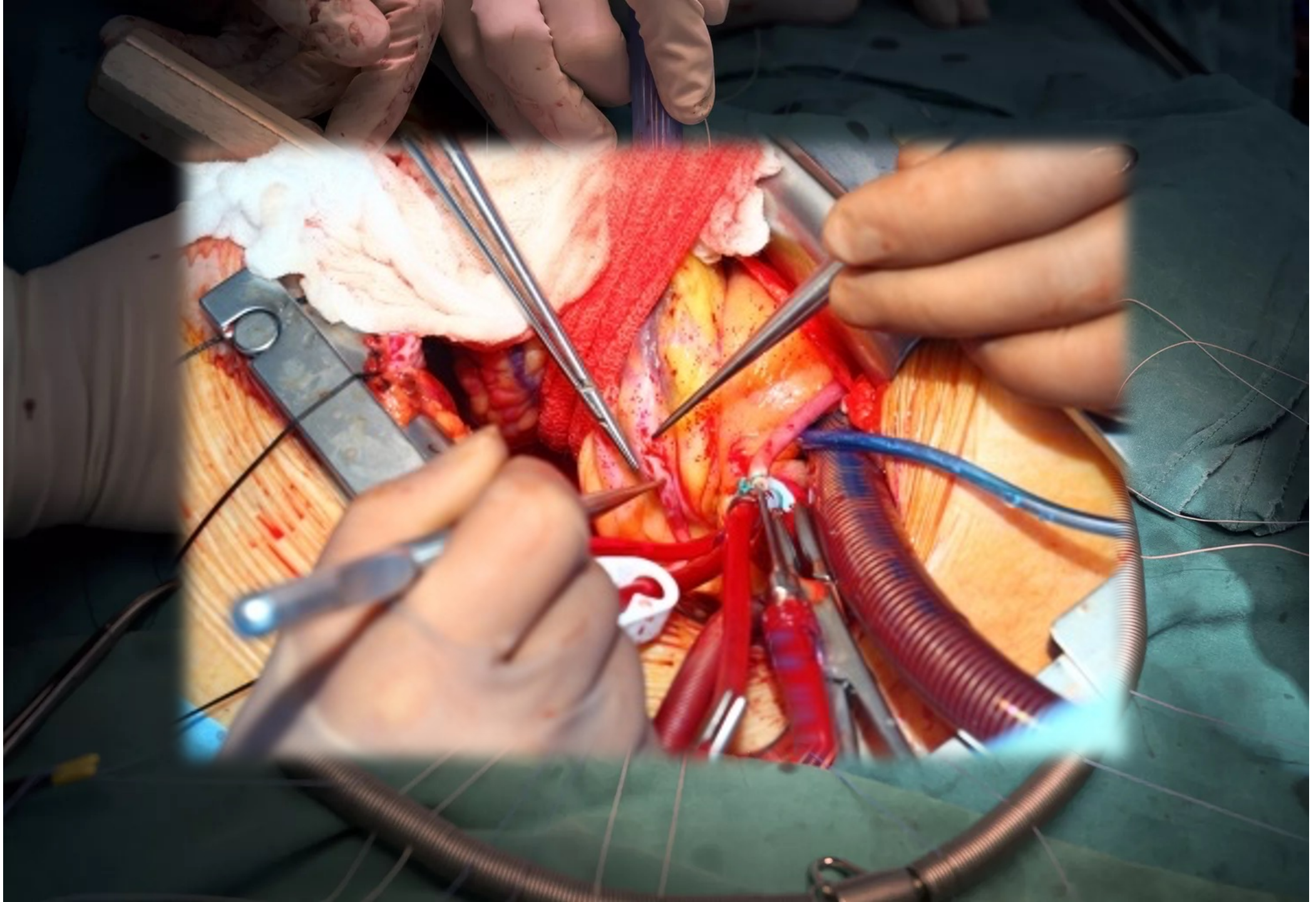
"HF is a clinical syndrome with symptoms and or signs caused by a structural and or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels and or objective evidence of pulmonary or systemic congestion," according to the consensus document from the Heart Failure Society of America (HFSA), European Society of Cardiology, and Japanese Heart Failure Society.



All cardiovascular conditions and/or diseases, and every single cardiac medication will, to some degree, alter **preload**, **afterload**, or **contractility**







Class	Patient Symptoms
--------------	-------------------------

I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea (shortness of breath).
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II	Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea (shortness of breath).
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III	Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, or dyspnea.
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IV	Unable to carry on any physical activity without discomfort. Symptoms of heart failure at rest. If any physical activity is undertaken, discomfort increases.
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Class	Objective Assessment
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A	No objective evidence of cardiovascular disease. No symptoms and no limitation in ordinary physical activity.
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B	Objective evidence of minimal cardiovascular disease. Mild symptoms and slight limitation during ordinary activity. Comfortable at rest.
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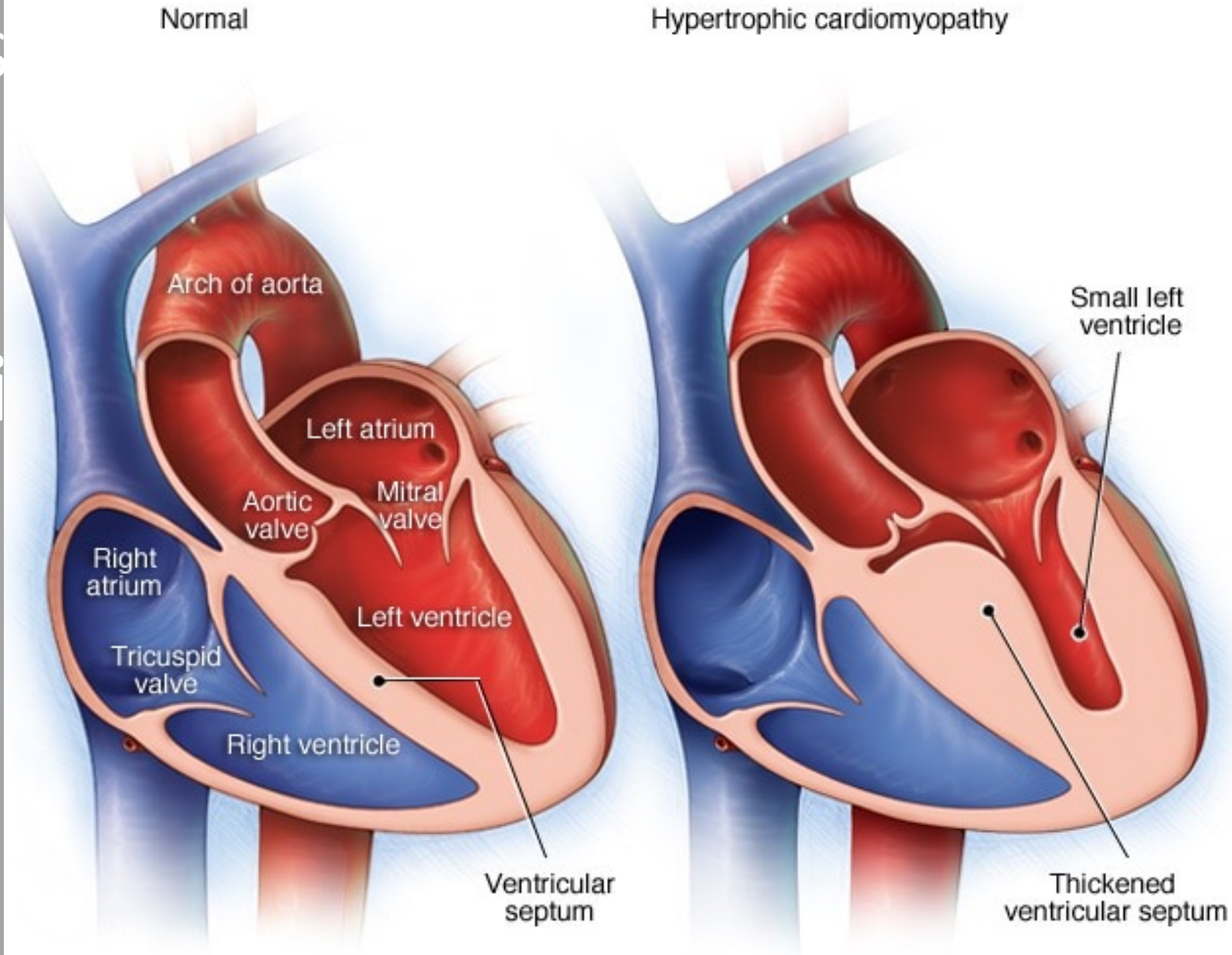
C	Objective evidence of moderately severe cardiovascular disease. Marked limitation in activity due to symptoms, even during less-than-ordinary activity. Comfortable only at rest.
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D	Objective evidence of severe cardiovascular disease. Severe limitations. Experiences symptoms even while at rest.
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Classifications *of* Heart Failure

“Conges

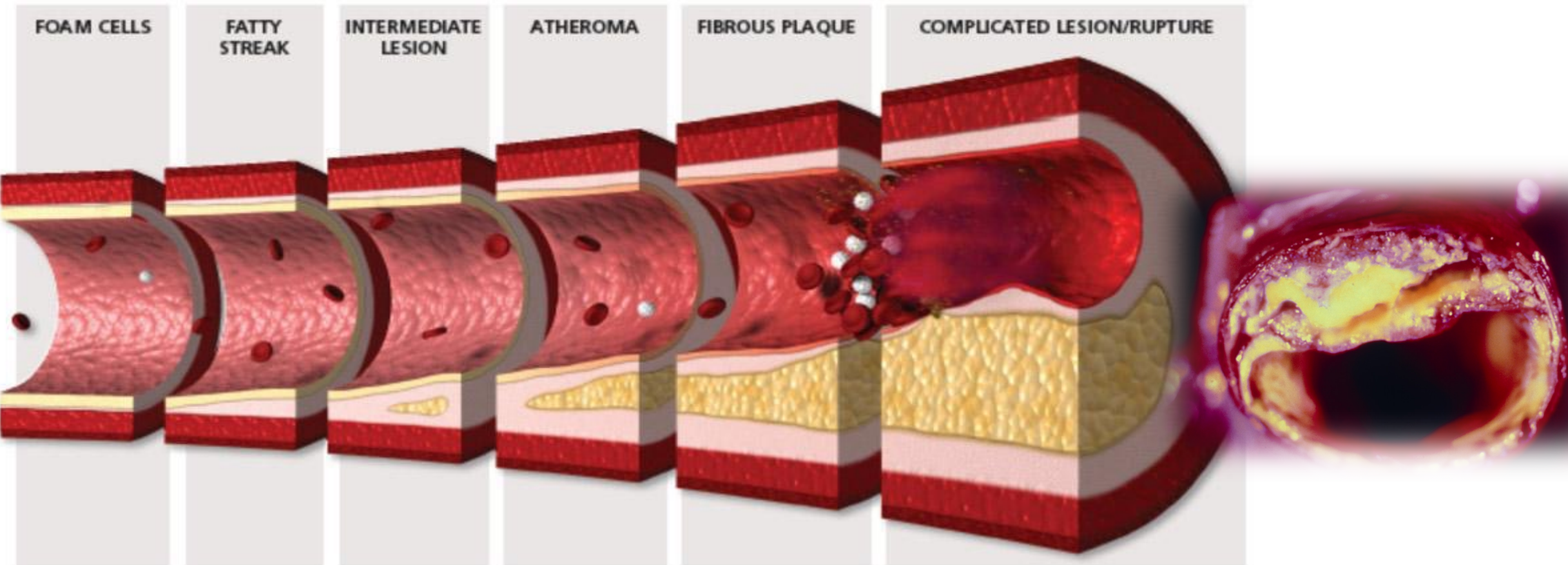
approxi



ration

weight

Coronary Atherosclerosis Timeline



Progressive Atherosclerosis

Heart Damage

Normal CRP, LpPLA₂ & MPO

High CRP, LpPLA₂ & Normal MPO

High CRP, LpPLA₂ & MPO

Growth mainly by lipid accumulation

Smooth muscle and collagen

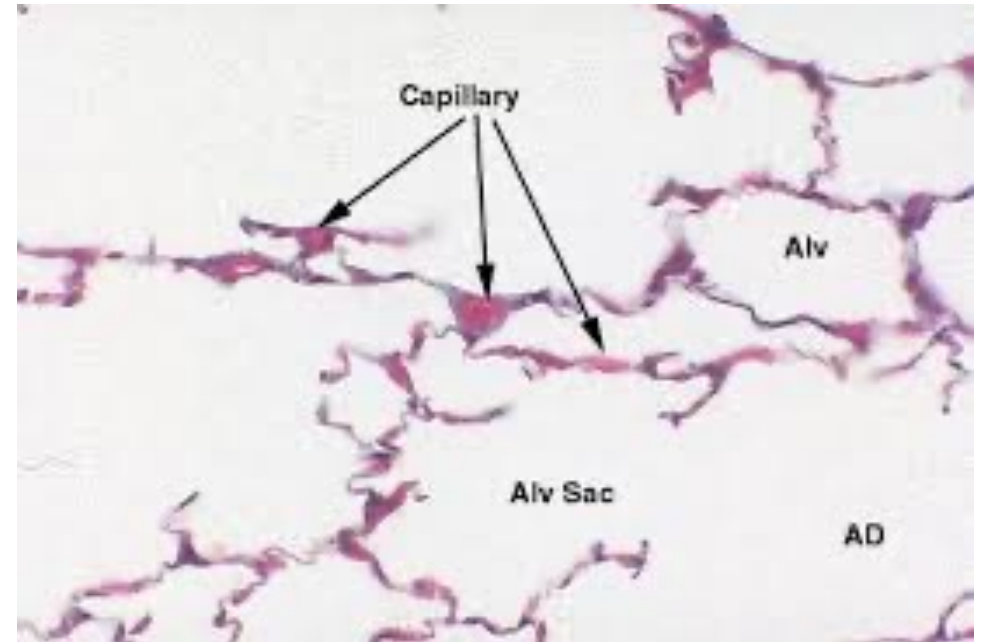
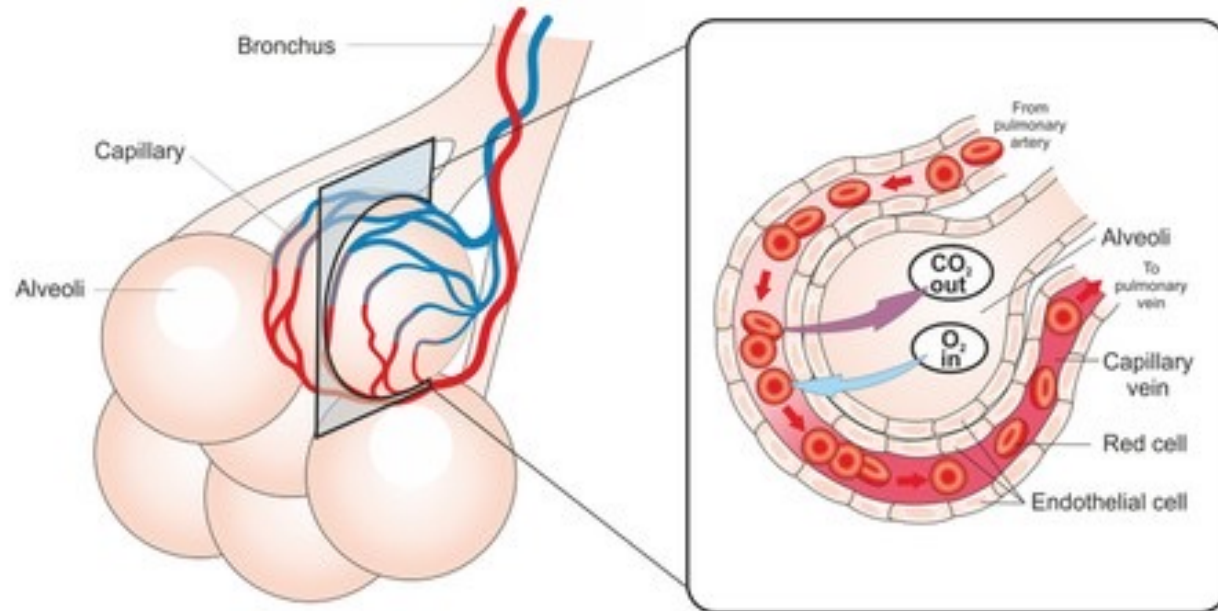
Thrombosis, hematoma

Transient Time?

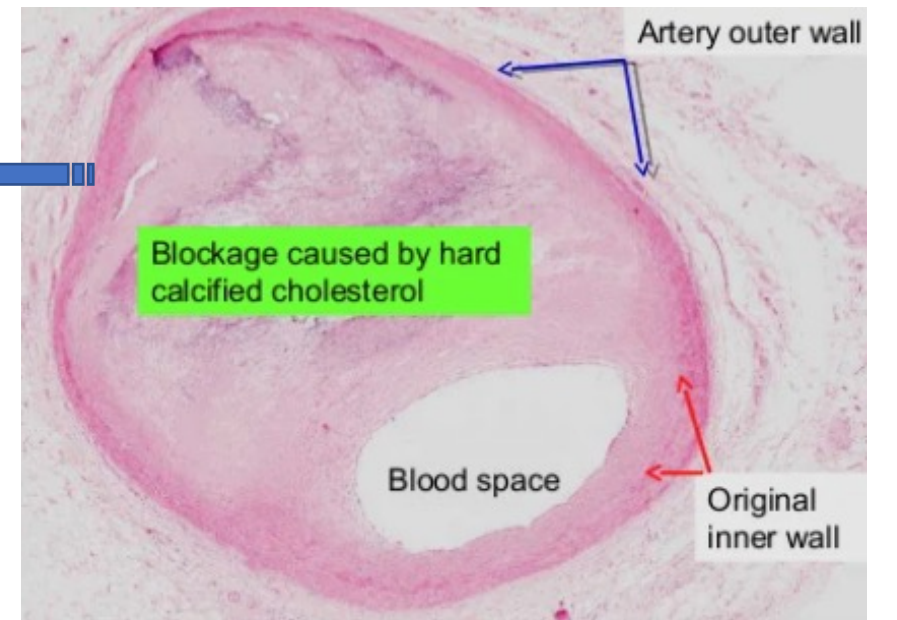
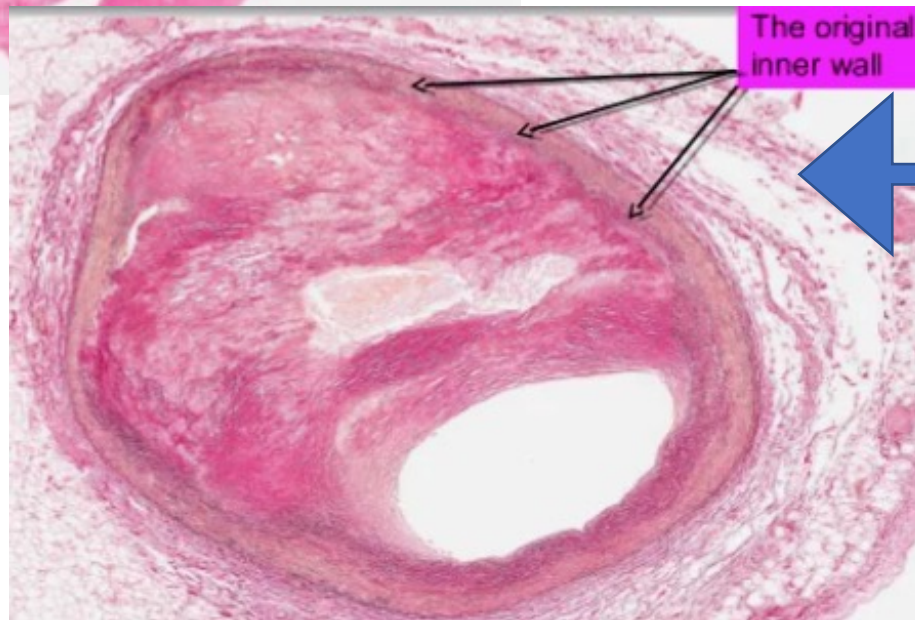
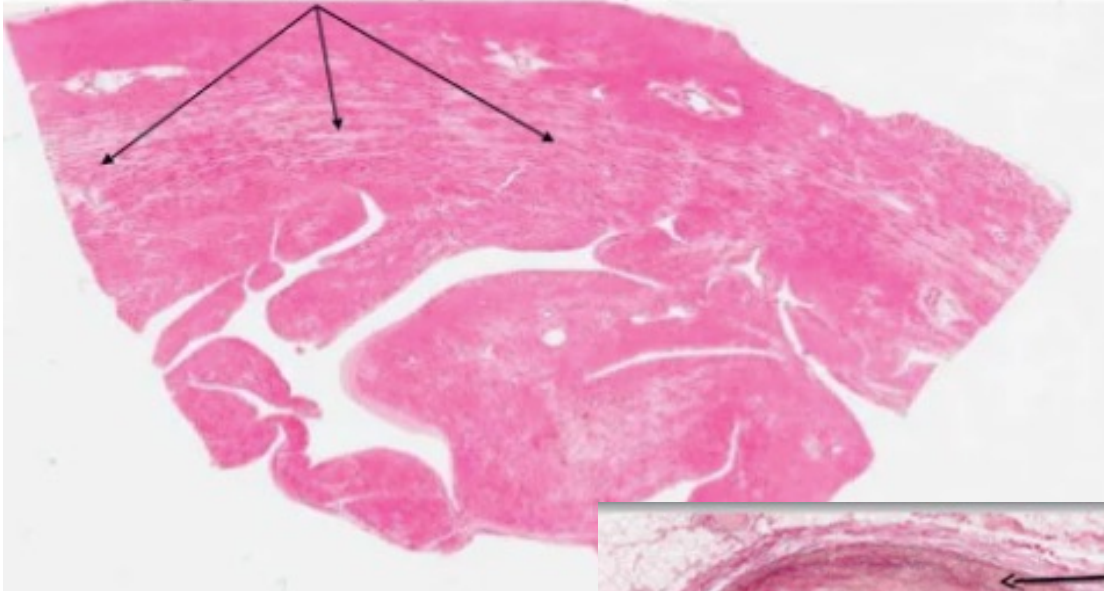
> *Am J Respir Crit Care Med.* 1995 Dec;152(6 Pt 1):2014-20. doi: 10.1164/ajrccm.152.6.8520770.

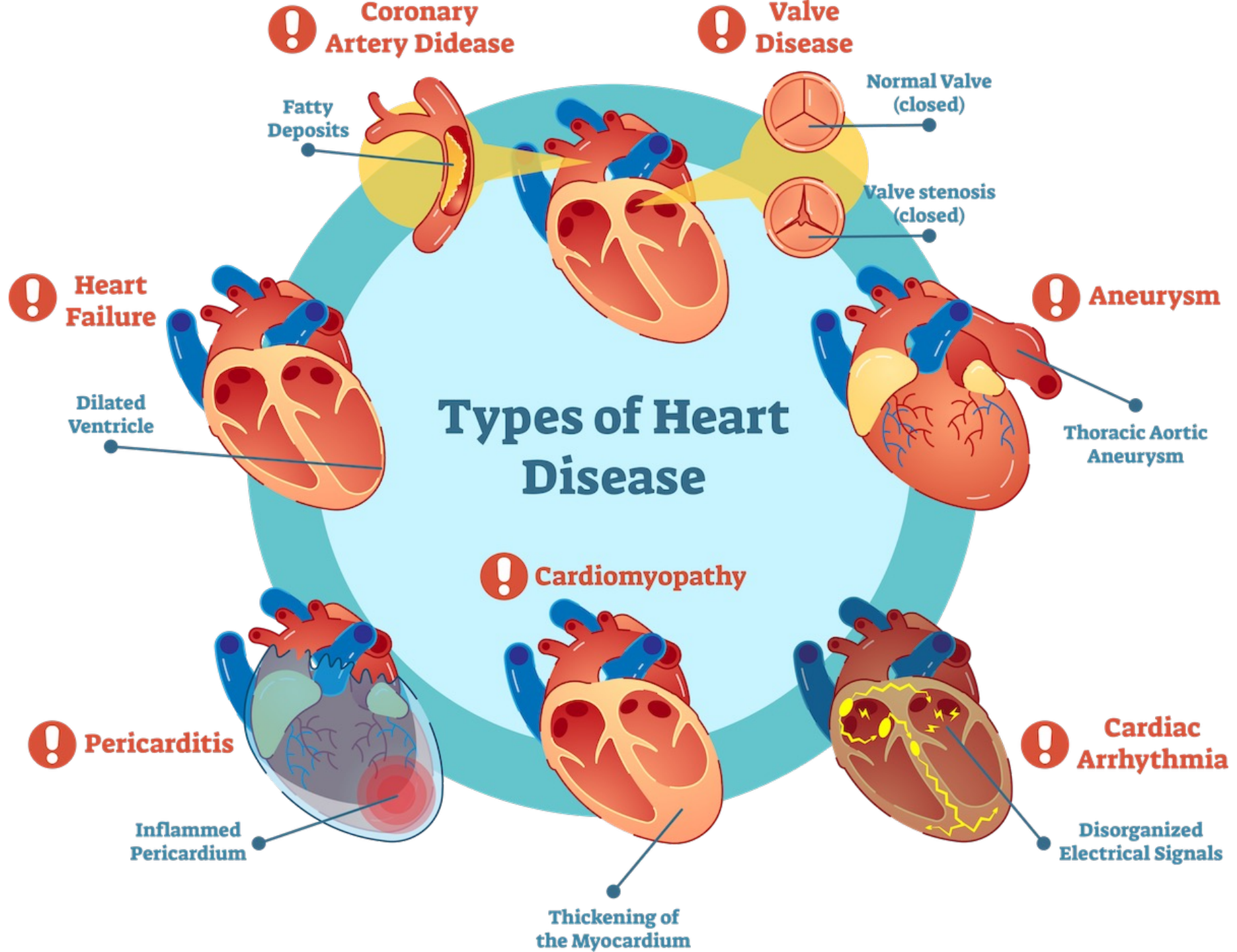
Distribution of pulmonary capillary transit times

R A Klocke ¹, H J Schünemann, B J Grant



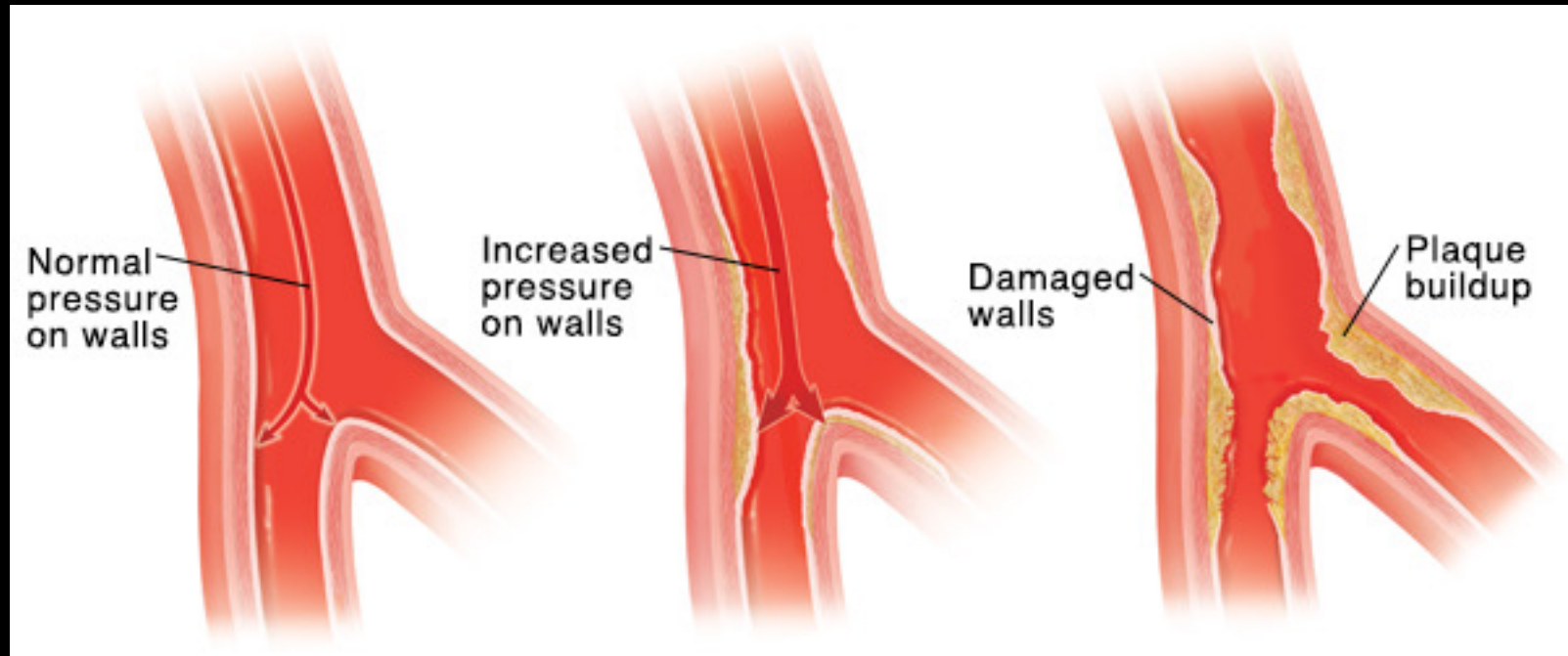
This shows pale bands of fibrosis in the diseased heart following an infarction (Heart Attack)

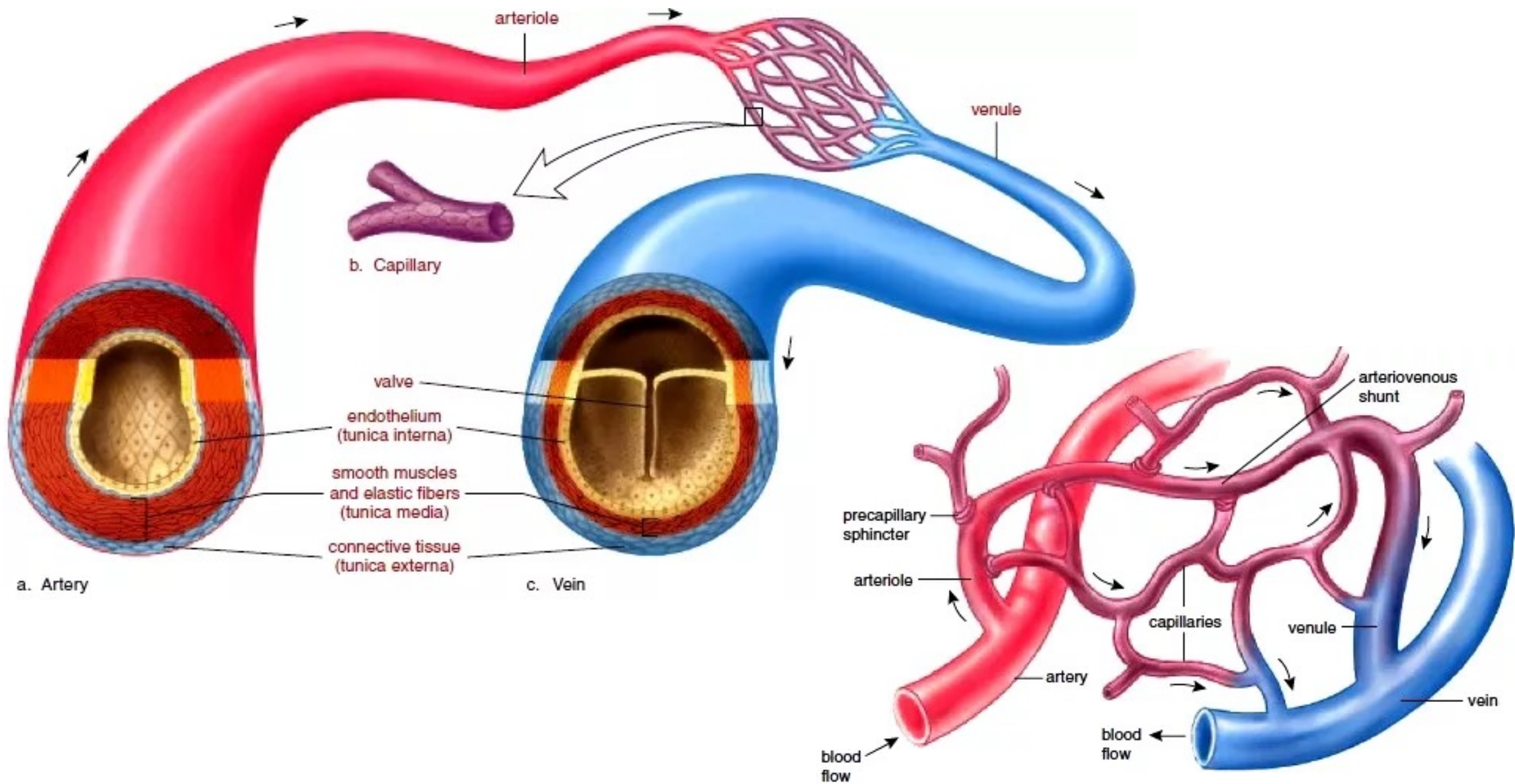




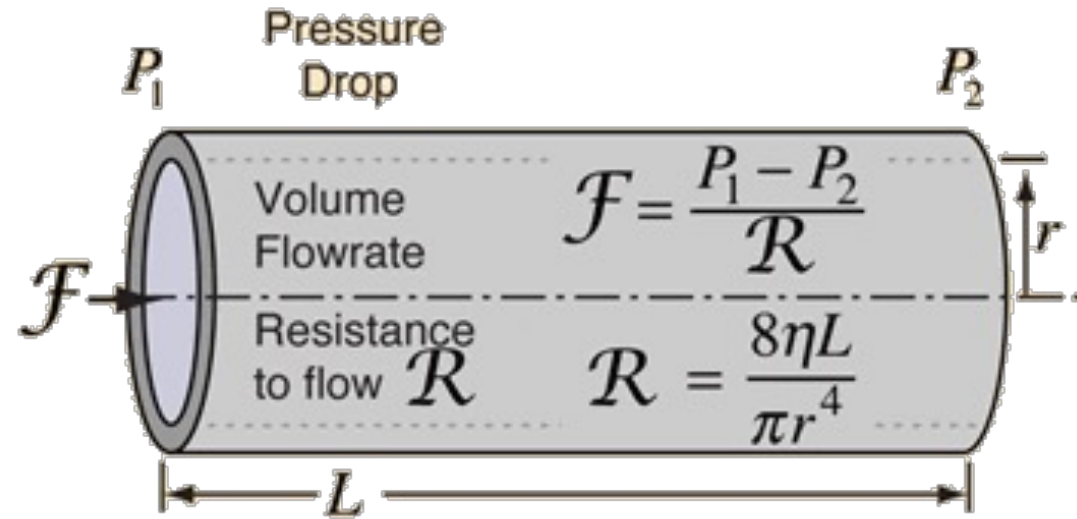
The global prevalence of **hypertension** is high, and among nonpregnant adults in the United States, treatment of **hypertension is the most common reason** for office visits and for the **use of chronic prescription medications**. In addition, **roughly one-half** of hypertensive individuals **do not have adequate blood pressure control**.

Blood pressure (BP) = Cardiac output (CO) x Systemic vascular resistance (SVR)





Poiseuille's Law

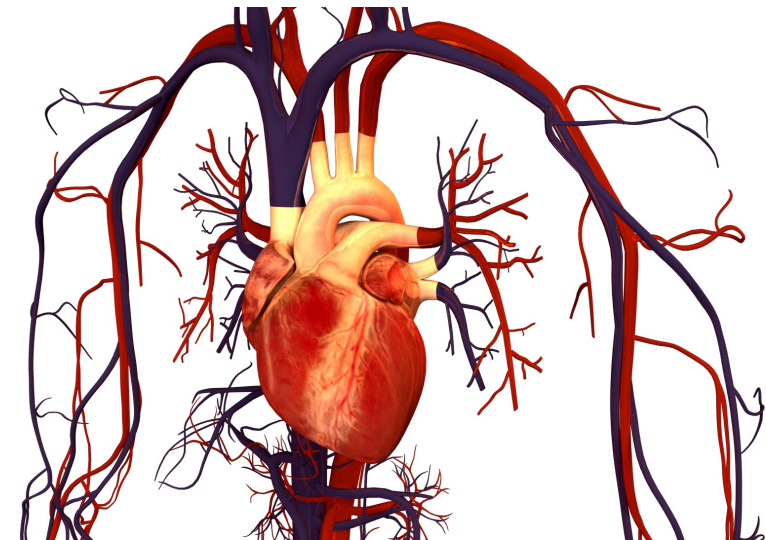


Q	Flow rate
P	Pressure
r	Radius
η	Fluid viscosity
l	Length of tubing

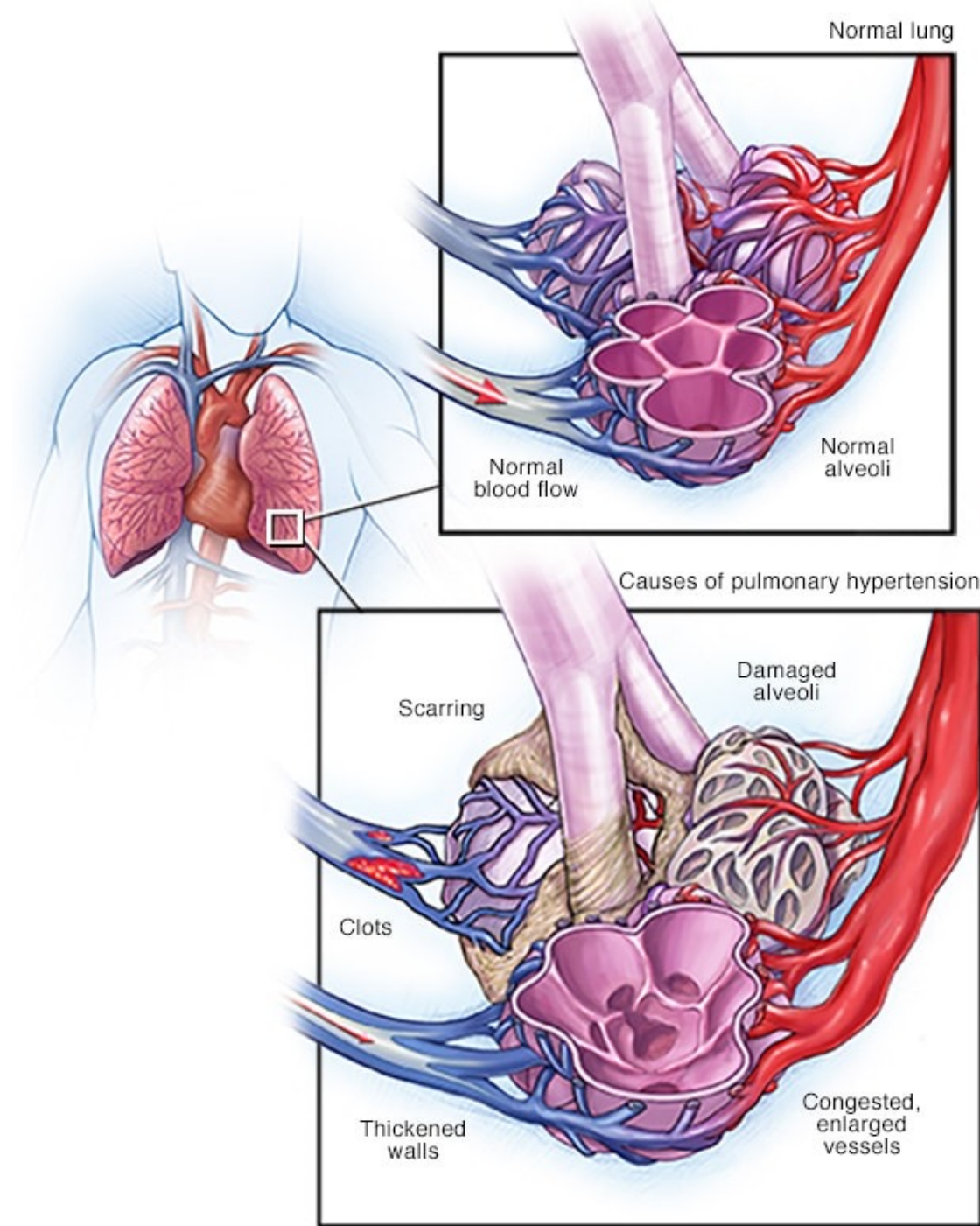
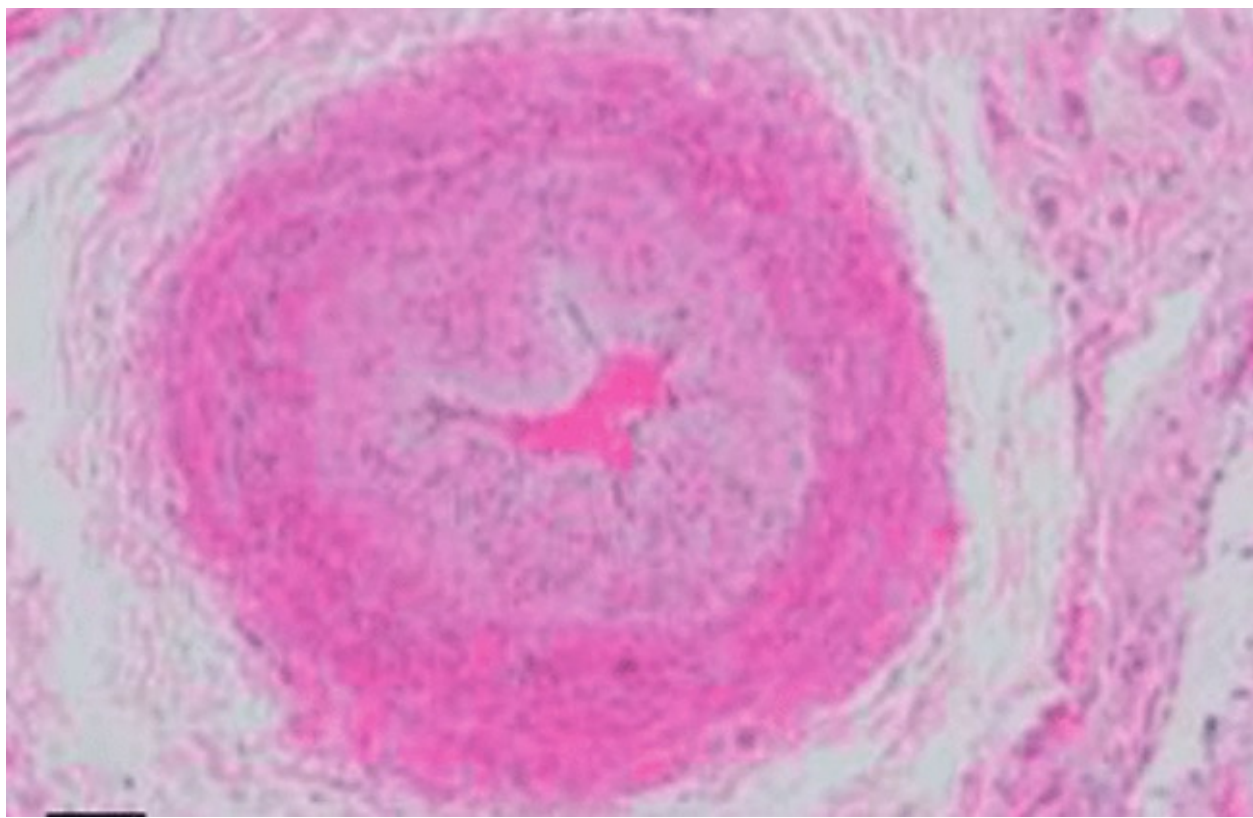
$$Q = \frac{\pi P r^4}{8\eta l}$$

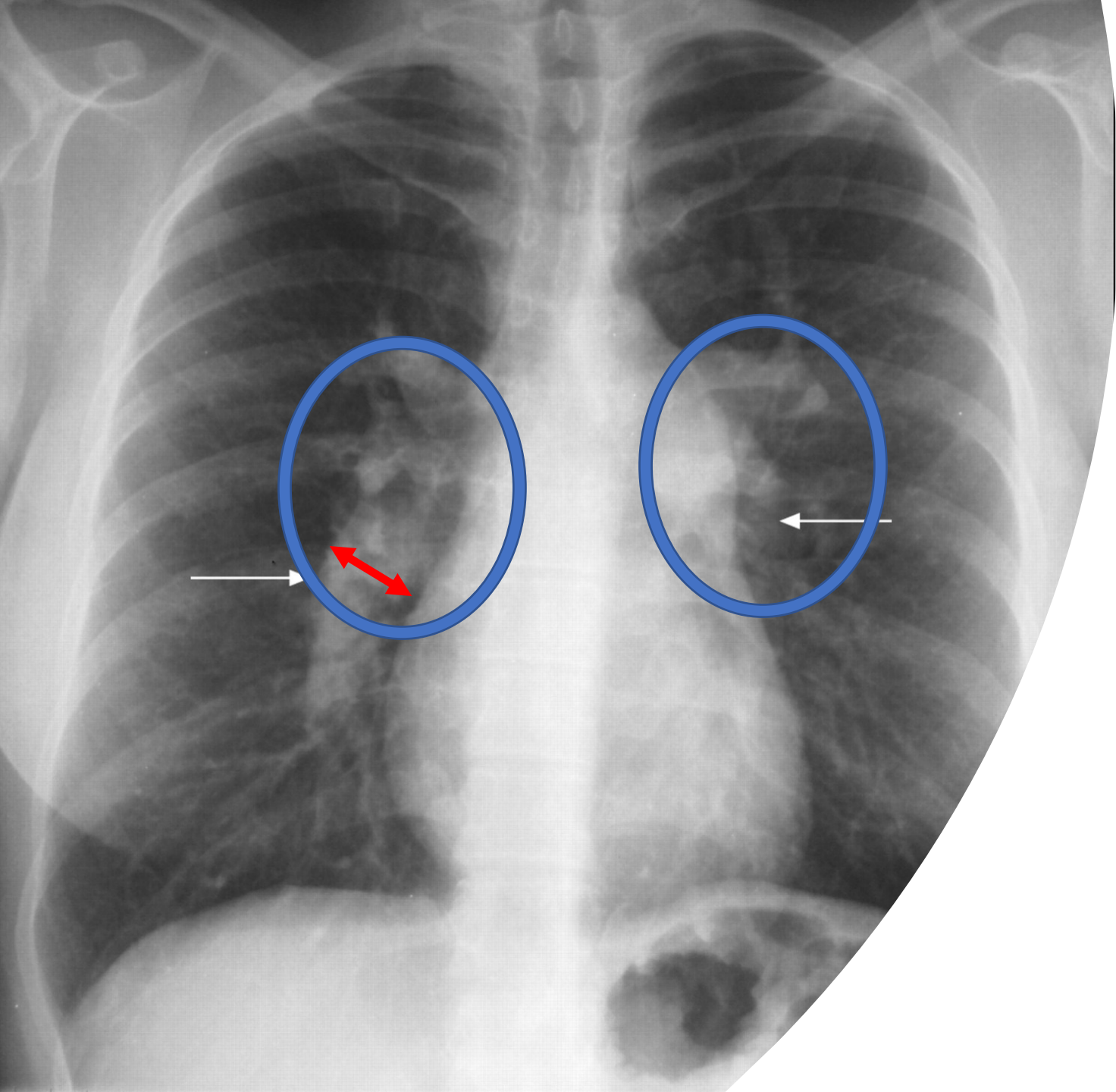
3 primary factors determine resistance to blood flow within a single vessel:

1. Diameter
2. Length
3. Viscosity.



What about pulmonary hypertension?

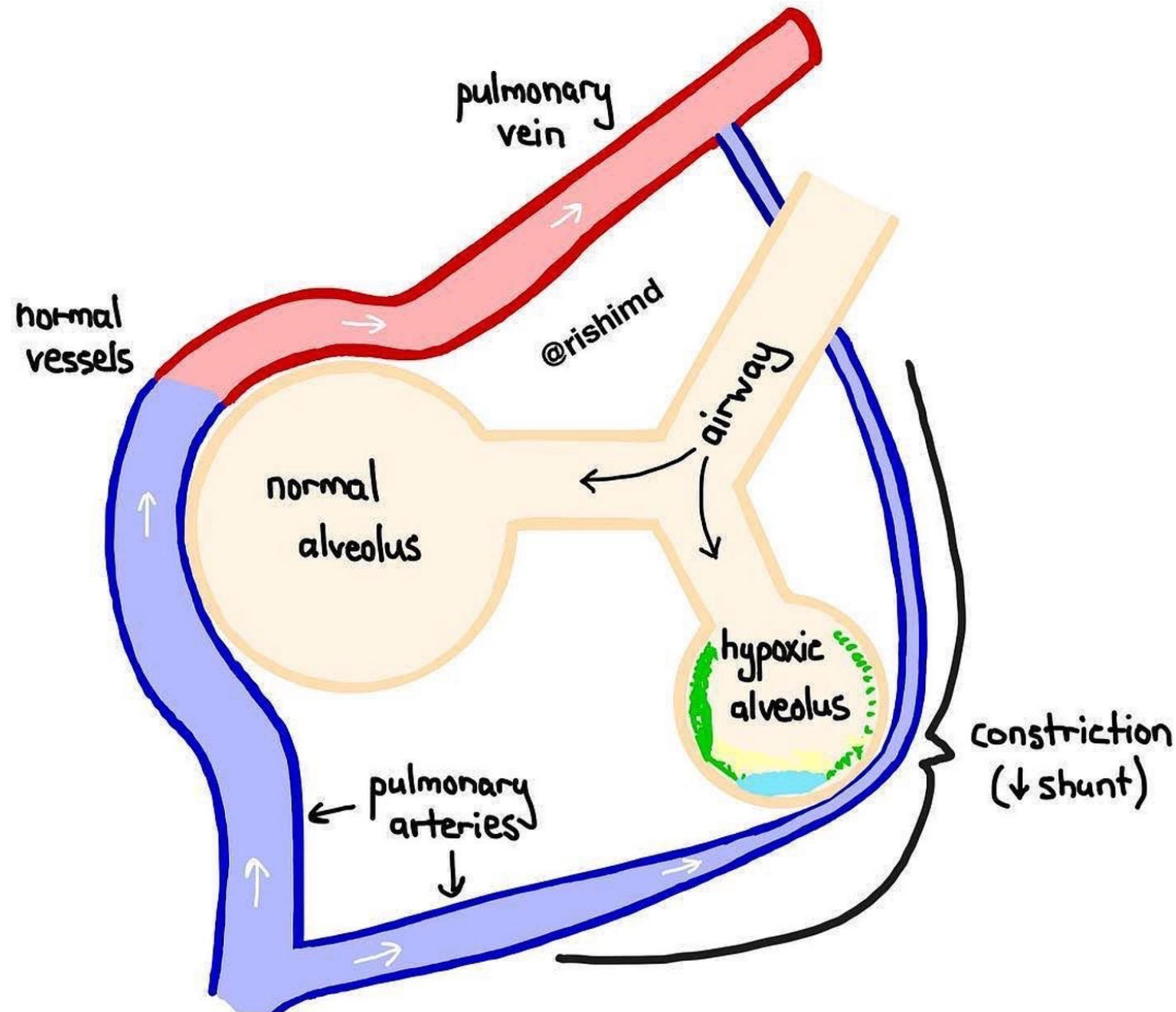


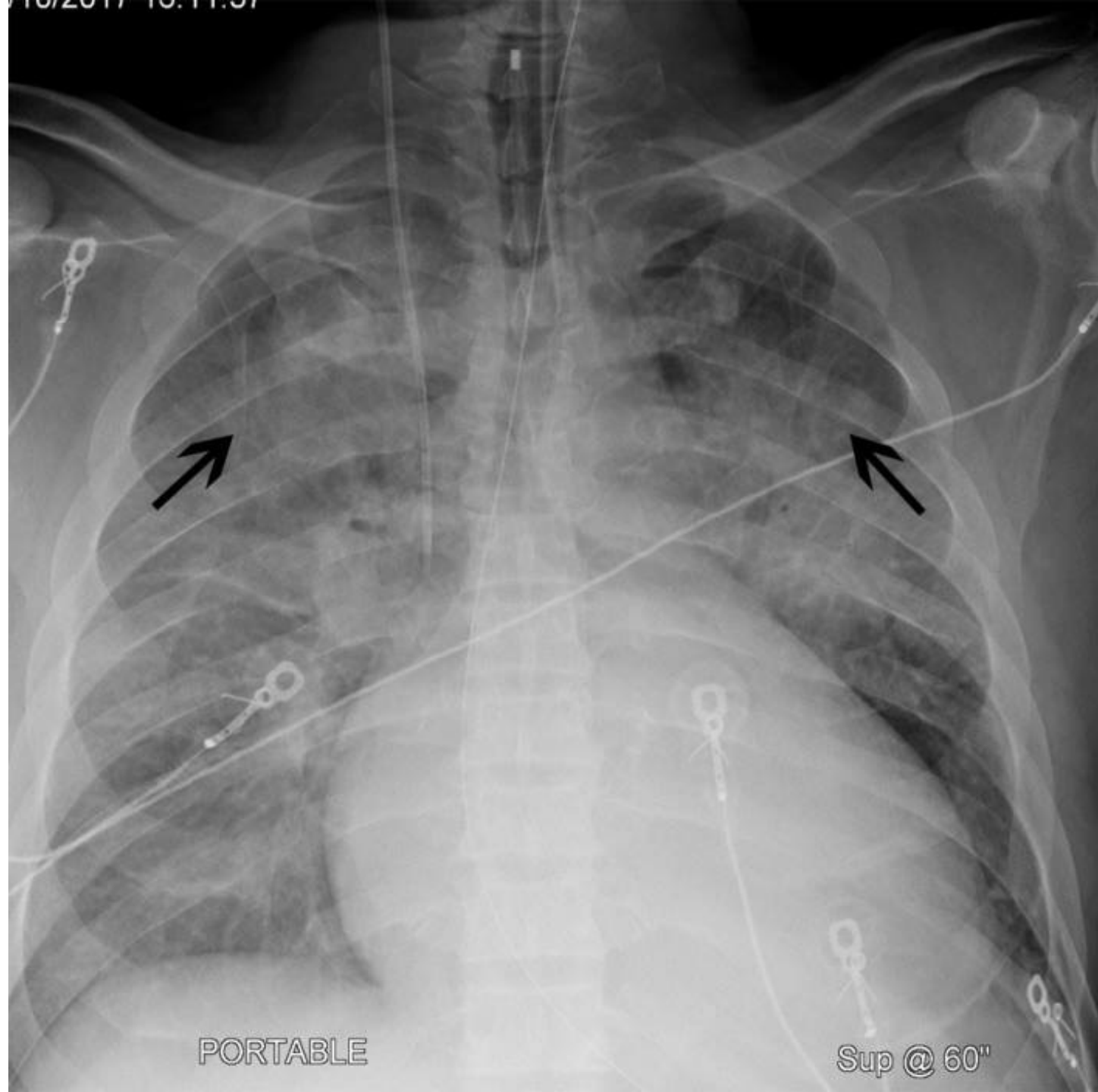


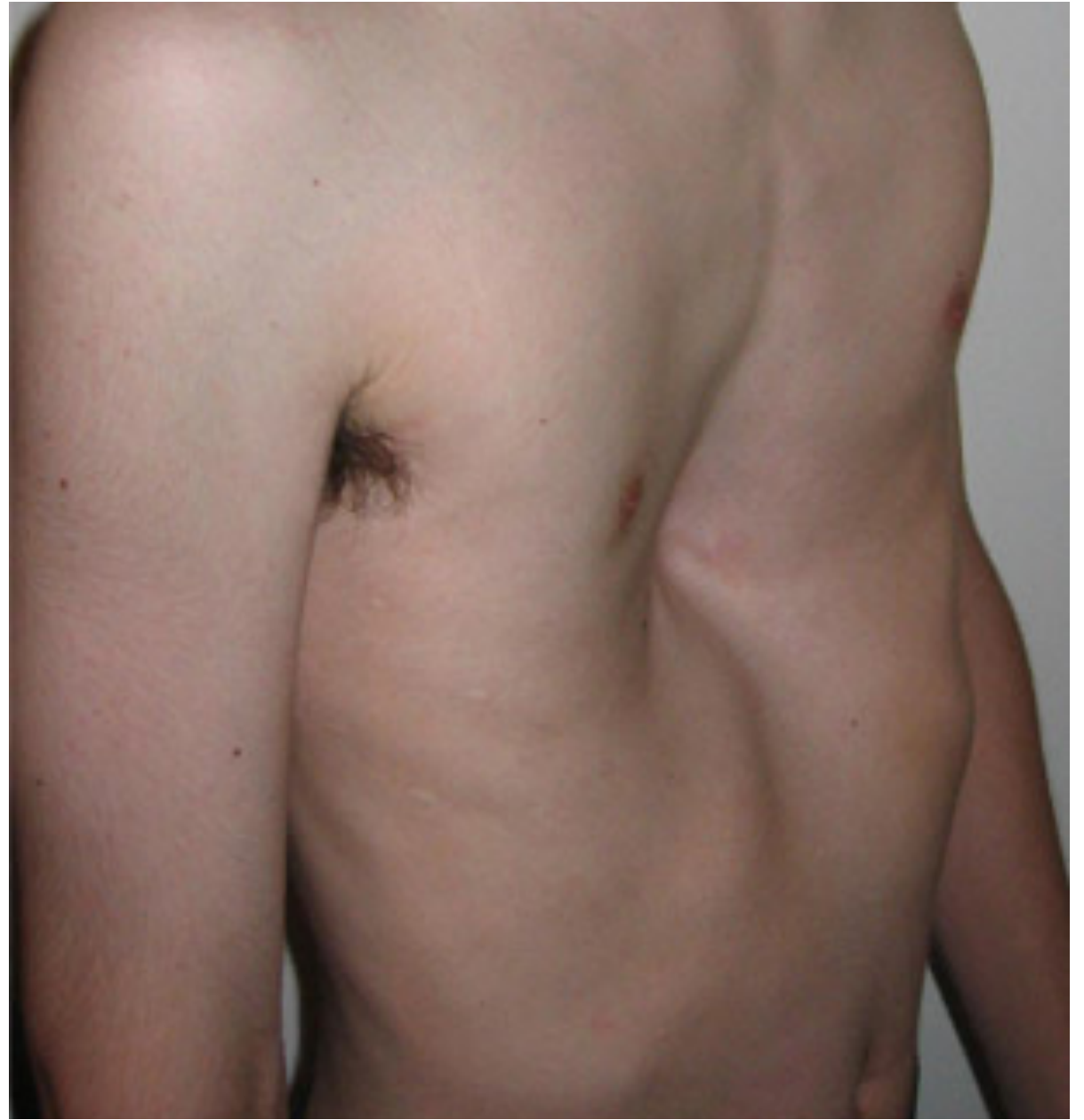
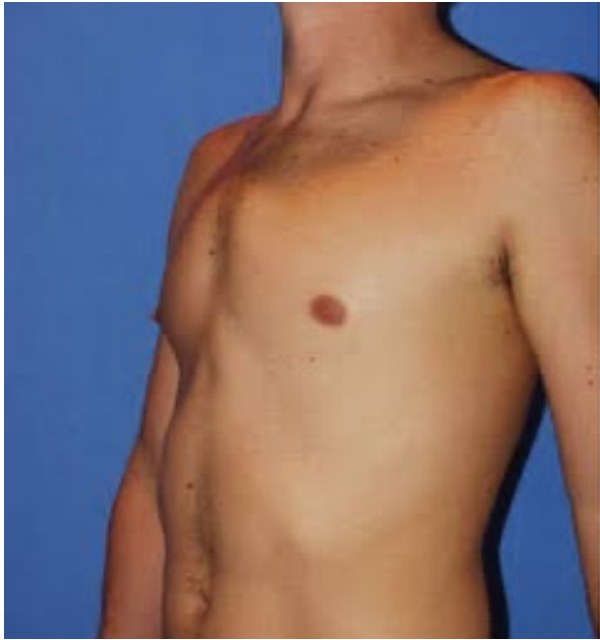
**Pulmonary
Hypertension**

Low O₂ at the lung causes pulmonary vessels to **constrict**, increasing pulmonary vascular resistance and right heart pressures

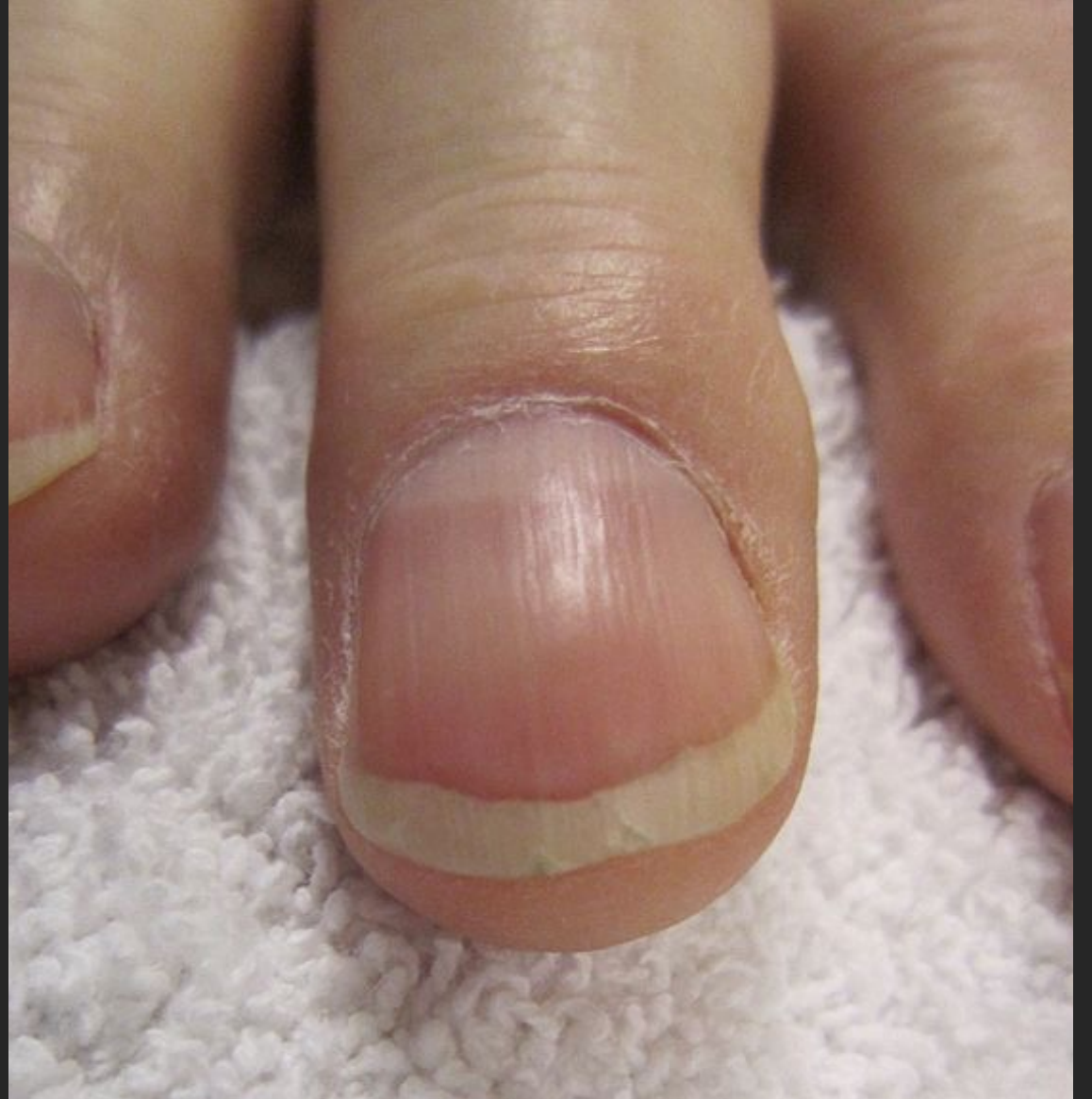
Friendly Reminder













Friendly
Reminder:
Manually
checking HR
is not illegal



All Respiratory Therapy, to
some degree, will alter
preload, afterload, or
contractility

“Case Study”

You are the only RT on the pediatric floors when you are notified of new orders, changing your 4-year-old female patient from 1.25mg Albuterol via HHN to 0.63 mg Xopenex.

After checking the Pyxis, you note no Xopenex is available. You contact the pharmacy and are told *only* 1.25mg Xopenex is available. It will be sent through the tubing system within the hour.



Xopenex[®] (levalbuterol HCl) Inhalation Solution, 0.31 mg*, 0.63 mg*,
1.25 mg*

*Potency expressed as levalbuterol



PRESCRIBING INFORMATION

- 208 4. Cardiovascular Effects: Xopenex Inhalation Solution, like all other beta-adrenergic
209 agonists, can produce a clinically significant cardiovascular effect in some patients, as
210 measured by pulse rate, blood pressure, and/or symptoms. Although such effects are
211 uncommon after administration of Xopenex Inhalation Solution at recommended doses,
212 if they occur, the drug may need to be discontinued. In addition, beta-agonists have been
213 reported to produce ECG changes, such as flattening of the T wave, prolongation of the
214 QTc interval, and ST segment depression. The clinical significance of these findings is
215 unknown. Therefore, Xopenex Inhalation Solution, like all sympathomimetic amines,
216 should be used with caution in patients with cardiovascular disorders, especially
217 coronary insufficiency, cardiac arrhythmias, and hypertension.

American Journal of Respiratory and Critical Care Medicine

Particulate Matter and Cardiovascular Risk in Adults with Chronic Obstructive Pulmonary Disease

Stacey E. Alexeeff¹, Kamala Deosaransingh¹, Noelle S. Liao¹, Stephen K. Van Den Eeden¹, Joel Schwartz², and Stephen Sidney¹

¹Kaiser Permanente Division of Research, Kaiser Permanente, Oakland, California; and ²Harvard T.H. Chan School of Public Health, Harvard University, Boston, Massachusetts

Abstract

Rationale: People with chronic obstructive pulmonary disease (COPD) have an increased risk of cardiovascular disease and may be more susceptible to air pollution exposure. However, no study has examined the association between long-term fine particulate matter exposure ($\leq 2.5 \mu\text{m}$ in aerodynamic diameter) and risk of cardiovascular events in this potentially vulnerable population.

Objectives: To estimate the association between long-term fine particulate matter and risk of cardiovascular events among adults with COPD.

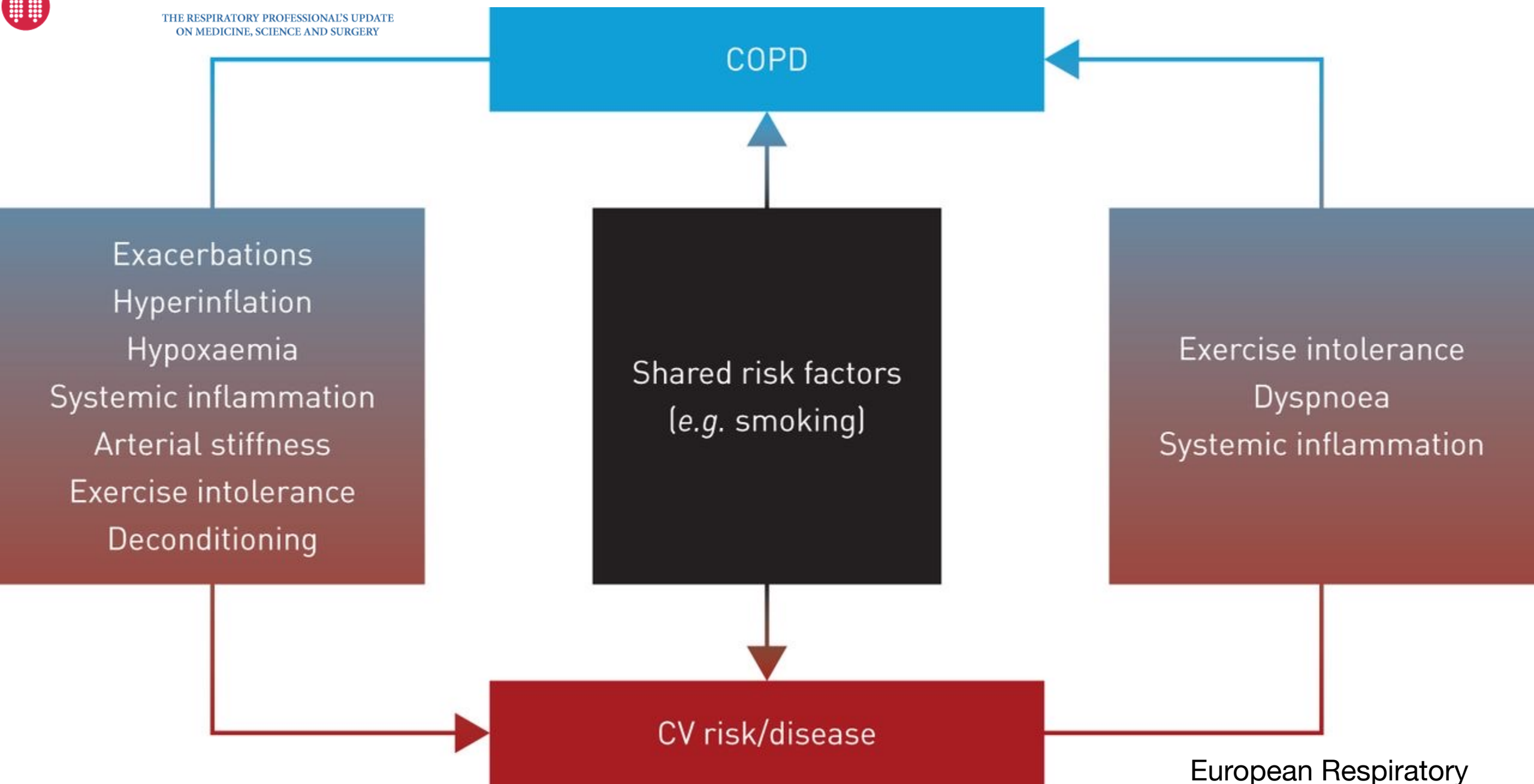
Methods: This retrospective cohort study included 169,714 adults with COPD who were members of the Kaiser Permanente Northern California health plan during 2007–2016. Electronic health record data were linked to 1 km modeled particulate matter $\leq 2.5 \mu\text{m}$ in aerodynamic diameter exposure estimates. We fit Cox proportional hazard models, adjusting for age, sex, race/ethnicity, calendar year,

smoking, body mass index, comorbidities, medications, and socioeconomic status. In low exposure analyses, we examined effects below the current regulation limit ($12 \mu\text{g}/\text{m}^3$).

Measurements and Main Results: Among adults with COPD, a $10\text{-}\mu\text{g}/\text{m}^3$ increase in 1-year mean fine particulate matter exposure was associated with an elevated risk of cardiovascular mortality (hazard ratio, 1.10; 95% confidence interval [CI], 1.01–1.20). Effects were stronger in low exposure analyses (hazard ratio, 1.88; 95% CI, 1.56–2.27). Fine particulate matter exposure was not associated with acute myocardial infarction or stroke in overall analyses.

Conclusions: Long-term fine particulate matter exposure was associated with an increased risk of cardiovascular mortality among adults with COPD. Current regulations may not sufficiently protect those with COPD.

Keywords: air pollution; cardiovascular diseases; lung diseases



Review

FREE

March 27, 2020

Potential Effects of Coronaviruses on the Cardiovascular System

A Review

Mohammad Madjid, MD, MS¹; Payam Safavi-Naeini, MD²; Scott D. Solomon, MD³; et al

JAMA Cardiol. 2020;5(7):831-840

COVID-19 and its cardiovascular effects: a systematic review of prevalence studies

Pierpaolo Pellicori, Gemina Doolub, Chih Mun Wong, Keng Siang Lee, Kenneth Mangion, Mahmood Ahmad, Colin Berry, Iain Squire, Pier D Lambiase, Alexander Lyon, Alex McConnachie, Rod S Taylor, ✉ John GF Cleland

“...**hypertension**, **diabetes**, and **ischemic** heart disease are highly prevalent in people hospitalized with COVID-19 and are associated with an increased risk of death. In those admitted to hospital, biomarkers of cardiac stress or injury are often abnormal, and the incidence of a wide range of cardiovascular complications is substantial, particularly **arrhythmias**, **heart failure** and **thrombotic complications**.”

Risk of acute myocardial infarction and ischaemic stroke following COVID-19 in Sweden: a self-controlled case series and matched cohort study

[Ioannis Katsoularis, MD](#) [†] • [Osvaldo Fonseca-Rodríguez, PhD](#) [†] • [Paddy Farrington, PhD](#) • [Krister Lindmark, PhD](#)

[Anne-Marie Fors Connolly, PhD](#)   • [Show footnotes](#)

“...COVID-19 is a risk factor
for **acute myocardial
infarction** and ischemic
stroke.”

86,742

C-19 patients;

348,481 control

matched patients

THE LANCET



C. ANTHONY, CLAUDE
Aug 1994 14:52:23
P44 Time: 10:23:23
SERV

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0191
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American Journal of Respiratory and Critical Care Medicine

Weaning from Veno-Venous ECMO: Lessons from 60 Years of Weaning from Mechanical Ventilation

 Ricardo Teijeiro-Paradis 1, Antonio Pesenti 2,3, and Eddy Fan 1,4

PET_{CO_2}/Pa_{CO_2}

Summary Considerations

- Be aware of the increased likelihood of **CV related complications** in many patients with respiratory disease
- Expect a significant portion of your patients to require **creative approaches to care** due to CV disease
- **Be kind**, ...remember your patient has no choice (really) in how they're cared for, but you do

Thanks for listening.

tim.gilmore@lsuhs.edu

